Published in final edited form as:
Avian Dis. 2011 March ; 55(1): 35–42.

Ongoing Estimation of the Epidemic Parameters of a Stochastic, Spatial, Discrete-Time Model for a 1983–84 Avian Influenza Epidemic

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SUMMARY

We formulate a stochastic, spatial, discrete-time model of viral "Susceptible, Exposed, Infectious, Recovered" animal epidemics and apply it to an avian influenza epidemic in Pennsylvania in 1983–84. Using weekly data for the number of newly infectious cases collected during the epidemic, we find estimates for the latent period of the virus and the values of two parameters within the transmission kernel of the model. These data are then jackknifed on a progressive weekly basis to show how our estimates can be applied to an ongoing epidemic to generate continually improving values of certain epidemic parameters.

Keywords

epidemics; estimators; avian influenza; parameter estimation; mathematical models

Mathematical models of infectious disease transmission are useful adjuncts to all the other arguments that go into selecting one control strategy rather than another (13). For those infectious diseases of farmed animals that are dealt with by stamping out and/or reactive vaccination and specifically for those diseases such as avian influenza (AVI), for which all of the animals on a farm are treated identically regardless of their actual infection status, the most appropriate unit of infection is the farm itself. This is not only appropriate but also convenient because much of what we know about risk factors for infection and/or transmission is derived from studies in which populations on farms are the unit of study. For example, in the case of AVI, we know that flock size and proximity to an infectious flock are clear risk factors for infection (18,19,28,35). It is also typical, in the event of an outbreak of a serious epidemic in domestic animals, to implement a movement ban to restrict long-distance transmission, although this may not have much effect on certain aspects of short-distance transmission, such as the windblown or fomite carriage of pathogen (21). It is for this reason that the straight-line distance between infectious and susceptible premises, rather than the shortest or quickest road distances between premises, is the typical and most useful measure of proximity between premises in risk-factor studies (25).

The model presented here is a stochastic, spatial model of highly pathogenic AVI in which the risk of transmission between the sets of contiguous premises housing the flock(s) is assumed to be a function of flock size and the straight-line distance between susceptible and infectious premises. Such models are ideally suited to evaluating control strategies that are

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by their very nature spatially heterogeneous; for example, the ring culling that was implemented during the highly pathogenic AVI (H7N3) outbreak that occurred in the Fraser Valley of British Columbia, Canada (1). The United States has been fortunate in that it has not experienced a serious highly pathogenic AVI outbreak for over 25 yr. However, this also means that we have little opportunity to be able to estimate the length of the latent period and the parameter values that determine the rate of spatial spread of the infection that would apply in the United States and that are crucial to any attempt to define an optimal strategy, given a particular definition of success (4,7,9,17,32). Even if there had been a recent outbreak, it is likely that the proper constraints of confidentiality would limit what data would actually be available in the public domain.

In a previous article (23) we formulated three methods for determining the kernel parameters of a stochastic, spatial, premises-based, discrete-time "Susceptible, Exposed, Infectious, Recovered" (SEIR) model of viral epidemics from synthetic data sets that varied with respect to their degree of completeness. Our purpose was to deal with the problem of how to determine model parameters based on the varieties of data that are actually available during and after an epidemic. The very damaging 1983–84 Pennsylvania outbreak of highly pathogenic H5N2 AVI (16) illustrates the task nicely. The original outbreak reports appear to have been lost. All that exists today are the (slightly discrepant) reports of the number of new cases detected each week over a 26-wk period in 1983–84 (6,11). Although the extent and epicenter of the epidemic are known to have been in the southeastern portion of Pennsylvania, no data regarding the then-numbers of poultry farms, their geometric locations, and their sizes are currently available in the public domain. In this article we investigate whether the method we termed Method 2 in our previous work is able to characterize the relevant model kernel for the 1983–84 AVI outbreak in Pennsylvania.

In the Methods section we first present the mathematical model we used to describe the 1983–84 epidemic and the unknown parameters values within it that we wish to determine. Then we describe the synthetic geographic data we used for the locations of poultry farms in Pennsylvania and the number of birds that each contains. We next exhibit the epidemiologic data collected during the 1983–84 epidemic and describe the procedure we used to estimate the epidemic parameters based on Method 2 of our previous article. Finally, in the Results section we present our numerical results and also show how these values could also have been estimated on an ongoing weekly basis wherein we obtain increasingly better values as more data are collected.

METHODS

Our model

The model for an AVI epidemic that we shall use can also describe other animal viral epidemics, such as those due to exotic Newcastle disease, infectious salmon anemia, and foot-and-mouth disease (FMD) (14). It is a SEIR model, so that each bird first goes through a susceptible stage, then passes through a latent period (in which it has been infected with the virus but is not yet infectious), and then passes through an infectious period, after which it is considered to manifest a recovered stage. The infectious period may come to an end naturally or through culling, and the recovered stage may be one in which the birds are alive but no longer infectious, have died naturally, or have been culled. We assume that the duration of an epidemic is such that any live recovered birds do not again become susceptible.

We follow the usual conventions for stochastic, spatial models of between-premises transmission: 1) the location of each set of farm premises is given by a single point on the map; 2) each set of infected premises has the same latent period and the same infectious

period as every other set; and 3) the infectiousness of a set of premises of a given size (defined by the number of animals on the farm) does not change with time (7,14,15,23,26,29,30,31,32,33,34). The robustness of this last assumption has been examined in detailed by Savill *et al.* (24) for the specific instance of FMD. They concluded that in the majority of cases there is no evidence of changing infectiousness over the infectious periods and that allowing infectiousness to change over time in such models does not significantly improve the fit of the model. AVI in farmed animals is frequently controlled by quarantining and then culling all the animals on the farm. Thus, the period for which a set of premises containing infectious animals presents a risk to other premises is not determined by the infectious period for any given animal but rather by the time it takes to detect the infection and quarantine or cull the premises. This is usually known with a fair degree of certainty, and for our calculations we take this period as 1 wk. The approximation represented by assumption 3 is then simply a consequence of ignoring the within-premises transmission dynamics and is equivalent to stating that the risk that infectious premises present to other premises does not change during the period of infectiousness.

