Spatio-temporal Modeling of Foot-and-Mouth Outbreaks

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Abstract

We present and analyze the data collected during a severe epidemic of foot-and-mouth disease (FMD) that has occurred, between July and September 2000, in a region of northeastern Greece with strategic importance since it represents the southeastern border of Europe and Asia. We implement generic Bayesian methodology, which offers flexibility in the ability to fit several realistically complex models that simultaneously captures the presence of "excess" zeros, the spatio temporal dependence of the cases, assesses the impact of environmental noise and controls for multicollinearity issues. Our findings suggest that the epidemic was mostly driven by the size and the animal type of each farm as well as the distance between farms

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while environmental and other endemic factors were not important during this outbreak. Analyses of this kind may prove useful to informing decisions related to optimal control measures for potential future FMD outbreaks as well as other acute epidemics such as FMD.

1. Introduction

Foot-and-mouth disease (FMD) is a highly infectious disease of cloven-footed animals, responsible for severe epidemics that lead in reduced productivity [1]. Animals generally recover from the disease but subsequent milk yields and weight are permanently reduced, hence the effects on the livestock industry can be substantial [2]. Although Northern Greece is free from FMD, sporadic epidemics may occur. The specific region is of strategic importance as it represents the southeastern border of Europe and Asia. However, despite the measures taken to prevent the introduction of FMD in the region northeastern Greece experienced a severe epidemic, during 2000. All infected farms detected at the start of the outbreak were in very close proximity to the Evros River, bordering Turkey, and the strain of the virus isolated in Greece during the 2000 epidemic was found to be identical to the strain isolated in Turkey in 1999 and 2000 ([3], accessed January, 2014).

Recent modeling approaches for the prediction of FMD occurrence during FMD epidemics include the development of Bayesian spatio-temporal regression models that introduce - in addition to covariate information – Ornstein-Uhlenbeck stochastic components ([4], [5]), or discrete-type distributions suitable for this type of data ([4], [6]). However, to the best of our knowledge, there is absence of analysis that simultaneously addresses certain characteristics of epidemic data, such as the

spatiotemporal dependence, the environmental noise, multicollinearity issues and the presence of "excess" zeros. An alternative class of models which have been successfully applied to FMD data is based upon suitable extensions of stochastic Susceptible-Infectious-Removed (SIR) models, see for example [7] and references therein. Although the two model classes share certain characteristics since they are, essentially, different versions of a general family of counting processes, there are a number of differences; SIR models focus upon detailed and explicit modeling of the transmission mechanism at the expense of increased computational cost and complex model analysis. On the other hand, our transmission models essentially look at discretised (like daily, weekly or monthly) data and in doing so we (i) gain in computational simplicity, since the model can be fitted in the WinBUGS software [8], and (ii) by creating the (artificial) extra zeros we are able to see which factors (e.g. environmental) assist in creating a disease-free environment through the covariates linked to the excess zeros.

The epidemiological objective of this work was to assess the impact of various explanatory variables, such as species, environmental factors and the spatial component on the spread of the FMD epidemic in this region. To achieve this we adopt a Bayesian modeling approach that accounts for the frequently observed non-occurrence of the disease in time and space, by implementing zero-inflated distributions [9]. We further incorporate spatial information associated with the locations of infected farms in the form of kernel functions. Multicollinearity issues are also addressed by implementing appropriate Bayesian variable selection techniques. Finally, the incorporation of an Ornstein-Uhlenbeck (OU) component into our models allows for structured, autoregressive-type, stochasticity, vital for temporal epidemic

data. We show that the utilization of all the above mentioned modeling strategies has a major impact on model fit and the prediction of disease spread.

2.1 Data on the 2000 FMD outbreak

Between July and September, 2000, Greece experienced a major epidemic of FMD. The conducted laboratory tests confirmed the FMD virus, of Asia-1 serotype. In total, approximately 5,600 cattle, 4,300 sheep/goats and 360 pigs were culled during the course of the outbreak. No vaccination was used to control the outbreak.

Farm-level data for the 2000 FMD epidemic were provided by the Veterinary Directorate of Northern Evros Prefecture (VDNEP). This dataset has not been previously presented or analyzed and presents a unique opportunity to give insight into the true patterns of behavior of a real epidemic situation of this kind. Figure 1 shows the temporal progress of the disease in terms of disease occurrence.

