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Simulative modeling to control the Foot and Mouth Disease epidemic

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Abstract

Reoccurring instances of Foot and Mouth Disease (FMD) in countries with underdeveloped infection surveillance have been taking a heavy toll on the lives of millions of livestock and causing annual financial losses worth billions of US dollars every year. FMD has been widely analyzed as a highly infectious disease that spreads rapidly with the movement of infected or contaminated animals of multiple species, and movement of people as well. Here, a meta-population based stochastic model is implemented to assess the FMD infection dynamics and to curb the economic losses in such countries. This model predicts the spatio-temporal evolution of FMD over a weighted contact network where the weights are characterized by the effect of wind and movement of animals and humans. FMD Incidence data from Turkey is used to calibrate and validate the model, and the predictive performance of our model is compared with baseline models as well. Finally, cost effective mitigation strategies are simulated using the theoretical concept of network fragmentation. Based on the theoretical reduction in the total number of infected animals, several practical mitigation strategies are proposed and their cost effectivenesses are also analyzed.

Keywords: Meta-population based model, Spatio-temporal, betweenness, closeness centrality, ergodicity

1. Introduction

Foot and Mouth Disease (FMD) is a viral infection that spreads rapidly among cloven-hoofed animals. It causes livestock to get sick, pregnant livestock to abort, dairy livestock to dry up and even death of infected animals. This disease poses a global threat due to the rapid rate of spread of FMD virus that result in massive unethical culls as the common measure to impede the rate of spread. Although vaccines have been developed to increase the immunity of livestock against FMD, administering vaccines is not preferred since the meat of a vaccinated livestock responds similarly to the test of infection as an infected livestock. Loss of meat, dairy and cattle trading privileges are the primary economic losses associated with a country reporting an FMD outbreak besides many other secondary losses involved. Hence, adequate pre-emptive modeling of disease dynamics is necessary to curb economic losses

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due to FMD.Consequently, several spatio-temporal models have been designed to study the underlying dynamics in the spread of FMD and to optimize the impact of FMD outbreaks both ethically and economically [2] [14]. Also, significant work has been done in simulating experimental instances of FMD outbreaks, and analyzing the cost of implementing optimal mitigation strategies [3].

Several countries like Turkey, Iran, Afghanistan and Thailand for instance have reported reoccurring instances of FMD over the past decade. However, such countries do not maintain a well developed database regarding the outbreak instances. One of the major reasons for reoccurring instance of FMD epidemics in such countries is the lack of surveillance and inadequate modeling of disease dynamics. Hence in this paper, we implement a meta-population based stochastic model to predict the disease dynamics in such countries, and we devise mitigation strategies based on the model to curb ethical and economic losses thus incurred.

We use an S-I-R compartmental model in which individuals are either in a Susceptible 'S' state, an Infected 'I' state, or a Recovered/Removed 'R' state to realize the evolution of FMD over a weighted contact network [8]. The weightages on the links of the FMD infection network are characterized by human interventions between geographic locations containing infected livestock, wind directions, grazing patterns, heterogeneity in livestock populations and the density of farms, feedlots, dairy and meat markets. The rate of transmission of infection between locations (nodes) in time is modeled stochastically to predict the probability of infection at different geographic locations in future time steps. The model is calibrated and validated using the OIE Incidence Reports regarding the incidence of the FMD virus in Turkish farms from January 2005 through December 2006 [15]. Our study invokes the contribution of wind in the spread of FMD since we analyze spatio-temporal predictions with and without the impact of wind as a contributor to the spread of FMD virus. Additionally, we study the direct costs that are associated with the implementation of mitigation strategies.

In this paper, the weighted network based model is defined and assessed in section 2. Section 3 delves into the simulations of infection networks resulting from the model. Next, mitigation strategies based on the theoretical concept of network fragmentation are discussed in section 4. Finally, concluding remarks and discussions are presented in section 5.