Case control studies have repeatedly found that flock size and the proximity of one flock to the next are important determinants of the probability of infection (18,19,28,31,35). These observations underpin the manner with which we represent transmission in the model. Other factors, such as management, poultry species, and the age of the flock, are less frequently identified as risk factors for infection, and our model ignores them. In this we follow Von Neumann's precept that for a model with many parameters it is easy to find values of the parameters that fit given experimental data, but it is not likely that these parameters will fit new data (8).

Our model is a stochastic one and is based on the probability that an infectious premises will infect a susceptible premises within a certain time period, which we take as 1 day. To estimate this probability, we assume that the probability that one infectious bird will not infect one susceptible bird a distance d miles away in 1 day is a function only of d, say, $p(d)$.

Our probability function, $p(d)$, starts at zero (when the distance between two birds is zero and transmission of the virus within 1 day is certain) and then monotonically increases to one as the distance tends to infinity (Fig. 1).

It is more common in mathematical modeling of epidemics to do calculations with a transmission kernel $K(d)$ rather than with the probability function $p(d)$, where the connection between the two is given by

$$K(d) = -\ln(p(d)) \quad \text{and} \quad p(d) = e^{-K(d)}. \quad (1)$$

The transmission kernel has the qualitative behavior given in Fig. 2: it begins at infinity at $d = 0$ and decreases monotonically to zero as d goes to infinity.

There are many functions that exhibit the behavior of such a transmission kernel, but the one we choose is a power-law function of d given by

$$K(d) = \left(\frac{\delta}{d}\right)^{\rho}, \quad (2)$$

where δ and ρ are two epidemic parameters, the values of which we are to determine. This is the simplest type of function having the desired properties and is very commonly used in mathematical modeling.

With this transmission kernel, the probability, *P(d)*, that one infectious bird will infect a susceptible bird a distance d miles away in 1 day is

$$P(d) = 1 - e^{-\left(\frac{\delta}{d}\right)^{\rho}}. \quad (3)$$

As can be seen in Fig. 3, this function decreases monotonically from 1 to 0 as d goes from 0 to infinity. The parameter δ is a distance-scaling factor; specifically, it is the distance at which the probability of infection is $1 - 1/e$, or 0.6321 …, for any value of the other parameter, ρ. The value of ρ determines how quickly the transition from one to zero takes place. A small value of ρ leads to a gradual transition, while a large value leads to a sudden transition at a distance δ.

A very large value of ρ would describe a virus that spreads with near certainty to all farms within distance δ in 1 day, but not any further. It is thus almost a deterministic model in which all susceptible farms within distance δ of an infected farm become infected within the 1 day of infectiousness of the infected farm. However, our experience with this model for many different viral epidemics indicates that ρ is usually rather small, typically between 2 and 3.

Next, suppose the i^{th} farm in the community is infectious and contains N_i birds and that the j^{th} farm in the community is susceptible and contains N_j birds. Then, if d_{ij} is the distance between the two farms, the probability that no infectious bird on the i^{th} farm will infect any bird in the j^{th} farm is given by

$$p_{ij}(\delta, \rho) = \exp\left(-N_i N_j \left(\frac{\delta}{d_{ij}}\right)^{\rho}\right). \quad (4)$$

This expression follows from basic probability theory under the assumption that infectious birds infect susceptible birds independently of each other.

The next step is to take into account all farms that are infectious in a particular day and determine the probability that none of the birds on the infectious farms will infect any of the birds on a susceptible farm. If we let *A(k)* be the set of indices of those farms that are infectious at the beginning of the k^{th} day of the epidemic, then

$$\prod_{i \in A(k)} \exp\left(-N_i N_j \left(\frac{\delta}{d_{ij}}\right)^{\rho}\right) \quad (5)$$

is the probability that the j^{th} farm (if susceptible) will not be infected during that day. Hence, the probability that the j^{th} farm will be infected during the k^{th} day is 1 minus this value, or

$$P_{jk}(\delta, \rho) = 1 - \prod_{i \in A(k)} \exp\left(-N_i N_j \left(\frac{\delta}{d_{ij}}\right)^{\rho}\right). \quad (6)$$

We used the expression in Eq. 6 in a MATLAB™ program to simulate epidemics. Given an index farm for the epidemic, we can program a bookkeeping problem in which the SEIR states of all farms are determined on a day-by-day basis until the epidemic is over.

Because Eq. 6 provides only the probability of infection, each simulation that we perform is only one of many possible epidemics that begins with a given index farm. After determining the probability that a susceptible farm becomes infected, our MATLAB program generates a random number from the interval [0,1] that is used to decide whether to infect the farm or not. For example, if the probability of infection of a particular susceptible farm during a

particular day is computed as β and the random number generated is γ, then we infect the farm if $\beta > \gamma$; otherwise, we do not.

To run our computer program, we must know the following: 1) The positions of all the farms; 2) the number of birds on all the farms; 3) the index farm; 4) the duration of the common latent and infectious periods; and 5) the numerical values of the epidemic kernel parameters δ and ρ.

As mentioned earlier, we shall take the infectious period as 1 wk, which we assume is the time it takes to determine whether an infectious farm is infectious and then to cull or quarantine it. The latent period is one of the parameters we wish to estimate on the basis of the collected epidemic data, along with the kernel parameters δ and ρ. In the next section we discuss the determination of the positions and sizes of the farms and the index farm.