INSERT FIGURE 1 APPROXIMATELY HERE

2.2 Statistical Analysis

2.2.1 Model structure

Let y_i denote the number of herds with new FMD infections at time t_i where $i \in \{0,1,...,72\}$ is ordered chronologically first by month and then by day. We assume that:

$$y_i \sim g\left(y_i \mid \theta_i\right) \tag{1}$$

with

$$\theta_i = h\left(\lambda_i\right) = \exp\left(\lambda_i\right) \tag{2}$$

and

$$d\lambda_t = \phi\left(\lambda_t - \mu_t\right)dt + dB_t \tag{3}$$

where g denotes the assumed distribution of the data, λ_i is the rate at which new infections take place during the progress/spread of the disease, B_t denotes the standard Brownian motion and ϕ the reversion parameter (i.e. the rate at which the process returns to its long-run mean) of the OU process, which is incorporated into our model through equation (3) for capturing distributional deviations in disease occurrence. Also, μ_t is a piecewise constant deterministic process given by:

$$\mu_t = \begin{cases} \mu^{(0)} & if \quad 0 \le t < t_1 \\ \mu^{(1)} & if \quad t_1 \le t < t_2 \\ \vdots \\ \mu^{(71)} & if \quad t_{71} \le t < t_{72} \end{cases} \tag{4}$$

with each $\mu^{(i)}$ corresponding to $t_i \le t < t_{i+1}$ ($i = 0,1,...,71$) given by:

$$\mu^{(i)} = \mathbf{X}_{(i)} \cdot \mathbf{\beta} + \tau \cdot y_{i-1} + K\left(d_i, \mathbf{\Theta_K}\right). \tag{5}$$

Here $\mathrm{X}_{(i)}$ is the matrix of covariates, and $\mathbf{\beta}$ the vector of the corresponding coefficients, τ is a simple autocorrelation term associated with the influence of the number of FMD cases in the previous day y_{i-1} on y_i. Finally, $K\left(d_i, \mathbf{\Theta_K}\right)$ is a spatial kernel for the incorporation of spatial information associated with the rate at which infection passes from an infected farm ℓ at times $i - j$ ($j = 1,2...,14$), i.e. assuming a two-week incubation period [10], to a susceptible farm k at time i, which captures the fact that FMD is more likely to spread between farms located nearby than farms located more distant apart:

$$K\left(d_i, \Theta_K\right) = \begin{cases} \dfrac{1}{|d_i|} \displaystyle\sum_{k \in S_i} \sum_{\ell \in I_{i-j}} K\left(d_{k\ell}, \Theta_K\right) & \text{if at least one } y_{i-j} > 0 \ (j = 1, 2, \dots, 14) \\ K\left(d_{\min}, \Theta_K\right) \text{if all } y_{i-j} = 0 \ (j = 1, 2, \dots, 14) \end{cases} \tag{6}$$

where $|d_i|$ denotes the cardinality of d_i, the term $\mathrm{K}\left(d_{k\ell}, \Theta_K\right)$ denotes some specified function of the distance between the infected and susceptible farms based upon their distance $d_{k\ell}$ and $d_{\min}$, which is set a priori, restricts the minimum distance over which infections do not occur [11]. The functional forms that we tested for K are given in Table 1. Selection of the most appropriate forms was based on a variety of relevant functions (e.g. [12], [13], [14]).

INSERT TABLE 1 APPROXIMATELY HERE

The model specified in (1)-(6) can be thought of as an elaborate version of a log-linear type of model for the temporal component, with the addition of an autoregressive term of order one (the O-U process) and a spatial component captured by $\mathrm{K}\left(d_{k\ell}, \Theta_K\right)$.

2.2.2 Candidate distributions for the epidemic data

For discrete epidemic data, we initially assumed a Poisson $\mathrm{P}(\lambda_i)$ or a negative binomial $\mathrm{NB}(r, q_i)$ distribution. That is

$$\mu_i = \log(\lambda_i) \tag{7}$$

and

$$\mu_i = \frac{r(1-q_i)}{q_i} \tag{8}$$

respectively.

