Dynamics of the 2001 UK Foot and Mouth Epidemic: Stochastic Dispersal in a Heterogeneous Landscape

Matt J. Keeling,[1]* Mark E. J. Woolhouse,[2] Darren J. Shaw,[2] Louise Matthews,[2] Margo Chase-Topping,[2] Dan T. Haydon,[3] Stephen J. Cornell,[1] Jens Kappey,[1] John Wilesmith,[4] Bryan T. Grenfell[1]

Foot-and-mouth is one of the world's most economically important livestock diseases. We developed an individual farm–based stochastic model of the current UK epidemic. The fine grain of the epidemiological data reveals the infection dynamics at an unusually high spatiotemporal resolution. We show that the spatial distribution, size, and species composition of farms all influence the observed pattern and regional variability of outbreaks. The other key dynamical component is long-tailed stochastic dispersal of infection, combining frequent local movements with occasional long jumps. We assess the history and possible duration of the epidemic, the performance of control strategies, and general implications for disease dynamics in space and time.

Foot and mouth disease (FMD) is a highly transmissible viral infection, which can spread very rapidly among livestock. The current major epidemic in the UK has devastated the livestock industry and caused severe economic consequences for the country as a whole. The epidemic has generated a unique data set describing the spatial spread of an infectious disease between fixed nodes, i.e., livestock farms. This, together with the availability of data on the location and livestock composition for all UK farms [collected by the Department of the Environment, Food and Rural Affairs (DEFRA)], offers an unusual opportunity to explore the impact of spatial and individual heterogeneities on the course of an epidemic and the importance of these variables for the design of appropriate disease control programs.

A key modeling decision is how to represent the local and regional spatial clustering of FMD cases (Fig. 1A), which precludes the use of standard models based on homogeneously mixed host populations (*1*). This contagion is quantified by the spatial infection kernel of the disease (*2*) (Fig. 1B); after the introduction of movement restrictions in late February, the kernel shows a high probability of local spread, with a tail of less frequent longer range "sparks" of infection. Some of the local effects caused by the clustering of infection can be modeled implicitly, with deterministic approximations (*3*, *4*). However, to explore the full spatiotemporal dynamics of the epidemic—in particular, the highly irregular behavior in the epidemic tail—we use a stochastic, spatial, individual farm–based model. The stochastic nature of transmission generates inherent uncertainty in the ability to predict events; however, in this epidemic, there are also two more systematic sources of uncertainty. First, we only have a qualitative grasp of the multifaceted nature of FMD transmission between farms (*5*–*8*); key transmission parameters must therefore be derived by fitting the model to the epidemic data. Second, there are biases and various lacunae in the epidemiological and management data used to construct the model (*9*). We summarize how these uncertainties affect our predictions in the supplementary material (*10*).

There are two important features of the outbreak, superimposed on the classical epidemic pattern (*1*) (Fig. 1C). First, there is marked variability in daily case reports—clear spikes and troughs indicate the likely importance of stochasticity in the epidemic dynamics. Second, the epidemic has a very long tail, fluctuating around four cases per day since mid-May; we believe that this is primarily attributable to the spatial nature of the infection. In a fully mixed system, the tail should decay exponentially fast because there are insufficient susceptibles to maintain the disease (*1*). However, with spatially localized infection, pockets of susceptibility remain, as well as virgin territory; these can be exploited by sparks of infection from outside the region (or "smouldering" old infections in some cases) (*10*) to produce isolated local epidemics.

Model formulation. Because of the rapid transmission of the virus between livestock in the same farm, it is reasonable to treat the farm as the individual unit (*4*, *11*–*13*), classifying each holding as either susceptible, incubating, infectious, or slaughtered. We also incorporate the heterogeneity in farm size and species composition (*13*) by allowing the susceptibility and infectiousness of farms to vary with the type and number of livestock (*14*). In principle, the necessary parameters can be estimated from the observed pattern of cases by maximum likelihood. However, we cannot rely only on this, because of spatial and temporal biases in the data (*9*). We therefore adopt a two-stage approach, generating an initial fit by maximum likelihood, then refining it by least squares fits to regional epidemics (*10*).

Given the estimated transmission parameters (*15*) and the spatial infection kernel, we model the daily course of the epidemic by Monte Carlo simulation (*16*–*18*). There is very good overall agreement between the average of the model replicates and the reported cases (Fig. 1). The observed qualitative pattern of variability is also captured by the simulations—note, though, that we do not include day-to-day environmental stochasticity in the model. The average of our simulations slightly underestimates the epidemic, after the decline in early April. The first part of this is probably due to overreporting of cases (*10*). We may also slightly underestimate the latter stages of the epidemic, probably because of small systematic secular changes in transmission not currently included in the model, such as the mid-May turnout of dairy cattle from winter housing onto pasture.