Our geographic data

Geographic data, specifically the locations of the farms and the number of birds each contains, are needed to run our model. Unfortunately, such information is not available in the public domain. Nevertheless, synthetic maps of the locations of farm premises with the same marginal properties of the U.S. AgCensus are available (5,31), and we use these as a surrogate for the actual map.

The 1983–84 epidemic was mainly confined to the southeastern portion of Pennsylvania, centered about the city of Lancaster. Because the index case of that epidemic is unknown, we took it to be an arbitrary fairly large farm in this region, as indicated by the white circle marker in Fig. 4.

All of the known infected farms were within 100 miles of the city of Lancaster (6), so we confined our simulations to a circular region of 100-mile radius within Pennsylvania, centered around that city. Additionally, we considered only those premises that contained more than 1000 birds, as our previous experiments with these models indicated that small or backyard flocks do not significantly affect the progress of an epidemic. The resulting 1096 farms are mapped in Fig. 5.

The numbers of birds on these 1096 farms ranged from 1000 to 654,000, and the index farm that we chose was the fifth largest farm, containing 357,000 birds. Fig. 6 is a histogram of the sizes of these 1096 farms, with the index farm pointed out.

Our epidemic data

Our basic epidemic data consist of the weekly number of newly infectious premises from September 1983 to March 1984, as reported in Buisch *et al.* (6) in 1984. For the first 5 wk, either no or one infectious premises was reported. Consequently, we took the sixth reported week, beginning October 2, 1983, as the first week of our epidemic for our simulations. In tabular form, our data then consist of the 26 numbers in the second row of Table 1, which represent the number of newly infectious premises in each of the 26 wk of the epidemic. It should be emphasized that these 26 numbers represent the only epidemic data that we used in determining our estimates for the latent period and the two transmission kernel parameters.

Our estimator

Given the geographic data (which includes the location of the index farm) and values of the duration of the latent period, call it m, and the two kernel parameters δ and ρ, we may run our model to obtain a day-by-day simulated epidemic. By running many simulations we may

gather statistical information from which to determine the expected behavior of a typical epidemic. Fig. 7 is an example of such statistical information. It shows the attack rates (the number of infected premises) of 100 simulated epidemics, ordered by size, using the arbitrary values $m = 5$ days, $\delta = 4.0 \times 10^{-6}$, and $\rho = 2.06$. We see that 25 of the 100 simulations resulted in 10 to 29 premises being infected, and 75 of the simulations resulted in 221 to 318 premises being infected. We used this typically bimodal nature of the outbreak of an epidemic to classify our epidemics as mild (≤100 infected premises) or severe (>100 infected premises). For the severe epidemics in Fig. 7, the mean attack rate was 275 premises (25.1% of all farms), with a rather small variance, as can be seen by the inset histogram.

Our objective is to obtain estimates for the duration of the latent period m and the two kernel parameters δ and ρ using only the data provided in Table 1. To this end we consider the following expression:

$$E(\delta, \rho, m) = \sum_{k=1}^{K} |Q_k - F_k(\delta, \rho, m)|, \quad (7)$$

where

Q_k = reported number of newly infectious farms on the k^{th} week of the epidemic ($k = 1, 2, \ldots, K$);

$F_k(\delta, \rho, m)$ = the expected number, based on our model, of newly infectious farms on the k^{th} week of a severe epidemic if the latent period is m wk.

This expression [$E(\delta, \rho, m)$] is thus a measure of the difference between the actual number of newly infectious farms and the expected number for specified values of m, δ, and ρ. We shall use this expression as our estimator for the optimal values of these three parameters; specifically, our optimal values will be those values that minimize this expression.

In Eq. 7, $K = 26$, the length of the epidemic in weeks, and the 26 values of Q_k are given in Table 1. The expression $F_k(\delta, \rho, m)$ is a mathematical expectation and so has an exact value for each set of values of k, δ, ρ, and m. However, it is computationally impossible to determine its exact value, and so we resorted to numerical methods to approximate it. Specifically, for a given set of values of δ, ρ, and m, we ran 100 simulations of epidemics, beginning with our chosen index farm, and took the average number of newly infected farms on each of the K wk of the severe epidemics among the 100 simulations as our approximation of $F_k(\delta, \rho, m)$ for $k = 1, 2, \ldots, K$. Notice that our simulations are run on a daily basis, while the expected number of farms is counted on a weekly basis, since data were provided only on a weekly basis.

We remark that we only used simulated severe epidemics (those that resulted in more than 100 infected farms) in determining the expected values given in $F_k(\delta, \rho, m)$. This is because we know that the actual epidemic was severe, so we are only comparing it to severe epidemics that arise from our simulations.

We mention that we initially tried a more general expression for $E(\delta, \rho, m)$, namely

$$E(\delta, \rho, m) = \sum_{k=1}^{K} |Q_k - F_k(\delta, \rho, m)|^p, \quad (8)$$

where p is some positive number. A common choice is $p = 2$ so that $E(\delta, \rho, \text{m})$ has the interpretation of the least-squares error between the set of the actual numbers of weekly

newly infectious farms and the expected numbers. However, a least-squares error criterion arises only when the error in question is known to have a normal distribution, and we have no reason to suspect that is the case in our model. In addition, we tried the values $p = 1, 2, 3, 4$, and 5 in our original investigations using synthetic epidemics generated from the model itself, and the best estimation of the error using Eq. 8 arose with $p = 1$.