Importantly, in order to account for the presence of excess zeros, often occurring in epidemic data, we also considered the zero inflated Poisson (ZIP) and zero inflated negative binomial model (ZINB). Previous work (e.g. [15]) has demonstrated that capturing the frequently observed non-occurrence of the disease, by modeling excess zeros in time and space, can considerably improve model fit. To do so, Eq. 1 takes the following form:

$$y_i \sim g(y_i \mid \theta_i; p_i) \tag{9}$$

with

$$g(y_i \mid \theta_i; p_i) = p_i I_{\{y_i=0\}} + (1-p_i) f(y_i \mid \theta_i) \tag{10}$$

Here $I_{\{y_{ij}=0\}}$ is an indicator variable for whether or not the FMD cases were observed at time i and p_i ($0 \le p_i \le 1$) the probability of observing excess zeros at time i. Finally, the zero-inflated probability p_i can be linked to covariates through:

$$\log\left(\frac{p_i}{1-p_i}\right) = \mathbf{X}_{(i)}^t \cdot \boldsymbol{\beta}^z + \tau^z \cdot y_{i-1} + K(d_i, \boldsymbol{\Theta}_{\mathbf{K}}^z) \tag{11}$$

with the superscript z distinguishing the parameters linking covariates with the probability of excess zeros from those linked to the infection rate in Eq 5.

Selection of the best between the P, NB, ZIP and ZINB models is not a trivial matter since the complexity of those models is unclear. In this paper we resort to deviance-based measures due to the well-known equivalence in model selection using cross-validation or AIC (see [16]). Given the unknown complexity of the entertained models, we chose to perform model selection based upon the mean deviance ($\bar{D}$) as well as the deviance information criterion (DIC), with smaller values for both criteria indicating better fit [17].

2.2.3 Screening selection process of the candidate variables.

Candidate variables for inclusion on the final model contained information on several meteorological/environmental predictors as well as size/type of infected farms. The specific variables were chosen based on previous research (see e.g. [4], [13]). A complete list of all candidate variables along with descriptive statistics can be found in the supplement (Table S1). The meteorological data were acquired from the Greek National Meteorological Service (http://www.hnms.gr/hnms/english/index_html). Initially we selected the best distributional form and the best kernel function, according to the $\bar{D}$ values. Then, conditional upon the selection of the best distribution and kernel, we performed a backwards elimination variable selection process, eliminating the least significant variable (at a 5% significance level) each time.

2.2.4 Prior specifications

For the fixed effects parameters $\boldsymbol{\beta}$ (accordingly $\boldsymbol{\beta}^z$) we adopt a g-prior type of approach for specifying the prior distributions in a way that accounts for potential correlations among the explanatory variables. Hence we assumed a multivariate

Gaussian prior density, with zero-mean and a prior variance matrix of the form:
$ge^{\beta_{\backslash 0}}\left(\mathbf{X}_{\backslash 0}^{t}\cdot\mathbf{X}_{\backslash 0}\right)^{-1}$, where $\beta_{\backslash 0}$ is the vector $\boldsymbol{\beta}$ excluding β_{0}, $\mathbf{X}_{\backslash 0}$ is the data matrix

without the intercept, and $e^{\beta_{\backslash 0}}$ is a rough estimate of λ_{i} (for more details see [18] or

[19]). Finally, a weakly-informative $N\left(0,10^{4}\right)$ prior was used for the intercept β_{0}

and the parameters τ, τ^{z}.

2.2.5 Assessing the impact of different parameters in efficacy of control

An interesting application of the modeling framework adopted in the current

study relates to an attempt to measure the relative contribution of the parameters fitted

in the final model. The latter is permitted by the decomposition of the covariates

included in the final model into two parts: an endemic disease dynamic process,

originating outside its internal history, which sums up the effects of significant

covariates like those measuring environmental factors and an epidemic component,

which summarizes the effects of significant covariates representing the internal

dynamics like farm-to-farm locations and type/size of farms (see [20]). The latter is

achieved via the additive decomposition of μ_{t}, the mean driving the log-rate of

infection λ_{t} through:

$$\mu_{t}=\mu_{endemic}+\mu_{epidemic}, \tag{12}$$

where $\mu_{endemic}$ ($\mu_{epidemic}$) denotes the time-dependent endemic (epidemic) component-

related parameters. Within this context, we have an indication of the relative

contribution of each part in the spread of the disease. A large epidemic component

would suggest imposing restrictions associated with the spatial allocation of farm

structure in the region of interest, whereas the opposite results may indicate that most

of infections are due to external factors and thus are less sensitive to such control measures.