The high degree of spatial correspondence between model results and data depends on the inclusion of species and herd-size heterogeneities in transmission (*10*). The model captures the main regional foci of infection in Cumbria and Devon, although there are some departures that may be attributable to biases in the data (*9*) or local heterogeneities. Rigorous statistical assessment of the spatial fit is complicated by farm-level variation between simulations. The numerical simulations from 23 February to August capture the overall shape of the epidemic. Although this is not an independent comparison (because the parameters are estimated from the fit), the model's ability to capture the shape, spatial distribution, and variability of the epidemic is encouraging.

Heterogeneities in transmission. A wide range of heterogeneities affect the dynamics of this epidemic; the most notable is the spatial aggregation of cases (Fig. 1A).

[1]Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, UK. [2]Centre for Tropical Veterinary Medicine, University of Edinburgh, Easter Bush, Roslin, Midlothian EH25 9RG, UK. [3]Department of Zoology, University of Guelph, Guelph, Ontario N1G 2W1, Canada. [4]Veterinary Laboratories Agency, New Haw, Addlestone, Surrey KT15 3NB, UK.

*To whom correspondence should be addressed. E-mail: matt@zoo.cam.ac.uk

The localized spread of the disease (as defined by the kernel; Fig. 1B) markedly reduces the reproductive ratio of the disease, R, because of a rapid depletion of susceptibles in infected regions (*3*, *4*, *19*). Superimposed on this spatial contagion, we find strong evidence for both the farm-size and host species heterogeneities noted above (*2*, *15*). Large farms are considered to be more susceptible to the disease, and this is supported by a decrease in the average size of infected farms during the observed epidemic and in simulations (Fig. 2A). If large farms are also assumed to be more infectious, their infection and subsequent removal early in the epidemic will act to reduce further the reproductive ratio R.

The 2001 FMD epidemic has been primarily confined to cows and sheep (*20*). In agreement with epidemiological observations (*20*), we find that cattle transmit the disease slightly more than sheep and have an order of magnitude more effective susceptibility. The species parameters subsume innate biological variations in infectiousness and susceptibility with potential differences in contact rate with infection. The much greater effective susceptibility of cattle may therefore reflect species differences in contact rates between livestock species and humans/vehicles as much as, or even more than, biological susceptibility. Despite the difficulties in parameter estimation, biases in the data, and regional variability (*10*), it is clear that, per capita, cattle have contributed far more than sheep to the spread of FMD (*10*). This, however is balanced by a much greater population of sheep in British farms. As with farm size (Fig. 2A), the variation in overall infection rate with farm composition is readily apparent in the raw census and epidemic data (Fig. 2B).