2. The Model

We implement a stochastic compartmental model based on a weighted contact network to map the dynamic spread of FMD independent of any geographic location [8]. This flexibility of epidemic prediction in space and time is achieved by the weights on the links of the weighted contact network that represent the heterogeneities in space and the parameterization of the proposed model characterizes the dynamic spread of infection spatio-temporally.

The initial realization of a weighted contact network is achieved by defining nodes as geographical locations represented by the latitude and longitude of regions housing livestock that can be potentially infected or that can contribute to the spread of the FMD virus. For our case study of FMD in Turkey from January 2005 through December 2006, we define a node as a single administration division in Turkey. The weighted links connecting the nodes of our meta-population based model are characterized using the following factors:

- Human interactions between the various locations based on the rate of thoroughfare between locations and the regional population densities [12], and
- Regional wind speed and direction [13].

Next, we characterize parameters that impact the spread of FMD infection. For example, different species of animals, such as cattle, sheep, and swine, react differently to the FMD virus, and thus the rate of infectivity varies with the type of susceptible flock [1]. Additionally, different grazing patterns at various locations add to the heterogeneities of the infection network [11], [12]. Thus, the type of livestock, the fraction of flocks of different species grazing away from, and grazing into a specific region, and the distribution of slaughter houses and meat markets in a particular geographical location characterize the transmission parameters in our stochastic prediction model.

Thus, at any time step t we define three sets of nodes N , $N_i(t)$ and N_s as the following:

$$N = \{x : x \in All\ nodes\}$$
$$N_{inf}(t) = \{y : y \in Infected\ nodes\}$$

$$N_s(t) = \{v : v \in Susceptible\ nodes\} \tag{1}$$

Such that the node sets N, $N_{inf}(t)$ and $N_s(t)$ are related as: $N_{inf}(t), N_s(t) \subseteq N$. Initially, at time $t = 0$ all nodes are equally susceptible and no node is infected. Thus, $N_{inf}(t) = \{\phi\}$, $N_s(t) = N$.

After the infection is seeded at time $t = 1$, $N_{inf}(t)$ and $N_s(t)$ change at each following time step, thus affecting the probability of infection at each node. At any given time step t, the probability ($\zeta_{t,i}$) that a node i, does not receive infections from infectious neighbors j, is given as the following [10]:

$$\zeta_{t,i} = \left\{ \prod_{j \in N_{inf}(t)} (1 - \frac{\omega_{i,j}\beta_{i,j}p_{t-1,j}}{\omega_{i,j}\beta_{i,j} + \delta_j(t)}) \right\} \forall i \in N \tag{2}$$

For any node i, the second term within the parentheses is the probability that at time step t, the node i receives infection from an infected node j without getting recovered/removed. On subtracting this probability from unity we obtain $\zeta_{t,i}$.

- $p_{t,i}$ is the probability of a node i being infected at time t and is given by the following [10]:

$$p_{t,i} = \{1 - (1 - p_{t-1,i})\zeta_{t,i} - \delta_i(t)p_{t-1,i}\}\ \forall i \in N \tag{3}$$

- $\delta_i(t)$ = the rate at which the FMDV is removed from a node i, or the disease cure rate. This value is estimated as the number of livestock removed due to slaughtering at a particular node at a particular time instant t.
- $\omega_{i,j}$= the weightage associated with a link between node i and j.
- $\beta_{i,j}$ = the rate at which infection is incident on node i due to an infected neighbor j.