2.2.6 Posterior predictive model checking

In order to assess the predictive accuracy of our modeling we compare replicated data constructed under the fitted model with the observed data. Hence, simulated values y_i^{rep} are drawn from the posterior predictive distribution of replicated data through:

$$y_i^{rep} \sim g\left(y_i^{rep} \mid \theta_i; p_i\right), \tag{13}$$

and compared to the observed data y_i, with similar values between y_i and y_i^{rep} indicating a good fit.

2.2.7 Bayesian Inference and Convergence Diagnostics

We have used the WinBUGS software to fit the models. The posterior results are obtained after discarding the initial 5,000 iterations, using an additional sample of 10,000 iterations (using a thinning lag of 10). Concerning the convergence of the parameters, examination of history plots indicated no lack of convergence for all fitted models. We have also examined for autocorrelation through visual inspection of autocorrelation plots for each estimated parameter and found acceptable autocorrelation levels for all parameters. The WinBUGS code for the best selected model with step by step explanations is available in the supplement.

3. Results

From the 1st of July 2000 until the 10th of September 2000 a total of 100 farms in the region got infected by FMD. The median number of farms that became infected

daily was 3 (minimum/maximum farms with infections per day was 1 and 12, respectively, excluding days with zero occurrence). For 42 days during this epidemic no new farms (zero observations) got infected.

Due to the presence of the latter one of the models that adjusted for excess zeros, the ZIP model, had the best fit to the data. Under this model, the type A transmission kernel function had the best fit. For all models incorporation of the various spatial kernel functions, which captured the fact that FMD is more likely to spread between farms located nearby, considerably improved model fit. $\bar{D}$ values for the four considered distributions and the alternative spatial kernel functions are in Table 2.

INSERT TABLE 2 APPROXIMATELY HERE

Under the best model - the ZIP model with the kernel function A - the important predictors were those associated with the epidemic component: the infection rate is only linked to the spatial kernel, whereas the zero part is linked – in addition to the spatial kernel – with the number of cattle and sheep in the farms, finding strong indications that the higher the number of animals within each herd the more likely that it gets infected. Posterior medians (and the corresponding 95% credible intervals) of the statistically significant estimated coefficients are summarized in Table 3. In addition to the important predictors associated with model's covariates, we also report parameter ϕ of the OU process, which is an indicator of the rate at which infections return to the mean infection rate after days with large number of cases. The value of 0.035 for the estimated parameter ϕ of the OU process is low [21] and hence indicative of a slow reduction in the spread of the infection and of the inability of control measures taken to reduce the infection spread

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rapidly. This is also expressed by $\exp(-\phi) = 0.965$, that resembles the autocorrelation parameter of an AR(1) process.

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INSERT TABLE 3 APPROXIMATELY HERE

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Finally, Figure 2 depicts the temporal endemic/epidemic decomposition of μ_t, i.e. the contribution of either the epidemic ($\mu_{t\_endemic}$) or endemic ($\mu_{t\_epidemic}$) component of the model during the 72 days of the epidemic to the number of newly infected farms.

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INSERT FIGURE 2 APPROXIMATELY HERE

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3.1 Model posterior predictive checking

The predictive accuracy of our modeling has been evaluated by comparing the predicted IPs obtained by the best selected model specification with the actual IPs. The quality of the fit of the best model (ZIP) as suggested by the selection criterion was satisfactory, as revealed by Figure 3, where occurrence predicted values are plotted along with the observed occurrence data.

In particular, the distribution of infected cases seems to closely match the distribution of predicted occurrences.