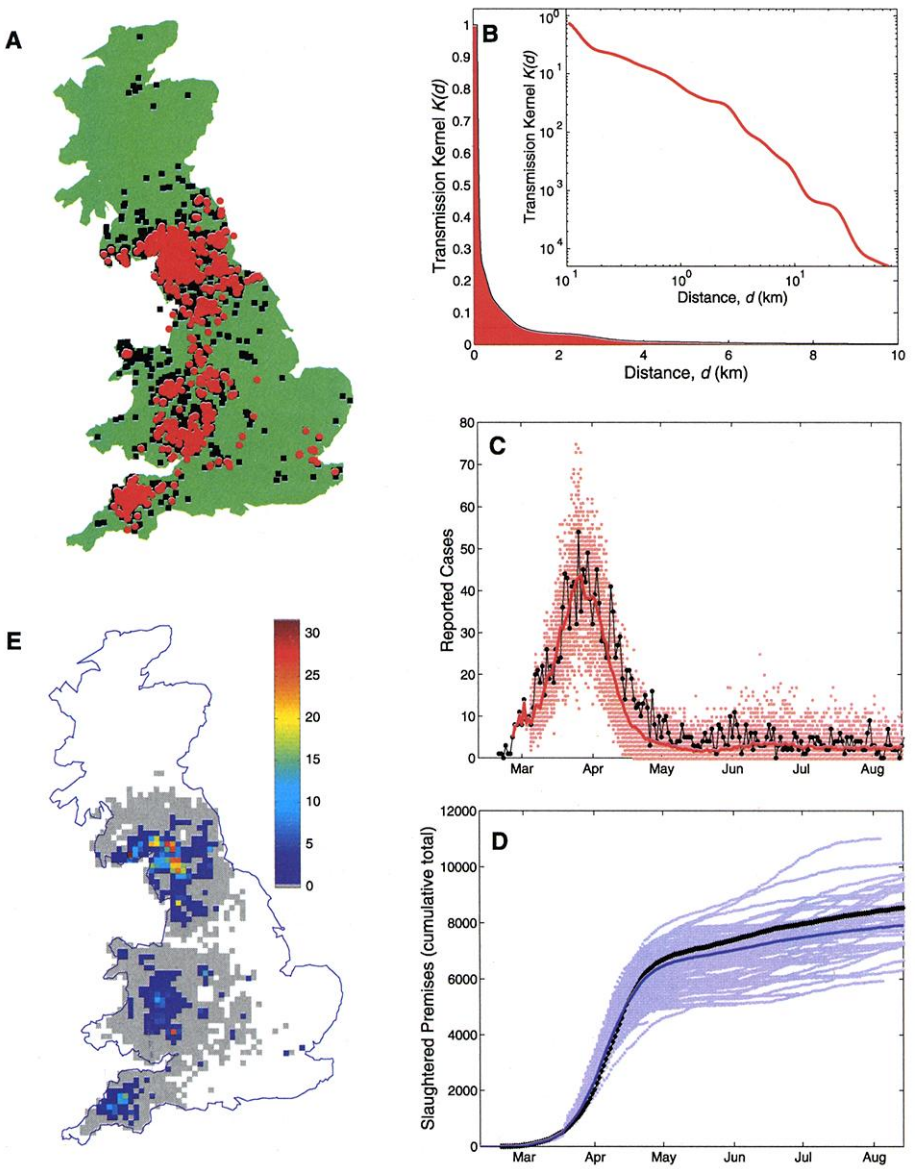

Fig. 1. A comparison between the observed epidemic and 100 replicates of the stochastic model. Simulations start on 23 February 2001 (when movement restrictions were fully in place) and use the reported cases to that date and the position of all susceptible farms as initial conditions. **(A)** The actual spatial distribution of IPs (red) and culled premises (black). **(B)** The transmission kernel K as a function of distance (d), calculated from the distance between sources of infectious and their secondary cases. **(C)** Comparison of the number of infected premises. **(D)** Comparison of the cumulative total of culled or slaughtered premises. Black dots show the actual number, pale dots (red or blue) show the results from simulations, and solid lines (red or blue) show the average of the simulations. **(E)** The average number of simulated cases in 10-km-by-10-km squares. The model results shown are from 100 simulations.

Many other forms of heterogeneity may influence the spatiotemporal dynamics of the disease. Variations in weather, regional geography, farming practices, and farm-level variability in biosecurity could all introduce spatial and temporal heterogeneities into the transmission kernel (*21*). We use the same national average disease dispersal kernel for all farms; we cannot therefore reliably predict the risk for an individual farm, but rather the model identifies areas that are potential "hot spots" for infection. Preliminary analysis of daily meteorological data and case reports has been unable to detect any clear association. This contrasts with conclusions from the 1967–68 foot and mouth epidemic (*22*), but may be due to the more minor role of aerosol transmission and pigs in the current epidemic (*20*).

Control strategies. One of the main roles of modeling this epidemic has been to predict the effects of various control strategies and, hence, inform policy decisions. Analysis of the early epidemic (*23*) demonstrates a change from a net increase in cases (on average, each infected premise causes more than one secondary case, $R > 1$) to a net decrease ($R < 1$) after the introduction of prompt culling and other measures (*24*, *25*). However, R could also be reduced by a local reduction in the number of susceptible farms, because of the progression of the disease, and the observed infection of larger farms early in the epidemic—models are required to explore the link between control measures and epidemic decline (*4*, *13*, *23*).

In qualitative agreement with simpler deterministic models (*4*), our results indicate that—if only infected premises (IPs) were culled—the epidemic would have been much larger, infecting around 20,000 properties (*10*) (Fig. 3A). The remainder of Fig. 3 shows the effects of different culling policies [see supplementary material (*10*)]. The main contrast is between a cull of IPs only and neighborhood culls (*26*), which attempt to remove infections that have spread from the IP before they spread any further. Earlier or more intensive instiga-

tion of the neighborhood control policy (Fig. 3, B and C) would have had a marked effect on the disease, substantially reducing both the number of cases and the total number of culled farms. A more extensive set of culling scenarios is detailed in Table 1. These underline the value of tightly focused neighborhood culling of sheep and cattle, compared with more extensive sheep-only culls actually implemented in some regions.

Vaccination has been proposed as a means of protecting valuable livestock and as a method of controlling the overall epidemic (*27–29*). We focus first on the proposal that vaccination of cattle can be used during an epidemic, along with IP culls, as an alternative to neighborhood culling. Even with optimistic assumptions about vaccine efficacy (*10*) and uptake (90% of farms vaccinated), this results in a much bigger epidemic and total of culled animals than the neighborhood cull (Table 1). Essentially, the delay between the decision to vaccinate and protection from the infection (*30*) (assumed to be 7 days)—together with the delay from infection to reporting—means that it is very difficult to get "in front" of the disease and prevent its spread. This echoes previous conclusions (*4*) against the use of vaccination during an epidemic as an alternative to neighborhood culling.