With our choice of $p = 1$, $E(\delta, \rho, m)$ has the interpretation of the cumulative weekly difference between the actual and the expected number of newly infectious farms. If this difference if zero, then we have an exact weekly match between the actual and the expected newly infectious farms. In addition, the following inequality,

$$\begin{aligned} \left| \sum_{k=1}^{K} Q_k - \sum_{k=1}^{K} F_k(\delta, \rho, m) \right| &= \left| \sum_{k=1}^{K} (Q_k - F_k(\delta, \rho, m)) \right| \\ &\leq \sum_{k=1}^{K} |Q_k - F_k(\delta, \rho, m)| \quad = E(\delta, \rho, m), \end{aligned} \tag{9}$$

tells us that the difference between the actual attack rate $\Sigma_{k=1}^{K} Q_k$ (the total number of premises infected during the epidemic) and the expected attack rate $\sum_{k=1}^{K} F_k(\delta, \rho, m)$ is less than $E(\delta, \rho, m)$. Consequently, by minimizing $E(\delta, \rho, m)$, we are also making the difference between the actual and expected attack rates small.

RESULTS

Minimizing $E(\delta, \rho, m)$ over all possible values of δ, ρ, and m led to the following optimal values of these three parameters: 1) $\delta = 9.5 \times 10^{-6}$ miles (kernel parameter); 2) $\rho = 2.20$ (kernel parameter); and 3) $m = 4$ days (duration of latent period). In addition, the actual attack rate was 286 infected farms (26.1% of the 1096 farms), and the estimated attack rate was 270 farms (24.6% of the 1096 farms).

We minimized the function $E(\delta, \rho, m)$ by brute-force methods, namely, by evaluating $E(\delta, \rho, m)$ over a large grid of values of δ, ρ, and m and by choosing the set of values for which $E(\delta, \rho, m)$ was minimal. We did this because our use of 100 simulated epidemics to estimate $F_k(\delta, \rho, m)$ introduced noise and false local minima into the function $E(\delta, \rho, m)$. These false local minima resulted in more sophisticated minimization algorithms (such as Nelder-Meade search (20)) getting stuck at these false local minima rather than converging to the true global minimum.

Fig. 8 shows our results in graphic form. The black curve is a graph of the weekly number of newly infectious premises, as given in Table 1. The light gray curves in the background are the analogous graphs for the severe epidemics among the 100 simulated epidemics using the optimal values of the epidemic parameters. The heavy dark gray curve is the average of these curves, which we take as the approximation to the expected number, $F_k(\delta, \rho, m)$, of newly infectious farms. The optimal values of the three epidemic parameters can be thought of as those values for which the area between the black curve and the dark gray curve is as small as possible.

In Fig. 9 we have plotted the 1096 farms in the epidemic region with shades of gray according to their probabilities of being infected during an epidemic. These probabilities were determined from the severe epidemics among 100 simulated epidemics that we ran using the optimal values of the epidemic parameters that we found. For each of the 1096 farms, the probability of infection was approximated by the fraction of times that the farm was infected over the severe epidemics. If we had information about which farms in the

1983–84 epidemic were actually infected, we could evaluate the accuracy of our probabilities. Unfortunately, the information is not available.

Our estimates for the epidemic parameters were obtained using the full data over the 26 wk of the epidemic. However, the same methods can be applied to generate weekly estimates for the parameters as new weekly data are collected during the epidemic. Mathematically, it is simply a matter of setting K in Eq. 7 equal to the number of weeks for which data have been collected.

In Table 2 we display estimates of the epidemic parameters δ and ρ and the attack rate using the data up to and including the data obtained during the seventh, 13th, 20th, and 26th weeks of the epidemic. In all cases, the optimal estimate for the latent period was 4 days. The estimates for the attack rate are particularly interesting since they can be compared with the reported attack rate of 286 farms. As previously mentioned, it is expected that the estimated attack rate should be in good agreement with the reported attack rate if the data over the entire 26 wk of the epidemic are used, but the estimated attack rate even after only 7 wk, 221 farms, is in good agreement with the reported attack rate.

In Figs. 10-12 we present graphic representations of our estimates based on using data up through that collected during the seventh, 13th, and 20th weeks of the epidemic. Fig. 10 shows the reported data on the newly infectious farms up to the seventh, 13th, and 20th weeks, and the dark gray curves are the corresponding estimates for all 26 wk. Fig. 11 displays probability-of-infection maps, similar to Fig. 9, based on the partial weekly data. Finally, Fig. 12 graphs the ongoing estimates of the attack rate based on the data from the third through the 26th week.

Notice that although the epidemic peaked at about the 10th week, the estimates that we obtained using the data up to the seventh week already gave results very close to those obtained using data for all 26 wk of the epidemic. This is important because being able to estimate the length of the latent period and the numerical values of the kernel parameters that determine the rate of spatial spread of the infection is crucial to any attempt to define an optimal strategy, given a particular definition of success (4,7,9,17,32).

DISCUSSION

We have shown how weekly data on the number of newly infectious farms can provide satisfactory estimates for the epidemic parameters of a mathematical model of the epidemic. Although we used a very specific exemplar (highly pathogenic AVI H5N2), the method we presented can be modified to fit a large number of mathematical models containing parameters that need to be determined. Nevertheless, because the parameter estimates presented here are the only available estimates for a highly pathogenic AVI outbreak in the United States, it is worth exploring the extent to which they may be useful in and of themselves.

We began with the statement that models are useful adjuncts to all the other arguments that go into selecting one control strategy rather than another. Similar models of AVI in Europe have also been useful in the creation of risk maps for infection (4) and for providing insights into the role of backyard flocks (3). However, we acknowledge that there is a continuing debate between the modeling community and those whose responsibility it is to actually implement the control strategies. To some extent this tension is explicable in terms of the different definitions of success that are implicit in the activities of each group, for these definitions are frequently mutually exclusive. For example, achieving disease-free status as quickly as possible or minimizing spatial spread can rarely be accomplished using conservative strategies (13,17). Also at issue is the unresolved argument about the extent to