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INSERT FIGURE 3 APPROXIMATELY HERE

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4. Discussion

FMD is not endemic in Greece. However, the country's prefecture of Evros has been considered as potential gateway of introduction and spread of FMD in Europe ([5], [22]) due to its geographical location being the natural border of Europe

with Asia (Turkey). Hence, the need to provide all necessary tools for the effective and early control of sporadically occurring epidemics in the region. In this paper we propose suitably chosen stochastic spatio-temporal models to describe the spread of FMD. Our modeling framework extends similar approaches on analyzing FMD data ([4], [5], [6]) in various ways. Specifically, we incorporate spatial information in the form of kernel functions and we adopt a *g*-prior approach to cope with potential multicollinearity problems associated with correlated predictors frequently met in such type of data. We show that utilizing suitable distributions for modeling excess zeros considerably improves model fit. Under this framework, the ZIP specification enabled the accurate description of the transmission of FMD in 2000 in northeastern Greece.

Few of the candidate variables were important predictors for the occurrence of FMD (Table 3). Importantly, none of the considered meteorological covariates was included in the final model. Meteorological covariates were also proved in [4] to be non-significant for FMD occurrences. This lack of importance for covariates related to the airborne spread of the disease suggests that the main route for the epidemic was direct contact between animals through short/long distances. Although there are small differences in the fit of the models with different kernels, the slightly better performance of the fat-tailed kernel (A) indicates that long distance infections are not unlikely.

Conversely, the parameters accounting for spatial information were statistically significant. This result is generally robust, in the sense that sensitivity analysis conducted by changing the durations of incubation period (from the recommended by the OIE 14day period to 1d) did not show differences in the obtained outcomes. Moreover, it has been widely proclaimed in the relevant literature

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(see [13], [23], [24]) that size of farms is a key factor in the transmission of foot-and-mouth disease as is the type of farm (e.g. cattle, sheep or mixed). The analysis of the current FMD epidemic data confirms this evidence and both the size and type of farms were important. Indeed, cattle (and, to a lesser extent, sheep) density is a statistically important predictor for the excess zero occurrence of the disease in agreement with the findings of [13], [25] and [26], who have shown that infectiousness increases as the number of animals (cattle) in the infected farms increase. Further, in line with our findings [7] reported that individual cows were more likely to transmit FMD and also likely to be more susceptible to FMD than individual sheep.

The increased significance of cattle in FMD spread, when compared to small ruminants such as sheep/goats can be attributed to several reasons. Clinical signs of FMD are more easily detectable to cattle than sheep, where signs of the disease are very difficult to be detected [27]. Generally, the dairy breeds of Europe, such as those of Evros prefecture, are characterized by severe clinical signs after infection with FMD virus, contrary to the typical breeds in Asia or Africa, where the signs are less obvious. Also, most frequent clinical examinations on organized cattle farms or the larger quantities of virus shed by cattle in their close environment may be associated with the relative importance of cattle in terms of spreading the disease, in comparison to sheep/goat farms. [28] stresses the high infectiousness of cattle during the 2000 epidemic in Evros accompanied by the fast transmission of disease from animal to animal. Another characteristic of the specific Evros epidemic, is that signs of foot-and mouth infections were mostly detected in cattle. Only a few cases of infections on sheep have been reported to the authorities. Hence, this specific outbreak was mainly driven by epidemic rather than endemic factors as is generally expected for the FMD

case. The latter implies that the disease transmission occurred mainly due to animal-to-animal contacts rather than survival of FMD virus in contaminated environments and spread of the infection through indirect contact. From Fig. 2 it is clear that the course of the infection rate is almost similar to the epidemic part indicating the trivial contribution of the endemic part.

5. Conclusion

We proposed a stochastic spatio-temporal modelling approach that extends existing approaches in various ways, by accounting for the "excess zeros", incorporating spatial information and autoregressive-type stochasticity and addressing multicollinearity in the covariates. Our modeling framework considerably improved model fit on the FMD data in comparison to the previous approaches. The association between FMD occurrence and covariates suggests that farm locations, as well as the type and size of the infected farms (epidemic component) is significant for the spread of an FMD epidemic rather than meteorological covariates (endemic component). Our modeling approach could be readily applied to other infectious diseases, thus providing insights to government agencies, and all those involved in the livestock industry for the prevention for acute epidemics such as FMD.

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Tables

Table 1. Summary of spatial kernels $K(d_{k\ell}, \Theta_K)$ included in spatio-temporal models.