As a novel contribution, we parameterize the weights $\omega_{i,j}$ on links of a fully connected contact network using the data on human interventions between locations and the impact of wind.

$$\omega_{i,j} = \left\{ c_m[\frac{m_i * m_j}{d_{i,j}}]^{-\gamma_m} + c_w \| \frac{\vec{D_{i,j}}(\vec{W_{i,j}} \cdot \vec{D_{i,j}})}{|\vec{D_{i,j}}|^2} \| \right\} \quad [\forall i, j \in N,\ \ i \neq j] \tag{4}$$

- m_i = the scaled measure of population density in location i
- m_j = the scaled measure of population density in location j
- $d_{i,j}$ = the scalar distance between node i and i
- c_m, c_w = the constants due to human interventions and wind respectively.
- γ_m = the exponent to represent nature of human interventions [9].
- $\vec{D_{i,j}}$ = the vector corresponding to the separation between node i and j in terms of the latitudinal and longitudinal coordinates.
- $\vec{W_{i,j}}$ = the vector corresponding to the velocity of wind blowing between node i and j in terms of the latitudinal and longitudinal coordinates.

Next we parameterize the FMD transmission characteristics $\beta_{i,j}$ that maps the flow of infection through the contact network.

$$\beta_{i,j} = \left\{ (S_i\sigma_i N_{inf} M_i)(T_j\tau_j n_j M_j)K(d_{i,j}) \right\} \quad \left[\forall i \in N, j \in N_{inf}(t), i \neq j \right] \tag{5}$$

- S_i = susceptibility of livestock in location i
- σ_i = fraction of the infected livestock grazing into the location i.
- N_{inf} = number of susceptible livestock in location i.
- M_i = weightage associated with node i due to the presence of slaughter houses and meat markets.

- T_j = transmissibility of infected livestock in neighborhood j.
- τ_j = fraction of the infected livestock grazing away from the location j.
- n_j = number of susceptible livestock in location j.
- M_j = weightage associated with location j due presence of slaughter houses and markets.
- $K(d_{i,j})$ = Transmission kernel function depicting the relative risk of infection based on the distance between location i and j defined by the following:

$$K(d_{i,j}) = c\left\{1 + \frac{d_{i,j}}{a}\right\}^{-\gamma} \tag{6}$$

Thus, $\beta_{i,j}$ depicts the rate of transmission of infection from node j to node i due to the grazing movements of infected livestock. A non-linear kernel function characterizes the relative risk of infection with varying Euclidean distance between nodes [4]. We assume that the relative spread of infection decreases exponentially with increasing distance which is parallel to the general assumption that FMD infection at a particular location affects regions within a 10 km radius. The parameter c is a multiplicative constant such that $c = 1$ under normal conditions of infection spread without mitigation strategies. Additionally, the parameters a and γ are known as the kernel offset and kernel exponent respectively which are estimated from the data set during the model calibration phase.

2.1. Model Assessment

Predictive performance of our model is assessed in comparison to three other network based models. The four network based models under analysis are defined as:

- W = The proposed Weighted, Parameterized model.
- NW= Weighted, Parameterized model without considering the impact of wind.
- $B1$ = Unweighted, Parameterized Baseline model with $\omega_{i,j}$ in Equation 4 constant.
- $B2$ = Unweighted, Unparameterized Baseline model with $\omega_{i,j}$ in Equation 4 and parameters S_i, σ_i, T_j, τ_j and M_j in Equation 5 constant.

We assess the contribution of wind in the spread of FMD by comparing the predictive performance of model W against model NW. Besides, the contribution of a weighted underlying contact network and disease transmission parameters that are novel to our model are assessed by comparing model W with baseline model $B1$ and $B2$ respectively.

2.1.1. Model Calibration

The parameters specific to the network based models need to be estimated so as to fit the data set of Turkey accurately. For calibration purpose, we consider 60% of the data set, which is FMD infection incidence reports for a 15 month period from January 2005 March 2006, as training data. To obtain the parameter sets for each of the four network based models, we minimize the root mean squared error (RMSE) between the true monthly probability distribution of infection, and the simulated infection probability across all nodes using Nelder Mead Optimization.