which blanket recommendations for control should be modulated by local veterinary knowledge (10,12). From a simply technical point of view, our model could be criticized on two counts. First, we used a synthetic map of the at-risk community, not a real one. While the map was constructed such that it had the same marginal properties as the real map, it could be argued that not knowing the actual location of the poultry premises would surely compromise any effort to use the estimated parameter values in the selection of putative control strategies. The utility of synthetic maps as the basis for agent-based models of infectious animal disease has been systematically studied (22,31). Tildesley *et al.* (31) studied the specific case of ring culling against FMD and compared the model output based upon real maps with model output based upon synthetic maps in which farms were randomly distributed over the landscape. They found that, provided the model is parameterized to match epidemic behavior, then using aggregate farm data is sufficient to "closely determine" optimal control measures. This is a useful result because the recent modification of the U.S. National Animal Identification System (2) means that there is little likelihood—in the foreseeable future—that spatial, stochastic models of disease transmission among farms in the United States will involve real maps of farm locations, except for those generated by individual research groups. There is clearly a role for synthetic maps. As for the second criticism, we have assumed that the kernel that determined the rate and extent of spatial spread of AVI was a monotonically declining function. In other words, the probability of transmission decreased with distance from an infectious set of premises. There is some empirical evidence that other kernel shapes might be more appropriate for some other diseases, especially if compliance with movement bans is poor (27). However, there is currently no evidence that this is so in the case of AVI, and parsimony dictates that we choose the simplest defensible kernel.

Our main goal was to describe a method for estimating model parameters using a rather limited data set. The method is applicable to a wide variety of model architectures, not just the one we chose. However, for the reasons just outlined we believe that the parameter values estimated from our exemplar have some value in and of themselves and may at least provide a place to start in the event of an actual outbreak of highly pathogenic AVI in the United States.

Acknowledgments

The project described was supported by award 5U01GM-076426 from the National Institute of General Medical Sciences. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of General Medical Sciences or the National Institutes of Health.

Abbreviations

AVI	avian influenza
FMD	foot-and-mouth disease
SEIR	Susceptible, Exposed, Infectious, Recovered

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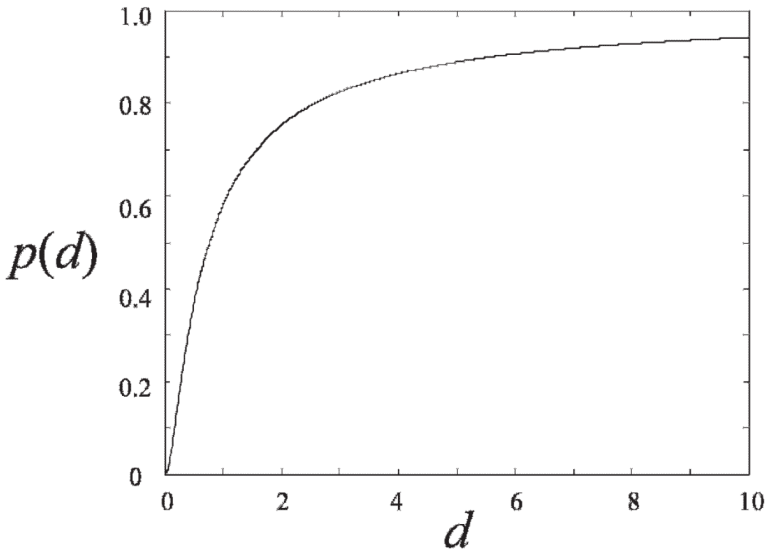

Fig. 1.
The qualitative behavior of the probability $p(d)$ that one infectious bird will not infect a susceptible bird a distance d miles away in 1 day.

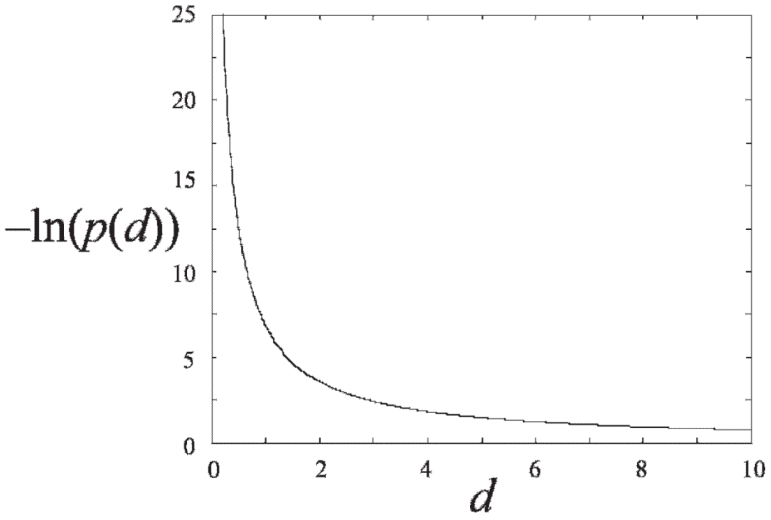

Fig. 2.
The qualitative behavior of the transmission kernel $K(d)$ [= −ln($p(d)$)].

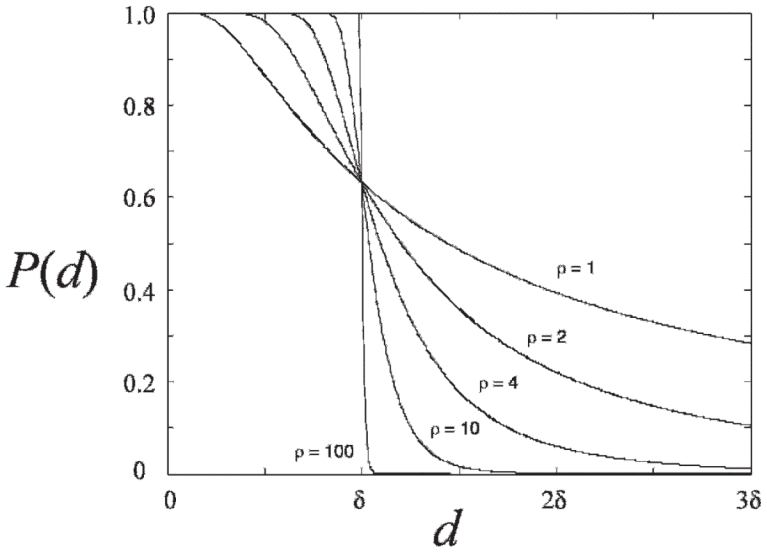

Fig. 3.