Next, we consider vaccination as an adjunct to the neighborhood cull. Table 1 shows the effects of superimposing 3-km "ring vaccination" (*10*) on various neighborhood culling policies. Two results emerge. First, vaccination from the start of the epidemic does achieve a reduction in cases and culls. However, an increase in the promptness of culling without vaccination can generate equivalent or even greater improvements. Second, vaccinating from near the start of the epidemic is an optimistic assumption given the logistical constraints inevitable in immunizing millions of livestock, so we also consider vaccination from 1 May superimposed on the neighborhood cull (Table 1). This produces effects almost indistinguishable from neighborhood culling only; local vaccination in the tail of the epidemic has a relatively small effect, probably because of the predominance of stochastic short-duration local epidemics at this point.

The most frequently proposed strategy in the tail of the epidemic is barrier vaccination of cattle, superimposed on the standard neighborhood cull in an effort to protect more distant susceptible areas. Results (Fig. 3D; Table 1) indicate that vaccination barriers would be unlikely to lead to a significant reduction in the spread of the disease in the current very disseminated epidemic. Again, the extra delay associated with vaccination reduces its efficiency as a regional tool during the epidemic (*13*). In practice, the implementation of a vaccination program would be constrained both by logistics and by vaccine performance [of which we have taken a rather optimistic view in this analysis (*31*)]. Ultimately, a decision as to whether or not the epidemiological benefits justify investment in a substantial vaccination program must include a comprehensive economic analysis.

The likely duration of the epidemic is complicated by the highly stochastic nature of the transmission process during the tail of the epidemic and may be dependent on regional het-

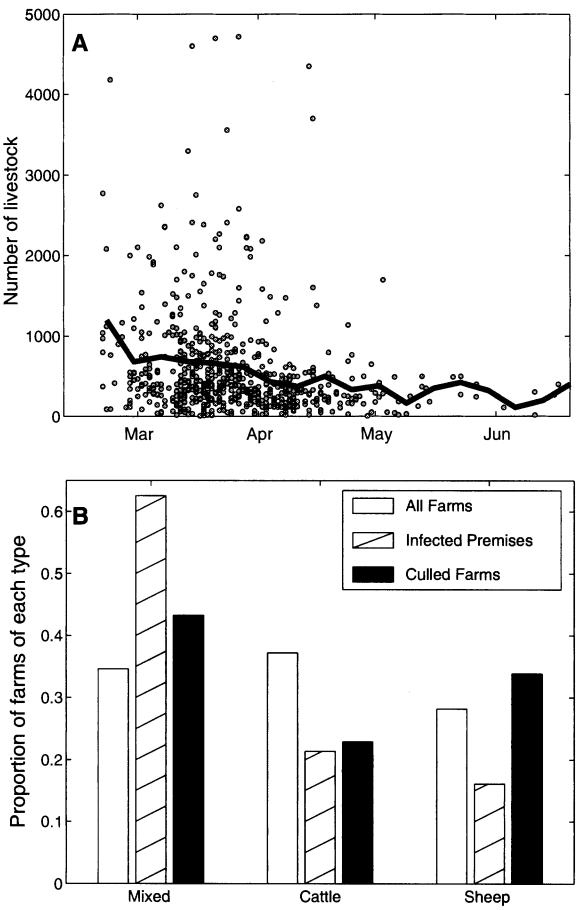

Fig. 2. The effects of heterogeneities in the number and type of livestock on a farm. (**A**) The number of livestock in infected farms in the northern Penrith valley (the solid line is the average farm size during a given week). Similar results are found in all localized outbreaks. Farms can be classified into three main types: (i) those with mixtures of sheep and cattle, (ii) those with cattle only, and (iii) those with sheep only. (**B**) The proportion of farms nationwide in each category; clearly, mixed farms are infected more than their relative abundance would suggest.

Table 1. Results from the stochastic spatial model (*2*, *10*) considering a variety of control options. The total reported cases (on an individual farm basis) for each control policy and the total cull (including IP slaughtering, DC, and CP culls) are given as a percentage of the results from the full model using the observed control policy, including the extended 3-km and welfare culls. The total number of farms vaccinated is given as a percentage of the total cull in the full model. All of the control policies tested below ignore the extended 3-km and welfare culls used in some locations. The standard control policy follows the timing and level of the observed measure. The prompt cull follows the level of the observed measures but achieves a 24/48-hour delay from reporting to slaughter/cull throughout the epidemic. The intensive cull follows the timing of the observed measures but matches the levels achieved in the latter stages of the epidemic. The 3-km ring cull removes infected premises and all other farms within a 3-km radius. The next three measures include vaccination of cattle (at 90% coverage) within a 3-km ring around all infected premises in addition to the slaughter and cull policy. Vaccination of all species gives somewhat better, but qualitatively similar, results. Finally, we consider barrier vaccination (as in Fig. 3D) at 90% coverage. More details about the various control measures are given in the supplementary material (*10*).