The model W requires optimized values for its set of five parameters $(c_m, \gamma_m, c_w, a, \gamma)$. While model NW requires optimization of its set of four parameters $(c_m, \gamma_m, a, \gamma)$. Baseline models $B1, B2$ require an optimized set of 2 parameters (a, γ). Additionally, a threshold probability (p_{th}) is defined such that if the probability of infection at a particular node $p_i(t) \geq p_{th}$, then node i is said to be infected, or else node i is susceptible. Out of a number of optimal parameter sets that minimize RMSE, the best parameter set θ is identified by the best point marked by the ring in the Receiver Operating Characteristic (ROC) curve in Figure 1. Additionally, the optimal parameter sets for the calibrated network models are summarized in Table 1.

2.1.2. Model validation

The calibrated network based infection models are validated for a validation data set that corresponds to the remaining 40% of the data set defined by a period of 9 months from April 2006 through December 2006. Also, an aggregated validation is performed on the entire data set defined by a period of 24 months from January 2005 through December 2006.

The statistical parameters used to validate the spatio-temporal predictive performance of the models are: sensitivity, specificivity, Positive Prediction Value (PPV), Negative Prediction Value (NPV) and accuracy. Here, the

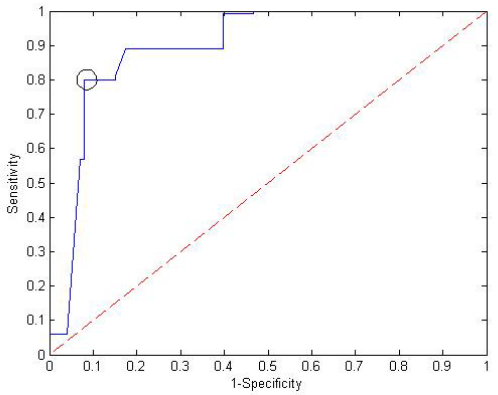


Figure 1: Receiver Operating Characteristic (ROC) curve for determining the optimal parameter set for model W. The optimal parameter set is denoted by the parameter set corresponding to the position marked by the dark ring on the curve.

Table 1: Optimized Model Parameters

Network Based Model	c_m	γ_m	c_w	Kernel offset (a)	Kernel exponent (γ)
W	0.5709	4.053	0.5642	459.3133	27.338
NW	0.4056	3.3248	-	215.74	49.486
B1	-	-	-	98.98	42.272
B2	-	-	-	170.0467	52.062

significance of the various parameters are defined as follows: the ability of a network based model to predict an infected node to be infected is depicted by a high value of sensitivity and PPV. Specificity and NPV denote instances when an uninfected node is correctly predicted to be uninfected. Accuracy of any model refers to the closeness of the prediction from the models to the actual data. Next, the temporal prediction performance of the models are assessed using the coefficient of determination (R^2) and symmetric mean absolute percentage error ($sMAPE$) which is an accuracy measure based on percentage (or relative) errors. A higher of value R^2 and a lower value of $sMAPE$ signifies a well fitted model. The predictive capability of the models is evaluated in terms of deviance information criterion (DIC) that penalizes a model with greater number of parameters and uses Bayesian estimators to compute the computational complexity associated with each model. Since we have previously calibrated the optimal parameter set θ for each network model, we evaluate the probability distribution of infection, and thus compute the DIC for the predictions produced by each model. A low value of DIC ensures a better predictive model. The statistical results after comparison of the four network models over the entire data set for 24 months and the validation data set of 9 months is summarized in Table 2.

Thus we infer that spatial predictions of weighted network based models are comparatively better than unweighted models. However, the accuracy of spatial and temporal predictions from model W and NW are found to be comparable. Additionally, the model W when compared to NW has a higher sensitivity, but a lower PPV. These observations result from the fact that we considered an incomplete wind data set for computation. However, it is noteworthy, that in spite of an incomplete wind data set, model W demonstrates a higher specificity, NPV, R^2, $sMAPE$ and a lower DIC than all the other network based models.