The probability *P(d)* that one infectious bird will infect a susceptible bird a distance *d* miles away in 1 day for various values of the epidemic parameter ρ.

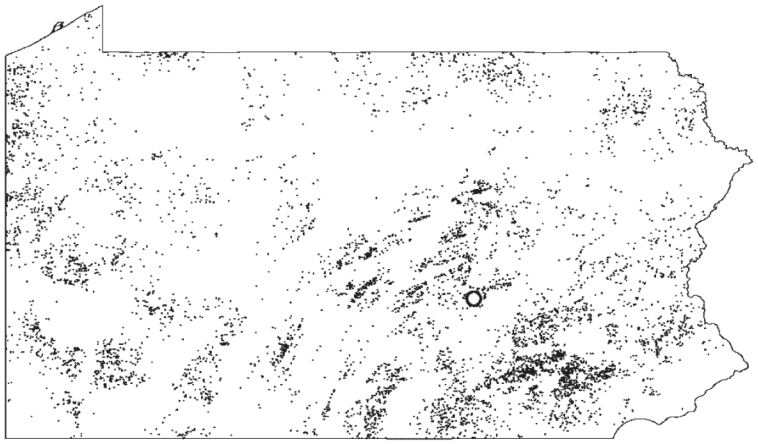

Fig. 4.
Synthetic map of 7043 poultry farms in Pennsylvania, with the index farm indicated by the white circular marker.

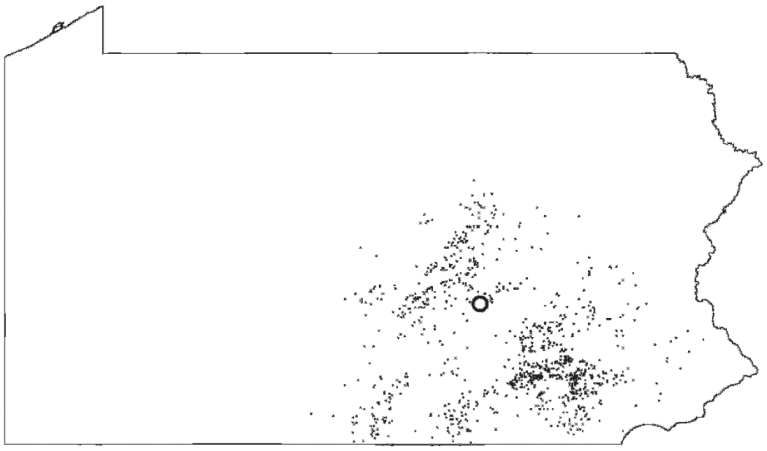

Fig. 5.
Synthetic map of the 1096 poultry farms in southeastern Pennsylvania containing more that 1000 birds that were used in our simulations. The index farm is indicated by the white circular marker.

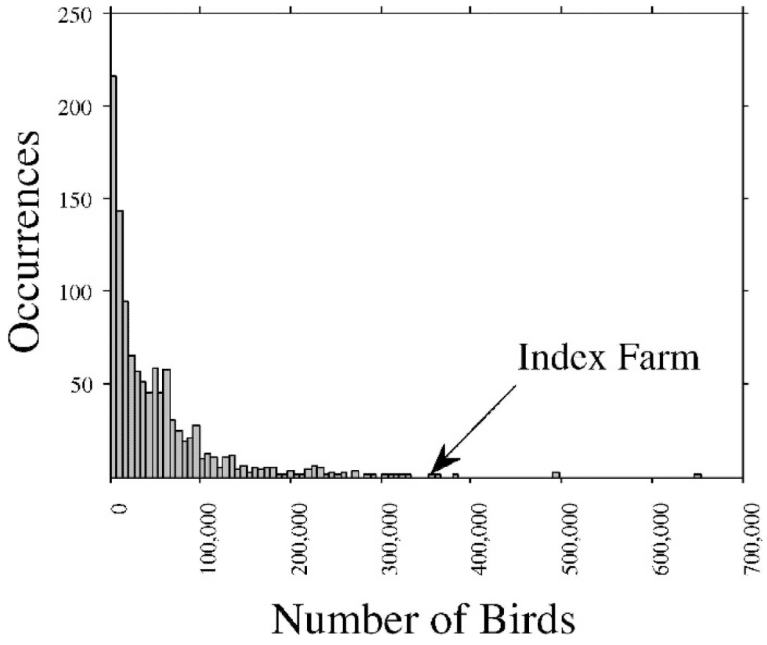

Fig. 6.
Histogram of the sizes of the 1096 farms used in our simulations.

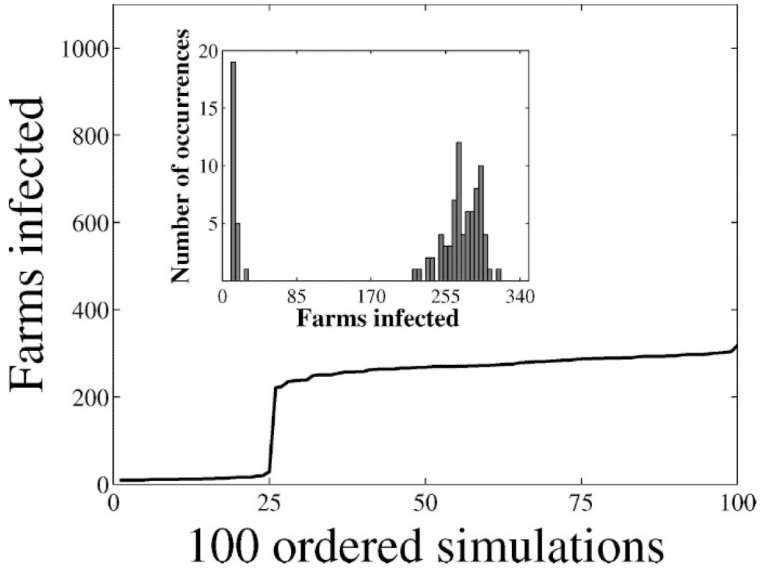

Fig. 7.
The attack rate (number of farms infected) for 100 simulated epidemics, ordered by magnitude. The inset is a histogram of the attack rates.