NOTATION	$K(d_{k\ell}, \Theta_K)$	Θ_K	Reference
A	$\left(1+\dfrac{d_{k\ell}}{\alpha}\right)^{-c}$	(α, c)	*Chis-Ster and Ferguson (2007)*
B	$\exp\left\{-\left(\dfrac{d_{k\ell}}{\alpha}\right)^{c}\right\}$	(α, c)	*Keeling et al. (2001)*
C	$\exp\left\{-\left(\dfrac{d_{k\ell}}{\alpha}\right)^{c}\right\}+r$	(α, c, r)	*Diggle (2006)*
D	$\alpha\exp\left\{-\alpha d_{k\ell}\right\}$	α	*Szmaragd et al. (2009)*
E	$\dfrac{a}{\sqrt{\pi}}\exp\left\{-\alpha^{2}d_{k\ell}^{2}\right\}$	α	*Szmaragd et al. (2009)*
F	$\dfrac{a}{4}\exp\left\{-\alpha^{1/2}d_{k\ell}^{1/2}\right\}$	α	*Szmaragd et al. (2009)*

Table 2. Mean deviance ($\bar{D}$) for the various fitted models, and mean deviance ($\bar{D}'$) for the models that do not include spatial kernels (DIC in the parentheses).

Kernel	Distribution			
	P	NB	ZIP	ZINB
A	144.1	146	**119.5**	122
	(169.6)	(170.7)	---	---
B	146	149.3	121.3	124.7
	(170.8)	(176.2)	---	---
C	147.5	151.4	120.2	123.1
	(171.8)	(177.8)	---	---
D	144.2	150.8	127.6	132.5
	(169.8)	(177.5)	---	---
E	143	148.7	128.7	132.1
	(167.5)	(174.6)	---	---
F	144.2	148.3	128.4	131.5
	(168.7)	(175.8)	---	---
$\bar{D}'$ (w/o kernel)	154	171.86	143.3	168.6
	(179.4)	(197.3)		

Table 3. Posterior medians and corresponding 95% credible intervals of parameters (ZIP model).