3. Simulation of Infection Networks

Once our model W is calibrated and validated, we analyze the FMD infection networks thus produced. We perform Monte-Carlo simulations with randomly selected threshold probabilities (p_{th}) to analyze the various infection routes

Table 2: Statistical Analysis of Spatial and Temporal Prediction

Entire Data Set					Validation Data Set			
Statistics	W	NW	$B1$	$B2$	W	NW	$B1$	$B2$
sensitivity	0.711	0.661	0.546	0.377	0.721	0.667	0.554	0.372
specificity	0.945	0.959	0.706	0.613	0.943	0.969	0.539	0.376
PPV	0.722	0.749	0.494	0.327	0.878	0.918	0.535	0.361
NPV	0.876	0.859	0.749	0.633	0.802	0.778	0.583	0.389
accuracy	0.878	0.872	0.719	0.626	0.847	0.837	0.595	0.449
R^2	0.993	0.955	0.725	0.492	0.993	0.972	0.629	0.417
$sMAPE$	9.391	14.914	21.190	33.181	12.628	16.691	25.513	41.705
DIC	483.203	486.296	832.937	1040.589	-	-	-	-

that can be traced during the spread of FMD. An important assumption for our simulations is that the state of complete removal of a node from the infection network is achievable only if either all the herds at that particular node have been culled, or they have all recovered from the infection such that they are immune to acquiring the infection in future time steps. Since our simulation is limited to a specific time period of observation, we do not encounter any node becoming completely recovered or removed from the infection network at any time step. A single realization of such a simulation is shown in Figure 2. These simulations depict the dynamic growth of the infection network both spatially and temporally. Additionally the rate of growth of infection networks represent the direction and velocity of the spread of the FMD virus based on which, effective mitigation strategies may be coined.

3.1. Ergodicity of Infection Networks

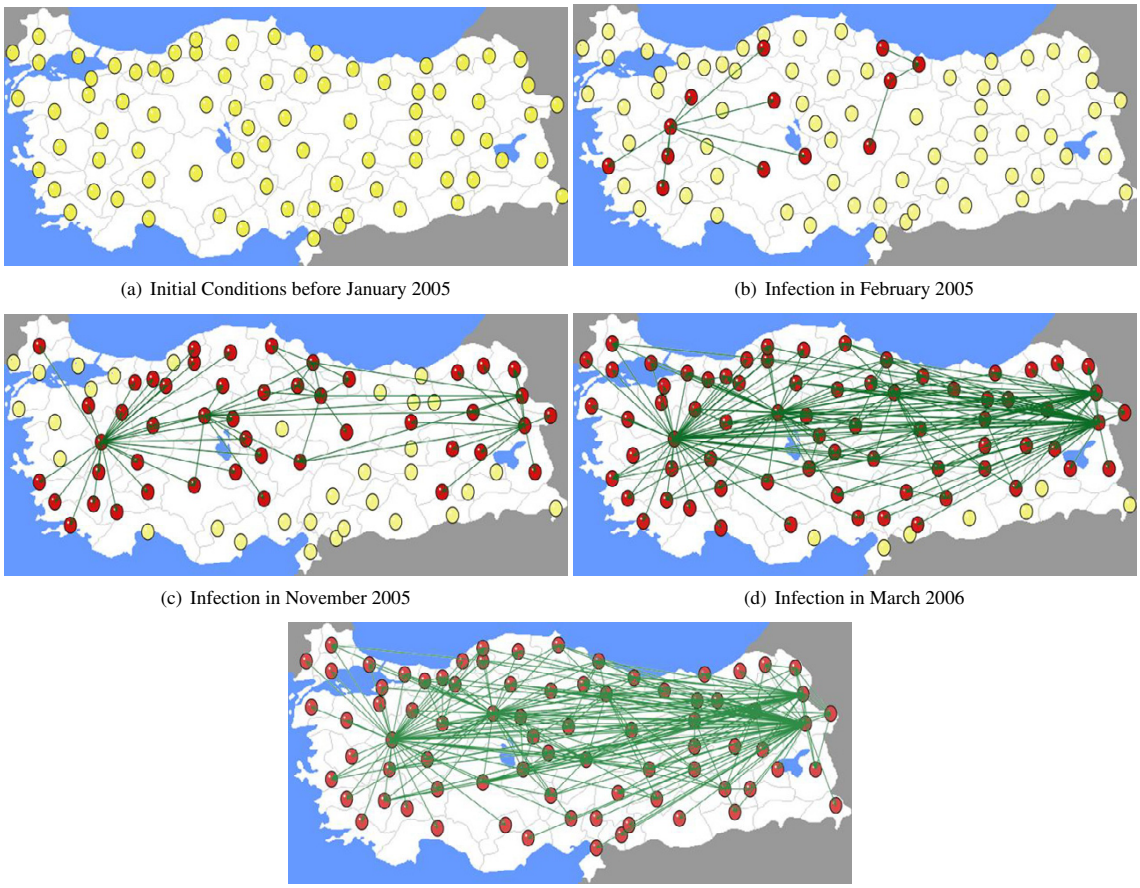
To ensure realistic network structure of the dynamically growing infection networks, it is essential to asses if the underlying behavior of the network remains unchanged over the entire course of the epidemic [5]. Although the underlying weighted contact network is fully connected, the infection networks which are traced out by the model W in different runs of simulation corresponding to different values of p_{th}, are not necessarily fully connected. The concept of ergodicity in networks assumes that the mean underlying network feature represented by the size of the giant connected component in the infection network versus the secondary reproduction ratio (R_0) shall remain consistent independent of the method of realization. Different infection networks are realized over all possible time instants starting January 2005 through December 2006 using the a single threshold probability for determination of infected nodes and links, as well as by randomly selecting different threshold probabilities to realize infection networks in a particular time instant only. Hence, multiple snapshots of the infection networks will give the same estimate regarding the distribution of the size of the giant component growing with R_0 as shown in Figure 3.