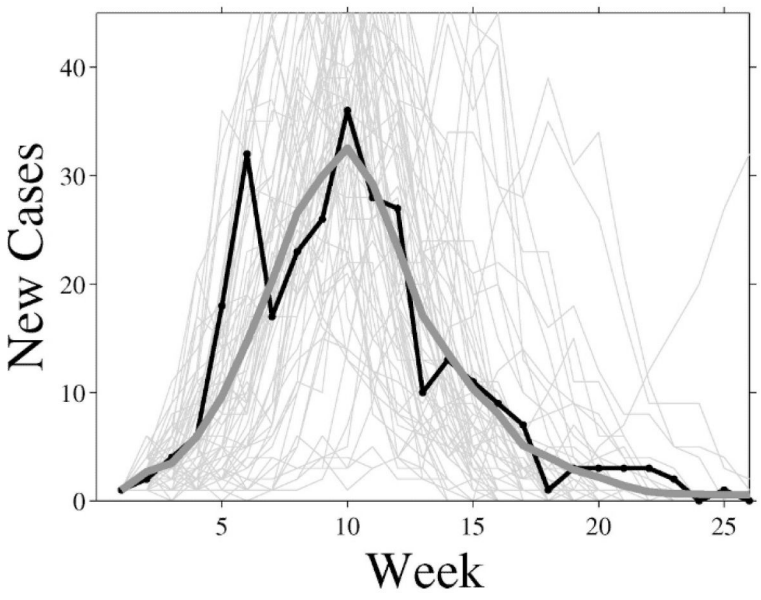

Fig. 8.
Graph of the weekly number of newly infectious premises Q_k (black curve) and the expected number $F_k(\delta, \rho, m)$ (dark gray curve) based on the optimal values of δ, ρ, and m.

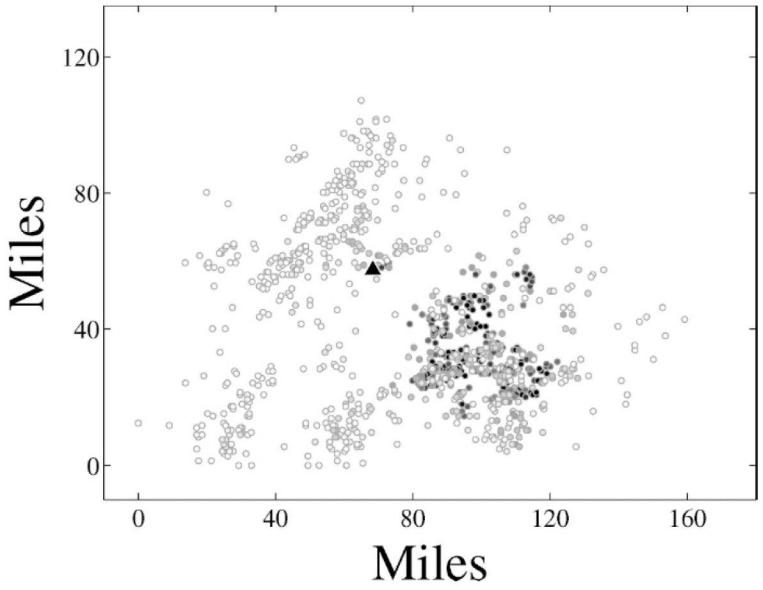

Fig. 9.
Probability-of-infection map of the epidemic region in southeast Pennsylvania. Each farm is shaded according to its probability of being infected in a severe epidemic.