Control measure	Total cases	Total cull	Total vaccinated
Standard	105%	84%	0%
IP cull only	927%	342%	0%
Prompt cull (24/48-hour delay throughout)	57%	54%	0%
Intensive cull (high levels throughout)	45%	73%	0%
3-km ring cull only	47%	142%	0%
Standard + 90% vaccination	84%	72%	76%
Standard + vaccination from May	97%	81%	8%
IP only + vaccination	784%	156%	453%
Standard + barrier vaccination	70%	69%	251%

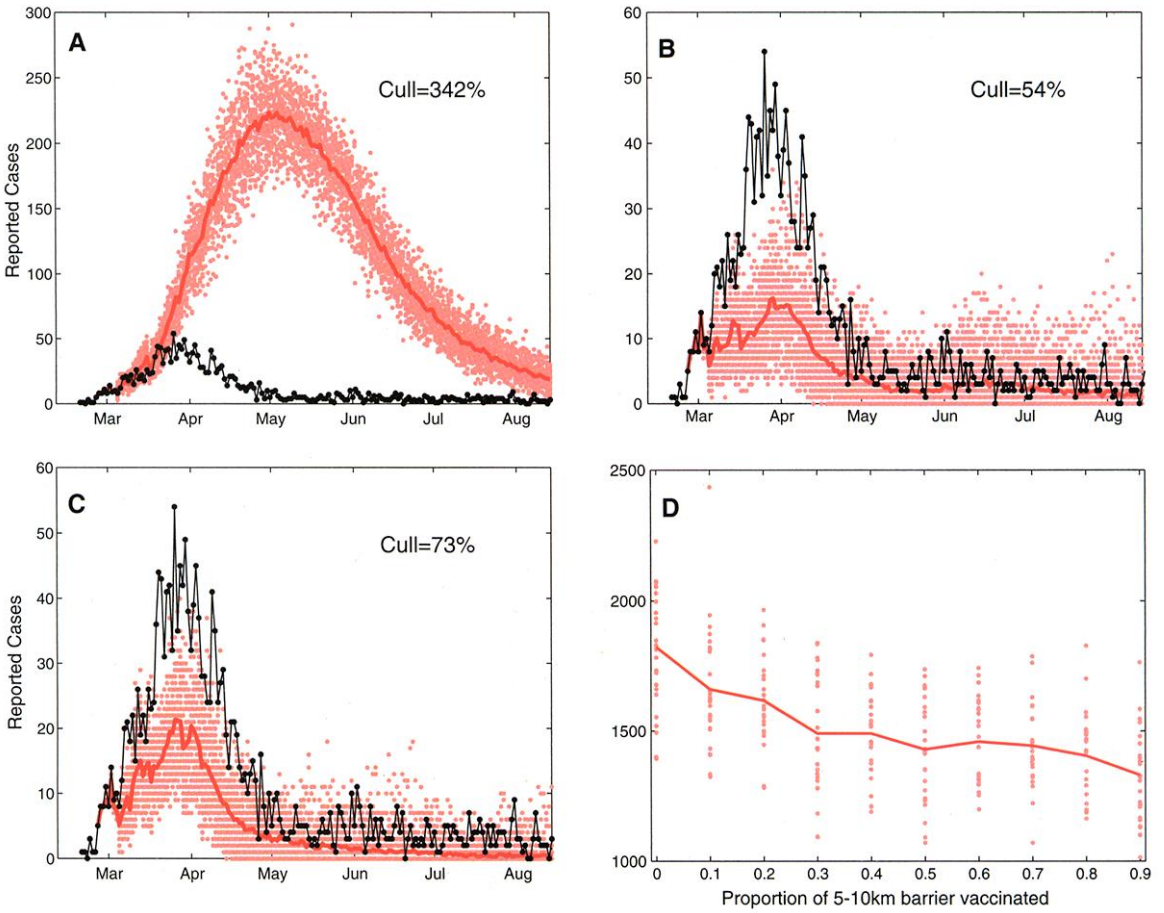

Fig. 3. The effects of varying the culling procedure (black dots denote the actual reported cases, pale red dots are the results of simulations, and the thick red line is the average of those simulations). In (**A**), just the IP cull is performed, so that only infected premises are removed; the delay in that removal follows the observed pattern. The culls in (**B**) follow the observed level (without the extended 3-km culls), but the delays from report to slaughter and report to cull are assumed to be 24 and 48 hours from the beginning of the simulation. In (**C**), the delays follow the observed pattern, but the rates of IP, CP, and DC culls (*2*, *26*) are constant throughout the simulation and mimic the levels achieved in the latter part of the epidemic. (All results are from 50 simulations.) The effects of barrier vaccination are explored in (**D**). A 90° ring, between 5 and 10 km from an IP, is vaccinated with varying degrees of uptake; this barrier is positioned between the disease focus and the largest local density of susceptible farms.