Parameter	Infection rate part	Zero part
α (kernel parameter)	1.549 (0.06, 6.308)	2.509 (0.286, 7.191)
c (kernel parameter)	2.982 (0.413-7.683)	2.454 (0.519-6.436)
number of cattle	---	-6.66 (-10.63, -3.605)
number of sheep	---	-1.576 (-3.9, -0.395)
ϕ (rate of OU process)	0.035 (0.011, 0.241)	---
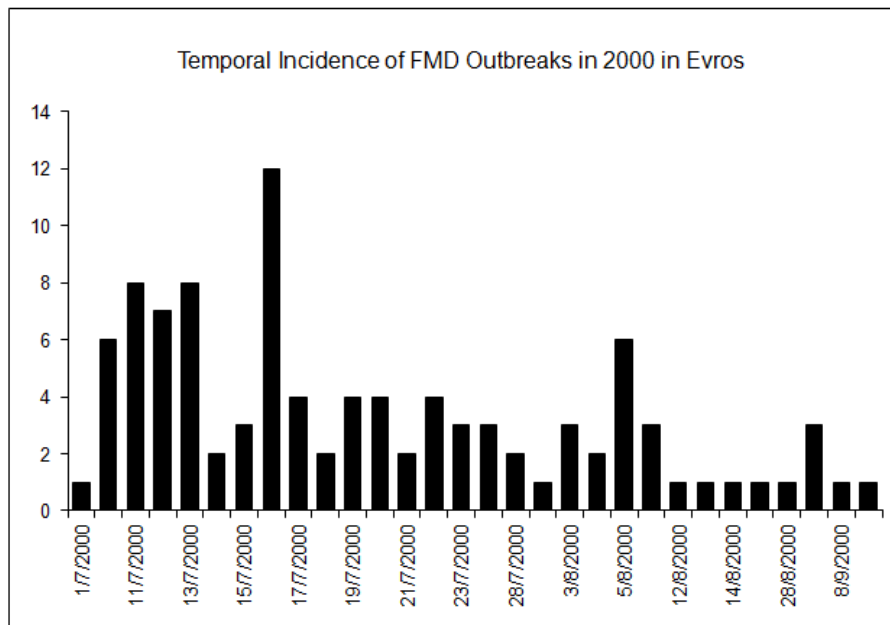
Figure 1. Temporal incidence of Foot-and-Mouth disease outbreaks in 2000 in Evros Prefecture, Greece.
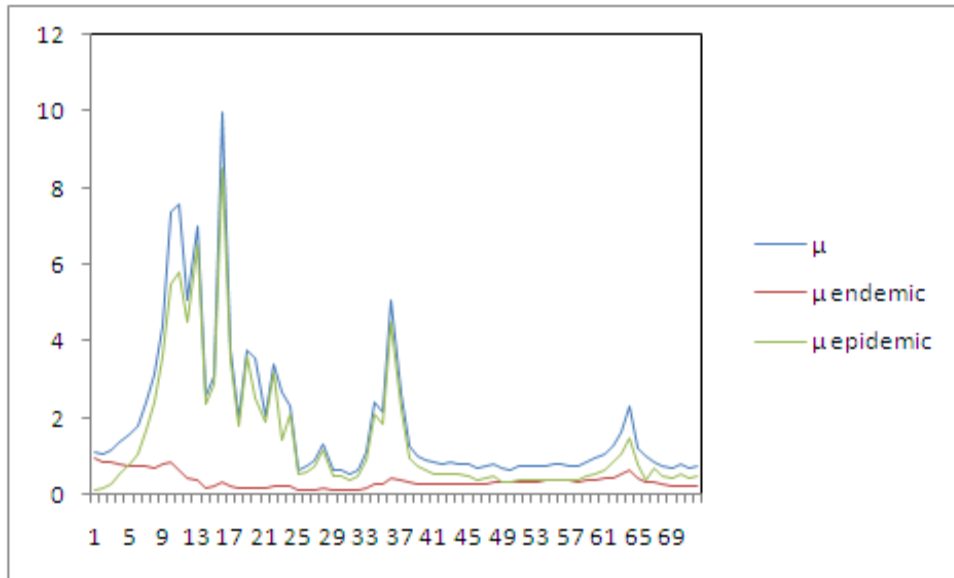
Figure 2. Temporal evolution of μ_t, $\mu_{t\_endemic}$ and $\mu_{t\_epidemic}$ during the 2000 FMD outbreak.
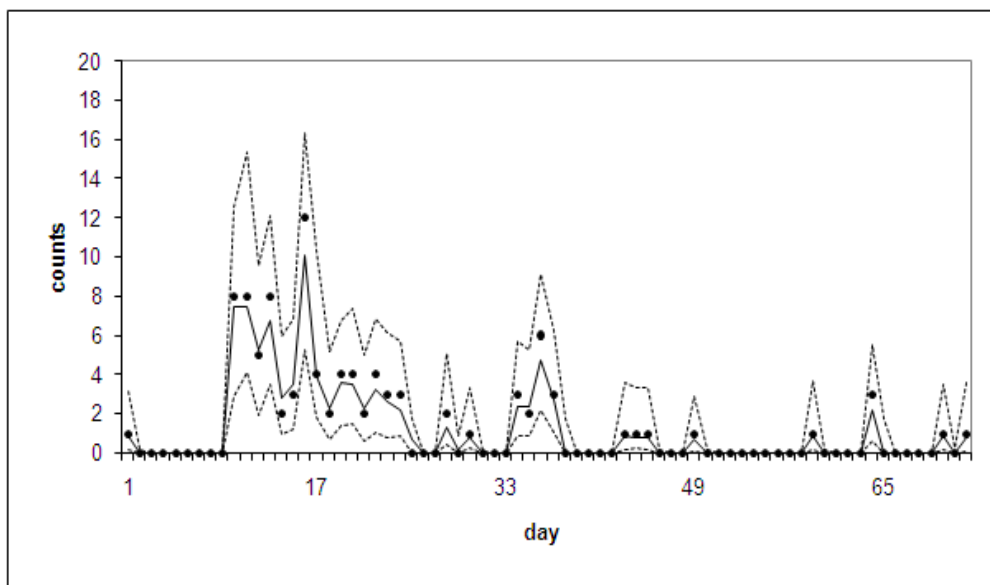

Figure 3. Predicted vs observed counts for the ZIP model (•, observed; - - - - -, 95% probability intervals; _____, predicted).

Supplementary Material

Table S1. Summary of candidate variables for inclusion on the final model.