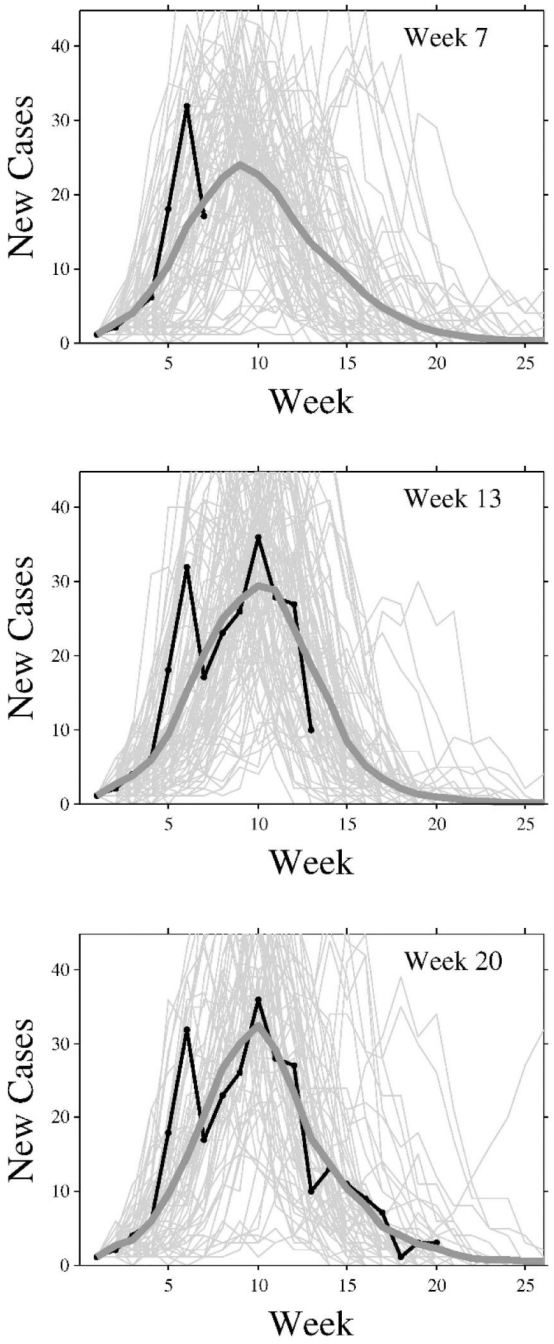

Fig. 10.
Three graphs of the weekly numbers of newly infectious farms up to the seventh, 13th, and 20th weeks (black curves) and the corresponding estimated expected number for all 26 wk of the epidemic (dark gray curves).

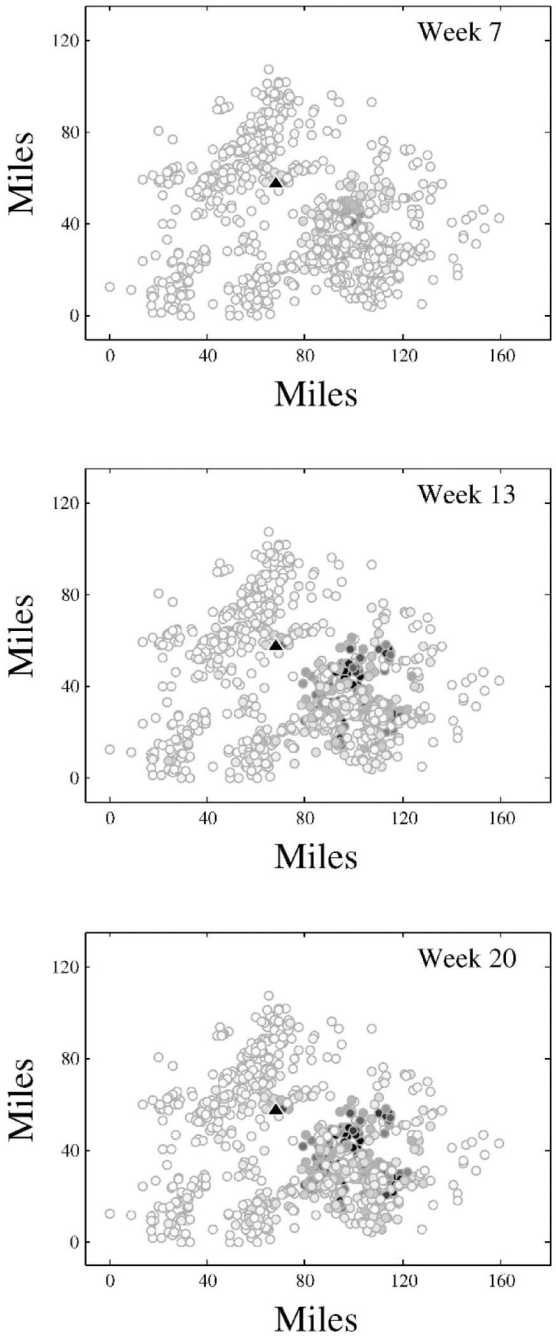

Fig. 11.
Three maps of the predicted probabilities of infection at the end of the epidemic based on the weekly numbers of newly infectious farms up to the seventh, 13th, and 20th weeks.

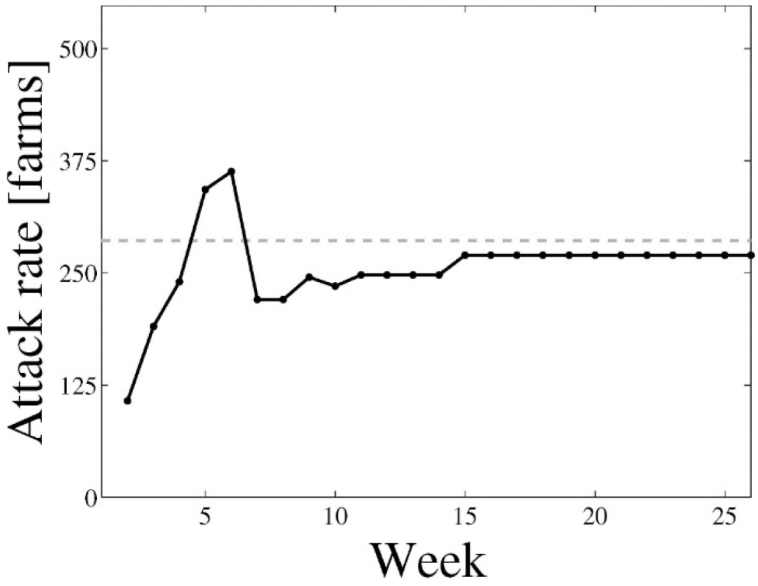

Fig. 12.
The expected attack rate (number of farms infected) of the epidemic estimated on a progressive weekly basis. The actual attack rate was 286 farms (horizontal dashed line).

Table 1

The number of newly infectious farms for the 26 wk of the epidemic.

	Week																								
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**
No.of farms	1	2	4	6	18	32	17	23	26	36	28	27	10	13	11	9	7	1	3	3	3	3	2	0	1	0

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Table 2

Weekly estimates of epidemic parameters.

Week	δ(miles)	ρ	Attack rate (No. of farms)
7	1.3×10^{-6}	1.92	221
13	1.6×10^{-6}	1.94	248
20	9.5×10^{-6}	2.20	270
26	9.5×10^{-6}	2.20	270