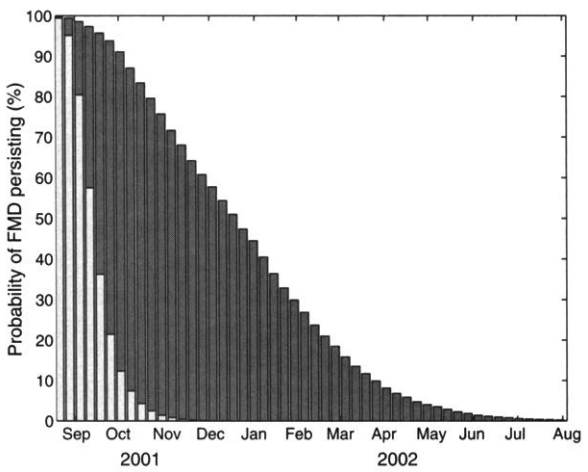

Fig. 4. The duration of the epidemic, aggregated into weekly intervals. Starting with the state of all farms on 20 August 2001 (i.e., all culls and infections to date have been included), the model was iterated forward until the disease died out. Two levels of control are implemented; the dark gray histogram assumes that neighborhood slaughter will continue at the same rate as at the end of July, and the light-gray histogram assumes that slaughter policy will be increased to the previous maximum value that was experienced in mid-April. The duration is highly dependent on the level of control implemented, as well as any future changes in disease transmission.

erogeneities in the transmission kernel. By running the epidemic forward from the latest available data (20 August 2001) and assuming that the transmission kernel and control measure remain constant from now on, we can determine the distribution of end dates (Fig. 4). There is much variability in the epidemic duration according to the intensity of future control measures. Up to 20% of simulations last beyond spring 2002 if control measures are not maintained at a high level, whereas the majority of simulations die out during the autumn if control is pursued vigorously. These predictions are probabilistic and should be interpreted only qualitatively, first because of the simplicity of our model of transmission, and second, because of temporal variation in control effort (*10*) or environmental parameters. Any variation in control measures, movement restrictions, or biosecurity—or changes in the transmissibility of the virus due to climatic or other factors—could significantly alter the situation.

Discussion. Our results illustrate a number of important control issues about the epidemiology and control of FMD, as well as more general implications for spatiotemporal disease dynamics. The main applied result is the importance of rapid implementation of properly focused disease control strategies (*4*, *11*–*13*). If FMD or another highly contagious livestock disease enters a country, then the immediate priority must be to decrease the mixing rate (*25*). This consistently reduces the number of cases, the number of culled animals, and the duration of the epidemic. Prompt instigation of neighborhood culls is also generally a beneficial policy (*4*, *12*, *13*). However, the definition of the appropriate neighborhood will be disease- and situation-dependent. Our study also underlines and characterizes the key interaction of stochastic dispersal, multispecies transmission, and herd size in determining the initial rapid spread and prolonged tail of the epidemic. The herd-

size effect has been noted before (*14*); however, the present work quantifies the key role of cattle and the epidemiological importance of large mixed farms in this epidemic. The coupling of these heterogeneities with a mixture of local and regional disease dispersal explains the observed sequence of epidemiological events. A striking feature of the 2001 epidemic is its much greater spatial extent than the previous major UK FMD epidemic in 1967. The comparative epidemiology of FMD is an important area for future work.

In terms of general lessons for disease dynamics, the following picture of the FMD epidemic emerges. The epidemic began with a massive early dissemination of infection—the size and spatial spread of the subsequent epidemic are very sensitive to the extent of this dispersal (*10*). Movement restrictions then led to a highly skewed spatial infection kernel, which drove both local spread of infection and rarer long-range dispersal. In a more homogeneous landscape, the local spread of a new infection should take a wave-like form (*19*). However, although there is some visual evidence of waves in animations of the observed Cumbria epidemic (*10*), the heterogeneity in farm distribution and structure—as well as tendency for some longer range jumps—generates a relatively complex spatial pattern. We can use the risk factors of farm size and cattle susceptibility to map the risk of infection at different scales. The resulting patterns of average infection potential at the individual farm level (*10*) paint a picture of great heterogeneity in potential spread at a range of spatial scales. Spatially explicit simulations are an essential tool for exploring these patterns, but there is also scope for comparison with a range of more analytical approaches (*3*, *4*, *32*–*34*). The extent and spatial pattern of local epidemics will also be influenced by regional variations in farming practices—for example, the spatial separation (*35*) and dispersion of individual holdings—not explicitly included in our current model.