4. Mitigation Strategies

Mitigation strategies to bring the FMD epidemic under control involve isolation of specific nodes which have a high probability of infecting other susceptible nodes. Here, isolation refers to removing individual markets (through a movement ban) or farms (through culling) from the FMD infection network by vaccination, or culling of all infected and neighboring livestock. Deletion of nodes based on highest betweenness centrality, lowest closeness centrality and highest node degree provides an insight into the optimal control strategies that may be adopted to control the FMD epidemic.

Although network fragmentation theoretically retards the spread of infection by reducing the size of the largest component, it is practically not possible to obtain a similar level of node isolation in real-world infection networks. Conversely, vaccination and movement bans are ethical but they are costly policies, and they fail to check immediate infection spread. Thus, it is absolutely imperative to formulate mitigation strategies that are both practically feasible, ethical and cost-effective too.

Mitigation strategies are simulated using the weighted network based model such that at the end of each month, each node is assigned a certain probability of infection $p_i(t)$ based on which a certain mitigation task is performed at each node. The followng mitigation tasks are adopted to control the spread of FMD by varying the constant c in the transmission kernel function.



(a) Initial Conditions before January 2005

(b) Infection in February 2005

(c) Infection in November 2005

(d) Infection in March 2006

(e) Infection in December 2006

Figure 2: Foot and mouth disease infection network in Turkey after seeding the infection in January 2005. The links signify the direction in which FMD spreads from a previously infected node to a recently infected node. Visualization is generated using KING [http://kinemage.biochem.duke.edu/software/king.php.]