Covariates*	min	median	max
average temperature (°C)	16.15	26.35	32.2
average maximum temperature (°C)	21.6	31.5	39.8
average minimum temperature (°C)	8.8	17.5	28.4
average relative humidity (%)	32.67	48.835	82
average wind speed (knots)	2.63	5.315	11.63
average soil temperature (at a 10 cm depth) (°C)	21.15	28.85	39.25
number of cattle	0	10	532
number of sheep	0	0	471

(*) Source: Greek National Meteorological Service

The WinBUGS program for the best selected model (ZIP model with g-prior, SM-OU specification, kernel (A))

```
model{

O[1] ~ dpois(lambda[1])
u[1] ~ dbern(p[1])
lambda[1] <- (1 - u[1]) * mu[1]
log(mu[1]) <- s + b[9]*(pow((1+(x9[1]/b[10])),-b[11]))
logit(p[1]) <-s + c[7]*x7[1]+c[8]*x8[1]+c[9]*(pow((1+(x9[1]/c[10])),-
c[11]))

for(k in 2:3129) {
D1[k]<-pow((1+(x9[k]/b[10])),-b[11])
D2[k]<-pow((1+(x9[k]/c[10])),-c[11])
                }
for(i in 2:72){
V1[i]<-sum(D1[startinds[i]:endinds[i]])
V2[i]<-sum(D2[startinds[i]:endinds[i]])

N1[i]<-sum(x7[startindn[i]:endindn[i]])
N2[i]<-sum(x8[startindn[i]:endindn[i]])

O[i] ~ dpois(lambda[i])
u[i] ~ dbern(p[i])
lambda[i] <- (1 - u[i]) * mu[i]
log(mu[i]) <-mu1[i]
mu1[i] ~ dnorm(M[i],U)
C[i]<- s + b[9]*V1[i]+ gam1*O[i-1]
M[i]<-C[i] + (log(mu[i-1])- C[i])*exp(-phi)
logit(p[i]) <-s + c[7]*N1[i]+c[8]*N2[i]+c[9]*V2[i]+gam2*O[i-1]

 }

U<-(2*phi)/(1-exp(-2*phi))

lamda<-exp(s)

for (k in 7:9) {b[k] ~ dnorm( 0, 0.0001)}
for (k in 7:9) {c[k] ~ dnorm( 0, 0.0001)}

for (k in 10:11) {b[k] ~ dnorm( 0, 0.0001)I(0,)}
for (k in 10:11) {c[k] ~ dnorm( 0, 0.0001)I(0,)}

gam1 ~ dnorm(0, 0.0001)
gam2 ~ dnorm(0, 0.0001)


for (i in 1:6)
     {
     for (j in 1:6)
          {
inverse.V[i , j]<-inprod(x[ , i] , x[ , j])
          }
      }

for (i in 1:6)
     {
```
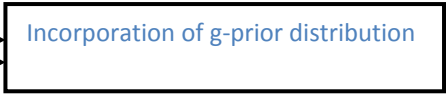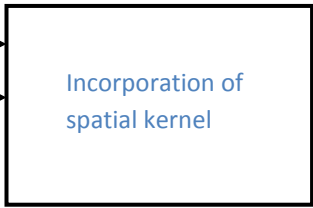
Incorporation of spatial kernel

Ornstein-Uhlenbeck process

Incorporation of g-prior distribution

```
        for (j in 1:6)
            {
prior.T[i , j]<-inverse.V[i , j]*lamda/(K/(1-K))
            }
        }

s~ dnorm( 0, 0.1)

b[1:6] ~ dmnorm( mu.beta[ ], prior.T[ , ])

for (j in 1:6)
      {mu.beta[j]<-0.0}

c[1:6] ~ dmnorm( mu.c[ ], prior.T[ , ])

for (j in 1:6)
      {mu.c[j]<-0.0}

K~dbeta(1,1)

phi<-exp(theta)
theta ~ dnorm( 0, 0.1)

 for (i in 1:72){
      x0[i]<-x[i,1]
      x1[i]<-x[i,2]
      x2[i]<-x[i,3]
      x3[i]<-x[i,4]
      x4[i]<-x[i,5]
      x5[i]<-x[i,6]
      x6[i]<-x[i,7]}

}
```

Incorporation of g-prior distribution

Ornstein-Uhlenbeck process