The balance between spatial distribution, individual heterogeneities, and stochastic transmission is crucial to the choice of control options based on neighborhoods (i.e., ring culling or ring vaccination) or larger populations (i.e., "firebreaks" or mass vaccination) and to what extent these should be targeted at particular farm types. There is well-developed theory for mass and targeted control programs (*1*), but very little for neighborhood control strategies. Another key task for modeling is to explore the role of mass prophylactic vaccination against future FMD epidemics.

Applying the above approach to other disease situations requires, first, good demographic data—including knowledge of seasonal patterns and rates of movement of livestock between regions. The information—as well as data from the current outbreak—should be in the public domain; this would enable the widest range of modeling and statistical techniques and expertise to be applied, aiding the design of optimal strategies against future infections. Second, it is crucial to quantify the spatial infection kernel or, at least, relative contributions of local and nonlocal spread. Third, we need to understand the essential natural history of infection and the strength of individual heterogeneities in transmission—a combination of experimental epidemiology and the assessment of risk factors is the key to this issue. Using this knowledge for disease control requires good disease surveillance, rapid diagnosis—with the associated development of new methods—and quick intervention.

References and Notes

1. R. M. Anderson, R. M. May, *Infectious Diseases of Humans* (Oxford Univ. Press, Oxford, 1991).
2. Transmission between farms is determined by the number and type of livestock and the distance between susceptible and infectious farms. Let $\mathbf{N}_i$ be a vector of the number of animals of each type in farm i, and **S** and **T** be the associated vectors of susceptibility and transmission. The probability that a susceptible farm i becomes infected in a given day

$$\text{Prob} = 1-\exp\left[-\mathbf{S}\cdot\mathbf{N}_i \sum_{j \in \text{infectious}} \mathbf{T}\cdot\mathbf{N}_j K(d_{ij})\right]$$