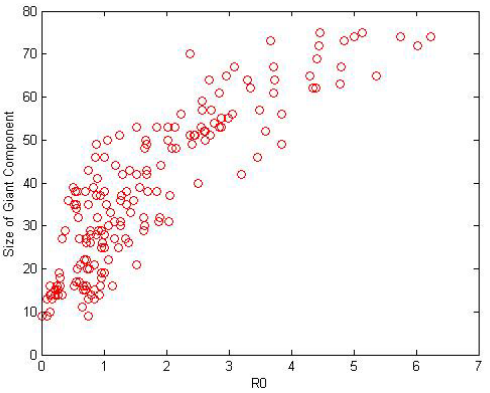


Figure 3: The growth of the size of the Giant connected Component for different values of secondary reproduction ratio R_0. The plot shows 20 realizations using the following two methods: 10 realizations of infection networks are traced by using a single randomly selected value of p_{th} in [0,1] over 24 time instants. Additionally, 10 realizations of infection networks are traced by using a randomly selected value of p_{th} over a particular randomly selected time instant. Both realizations provide similar estimates regarding the growth of the giant connected component, and thus the ergodicity of infection networks may be assumed.

1. Infected Premise cull (IP): This strategy is adopted at nodes with very high probability of incident infection. Accordingly, all livestock in the particular premise (node) are culled within next 24-48hours. IP culls are the best strategy to impede the spread of infection. $c = 0.60$ for IP culls within 24 hours, and $c = 0.70$ for IP culls within 48 hours.
2. Dangerous Contact cull (DC): This strategy is adopted at nodes that are not yet infected but they have an infected node in their neighborhood and hence, there is a high probability of infection in future. Accordingly, all livestock in such a premise (node) are culled within the next 4-7 days. $c = 0.75$ for DC culls within 4 days and $c = 0.82$ for DC culls within 7 days.
3. Vaccination (Vacc): If the vaccine to the particular strain of FMD virus is available, its administration will reduce the probability of future infections. The impedance to the spread of infection is however much lesser than that due to culling. For a potent vaccine $c = 0.85$ while a less potent vaccine assumes $c = 0.9$.
4. No Movement Bans (NM): For nodes having a low probability of incident infection but with a potential of getting infected in future, the grazing movements of animals are banned. Human trespassing is also banned from such territories to prevent the spread of the virus. However, a high cost per day is incurred due to these movement restrictions. Strict and non strict movement bans are implemented by varying $c = 0.9$ and $c = 0.95$ respectively.

For an effective mitigation strategy, we randomly evaluate the probabilities: $pth_1, pth_2, pth_3, pth_4$ such that:

- $pth_1 = 0.95 * p_{th}$
- $pth_2 = 0.9 * p_{th}$
- $pth_3 = 0.85 * p_{th}$
- $pth_4 = 0.8 * p_{th}$

Next, at each node i, having a probability of infection $p_i(t)$ in time step t, we perform the following set of decisions.

- If $p_i(t) > pth_1$, perform an mitigation task 1 at that node.
- If $pth_1 > p_i(t) > pth_2$, perform mitigation task 2 at that node.
- If $pth_2 > p_i(t) > pth_3$, perform mitigation task 3 at that node.
- If $pth_3 > p_i(t) > pth_4$, perform mitigation task 4 at that node.
- If $p_i(t) < pth_4$ no mitigation strategy is applied at that node.

Table 3: Sequence of Mitigation Tasks in different Mitigation Strategies.

Strategy	Mitigation Task 1	Mitigation Task 2	Mitigation Task 3	Mitigation Task 4
1	IP	DC	Vacc	NM
2	Vacc	NM	-	-
3	IP	DC	NM	-
4	IP	DC	NM	Vacc
5	IP	Vacc	-	-
6	IP	NM	-	-

Table 4: Effectiveness of mitigation strategies. The range of values represents the lower and upper bounds on the impact of mitigation strategies depending on the responsiveness to culls, potency of vaccines and strictness of movement restrictions.

Strategy	Percentage Reduction	Cost in US Dollars	Effectiveness
1	46.889 - 48.652	482.95 - 568.63	0.086 - 0.097
2	20.778 - 24.464	155.48 - 164.46	0.134 - 0.148
3	46.889 - 48.652	356.95 - 395.38	0.123 - 0.131
4	46.889 - 48.652	460.48 - 504.45	0.096- 0.102
5	46.406 - 49.268	355.58 - 377.28	0.131 - 0.132
6	46.406 - 49.268	495.06 - 481.07	0.094 - 0.102

Finally, we evaluate the impact of six different mitigation strategies on reducing the total number of infected animals at the end of December 2006. These mitigation strategies are identified by a set of mitigation tasks defined in Table 3.

To devise economic mitigation strategies, it is imperative to understand the cost-effectivenesses of the mitigation strategies. We assume that the cost per head to breed cattle ranges between 1100-1300 US dollars and that of sheep is between 110-140 US dollars [6]. Additionally, the cost of vaccinating cattle or sheep varies from 4.0-10.0 US dollars per head for varying potency of vaccine; the cost to administer euthanasia or cull cattle is 16.5 US dollars per head and 2.31 US dollars per head for sheep [6]. In case of movement restrictions, a cost range of 150,000-165,000 US dollars is incurred per day depending on the strictness of the ban. The cost-effectiveness of a mitigation strategy is defined as following:

$$Effectiveness = \frac{\%\ Reduction\ in\ number\ of\ infected\ livestock}{Cost\ incurred\ (million\ US\ \$)} \tag{7}$$

From Table 4 we infer that pre-emptive vaccination followed by movement restrictions imposed by mitigation strategy 2 is the most cost effective strategy. However, mitigation strategies that impose culls at the most probable nodes of infection are effective in reducing the total number of infected animals to almost 50%. Also, IP culls when followed up with vaccinations as in mitigation strategy 5, are cost effective as well as they achieve a high reduction in the total number of infected animals.

5. Concluding Remarks and Discussion

The primary contribution of this work is modeling disease dynamics in a country such as Turkey where FMD outbreaks have been insufficiently studied so far as compared to the highly analyzed outbreaks in the United Kingdom in 2001 and in 2007. We aim at devising models that studies the disease dynamics in such countries with undeveloped data bases. Additionally, we suggest practical mitigation strategies and their economic impacts with respect to immediate costs incurred for their implementation. To achieve modeling of disease dynamics in Turkey from January 2005 through December 2006, we characterize a meta-population based stochastic model for FMD that predicts the probability of infection at any location in discrete time steps. The novelty of this model is that it considers a weighted contact network wherein the weights on links are characterized by wind and human interventions between regions. Additionally, the parameterization of the spread FMD dynamics is achieved by considering the grazing patterns of

infected livestock, the species of livestock, and the number of meat markets and slaughter houses in the different regions.

Spatial and temporal predictability of the model is compared to that of unweighted and unparameterized baseline models. Additionally, the contribution of wind in the spread of FMD is also studied. Although the weights and parameterizations increase precision in spatio-temporal predictability, the contribution of wind needs to be studied more extensively on other data sets in future to infer the effectiveness of its contribution. However, for the current data set on Turkey, the predictive performance of our weighted network based model is noteworthy.

Finally, we simulate practical mitigation strategies based on the theoretical concept of network fragmentation. The proposed model is used to simulate cost effective mitigation strategies based on the acuteness of epidemic outbreaks. Different mitigation tasks such as Infected Premise (IP) culls, Dangerous Contact (DC) culls, and Vaccination (Vacc) and No Movement (NM) restrictions are simulated by varying the transmission kernel function. Conclusively, we observe that IP and DC culls followed up by vaccinations result in a strong reduction in the number of infected livestock and they retard the rapid spread of the FMD virus. However, the cost incurred in this process is quite high. Hence, this mitigation strategy should be adopted after the onset of an epidemic outbreak when immediate reduction in the infection network is mandatory. On the other hand, a potent vaccination strategy, followed by movement restrictions results in the most cost-effective control of the epidemic. This strategy may be adopted before the onset of an epidemic outbreak to control the rapid spread of the FMD infection.

Since the proposed simulative model is iterative, its time steps can be varied to suit the predictive requirements. Additionally, the proposed model is geographically independent, and thus it can be used to model the disease dynamics in other countries with undeveloped databases in the future.

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