where K is the infection kernel and d_{ij} is the distance between farms i and j. The kernel, K, is estimated from the tracing of infection (performed by DEFRA) after the movement restrictions were imposed. The kernel determines how the relative risk of infection changes with the distance between a susceptible and infectious farm; it is vital in determining the role of spatial heterogeneity and the rate of spread of the epidemic. Simulations show that the qualitative results presented are not sensitive to the precise shape of the transmission kernel. The number of neighborhood culls (*26*) is related to the number of infected premises, is temporally varying, and follows the observed cull rates. [Greater details of the model are given in the supplementary material (*10*)]. Given the distribution of farms in the UK and the state of those farms on a given day, the state of the epidemic is then updated sequentially. Each day, the probability of infection for every susceptible farm is calculated; a random number generator is then used to decide if the infection event occurs on the basis of this probability. Other operations such as the culling of infected and at-risk farms are also stochastic and carried out daily.
3. M. J. Keeling, *Proc. R. Soc. London B* **266**, 859 (1999).
4. N. Ferguson, C. Donnelly, R. Anderson, *Science* **292**, 1155 (2001).
5. T. W. Bates, M. C. Thurmond, T. E. Carpenter, *Am. J. Vet. Res.* **62** 1121 (2001).
6. R. M. Cannon, M. G. Garner, *Environ. Int.* **25**, 713 (1999).
7. M. Nielen *et al.*, *Prev. Vet. Med.* **28**, 143 (1996).
8. A. M. Hutber, R. P. Kitching, *Trop. Anim. Health Prod.* **32**, 285 (2000).
9. The number and type of livestock in each farm comes from the June 2000 census. Between then and February, when the epidemic began, there was substantial movement of livestock, in particular the movement of sheep out of the upland areas of Cumbria and Wales into lowland regions.
10. Supplementary material is available on *Science Online* at www.sciencemag.org/cgi/content/full/1065973/DC1. This describes in more detail the formulation and parameterization of the model, the various control measures used, and the role of farm and species level heterogeneities. Two animated images illustrate the observed spatiotemporal dynamics at the national and regional scale.
11. D. T. Haydon, M. E. J. Woolhouse, R. P. Kitching, *IMA J. Math. Appl. Med.* **14**, 1 (1997).
12. S. C. Howard, C. A. Donnelly, *Res. Vet. Sci.* **69**, 189 (2000).
13. R. S. Morris, J. W. Wilesmith, M. W. Stern, R. L. Sanson, M. A. Stevenson, *Vet. Rec.* **149**, 137 (2001).
14. M. E. Hugh-Jones, *Res. Vet. Sci.* **13**, 411 (1972).
15. From epidemiological and veterinary records, we have taken the incubation period to be 5 days and the period from infection to reporting to be 9 days. The model fit (*10*) yields the following parameter estimates (all values are per animal, per day): Susceptibility for cattle is 15.2 and for sheep is 1. Transmissibility for cattle is 4.30×10^{-7} and for sheep is 2.67×10^{-7}. We assessed the significance of the heterogeneities that these parameters reveal by fitting a nested series of simpler models omitting the farm-size and species effects (*10*). We use two criteria for significance: the first, the maximized log likelihood of the model, attributes massive significance of the full model compared with the simpler ones ($P < 10^{-100}$). However, the likelihood is too stringent a test for practical purposes because it measures variations in the spatiotemporal pattern of the epidemic at the farm level. We therefore also used a more heuristic measure: the sums of squares of deviations of observed and expected daily cases by county (*10*). Again, this gives a highly significant result for the full model compared with simpler versions with common susceptibility or infectiousness of cattle or sheep ($P < 10^{-10}$).
16. M. S. Bartlett, *Proc. Third Berkley Symp. Math. Stats. Prob.* **4**, 81 (1956).
17. E. Renshaw, *Modelling Biological Populations in Space and Time* (Cambridge Univ. Press, Cambridge, 1991).
18. R. Durrett, S. A. Levin, *Philos. Trans. R. Soc. London Ser. B Biol. Sci.* **343**, 329 (1994).
19. D. Mollison, *J. R. Stat. Soc. B* **39**, 283 (1977).
20. A. I. Donaldson *et al.*, *Vet. Rec.* **148**, 602 (2001).
21. R. P. Kitching, D. K. Mackay, *State Vet. J.* **4**, 7 (1994).
22. M. E. Hugh-Jones, P. B. Wright, *J. Hyg.* **68**, 253 (1970).
23. M. Woolhouse *et al.*, *Nature* **411**, 258 (2001).
24. M. Enserink, *Science* **292**, 410 (2001).
25. J. Giles, *Nature* **410**, 727 (2001).
26. Several types of cull have been defined. The IP cull is the mandatory slaughter of all livestock on an infected premise. Those farms that neighbor an IP are removed by contiguous premise (CP) culls, whereas farms where animal, human, or vehicle movement, or airborne transmission, may have transferred the infection are removed by the dangerous contact (DC) cull. Both of these forms of culls are generally in the neighborhood of the infected farm. In some regions, such as Cumbria, Dumfries, and Galloway, other more extensive slaughter policies have been applied.
27. J. Brownlie, *Vet. Rec.* **148**, 358 (2001).
28. P. W. Mason, M. J. Grubman, *Aust. Vet. J.* **79**, 342 (2001).
29. J. S. Salt, in *Veterinary Vaccinology*, P. P. Pastoret *et al.*, Eds. (Elsevier, Amsterdam, 1997), pp. 641–652.
30. S. J. Cox, P. V. Barnett, P. Dani, J. S. Salt, *Vaccine* **17**, 1858 (1999).
31. M. E. J. Woolhouse, D. T. Haydon, A. Pearson, R. P. Kitching, *Epidemiol. Infect.* **116**, 363 (1996).
32. R. M. May, R. M. Anderson, *Philos. Trans. R. Soc. London Ser. B Biol. Sci.* **321**, 565 (1988).
33. B. M. Bolker, S. W. Pacala, *Am. Nat.* **153**, 575 (1999).
34. B. F. Finkenstädt, B. T. Grenfell, *J. R. Stat. Soc. C* **49**, 187 (2000).
35. R. R. Kao, *Vet. Rec.* **148**, 746 (2001).

36. Supported by the Royal Society (M.J.K.), the Wellcome Trust (M.E.J.W., B.T.G., D.J.S., L.M., S.J.C., and J.K.), and Biotechnology and Biological Sciences Research Council (M.E.J.W. and M.C.T.). We thank the members of the UK Chief Scientific Advisor's Science Group on Foot and Mouth disease; O. N. Bjørnstad, C. A. Gilligan, K. Glass, C. Green, and B. Leung for useful discussions; and the British Atmospheric Data Centre for providing detailed meteorological information. Detailed farm-level data were provided by the Department of the Environment, Food and Rural Affairs, the Scottish Executive Rural Affairs Department, and the National Assembly for Wales Statistical Directorate.

4 September 2001; accepted 25 September 2001
Published online 3 October 2001;
10.1126/science.1065973
Include this information when citing this paper.