
Where diseases and networks collide: lessons to be learnt from a study of the 2001 foot-and-mouth disease epidemic

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SUMMARY

This paper uses a graph-theoretical approach to investigate the properties of the observed network of disease transmission in the 2001 foot-and-mouth epidemic in the United Kingdom. This analysis revealed both global and local heterogeneity in the contact pattern between the infected premises in the first 3 weeks of the disease. In particular, the global heterogeneity contributed to the failure of the culling strategy imposed by the UK government. However, a more effective strategy targeting selective deletion of key premises in the network was not available once the epidemic had begun. We recommend that *post-hoc* analyses of this sort should become part of preventative and proactive policy rather than part of a reaction to an ongoing crisis.

INTRODUCTION

One of the biggest challenges in epidemiology is understanding how transmission of disease varies with time and space [1, 2]. Early attempts at modelling disease spread in populations were based on mean-field approaches. In these models the population of susceptible individuals is assumed to be completely mixed, with all individuals equally likely to be infected by contact with an infected individual [3–5]. Where disease transmission is dependent on patterns of behaviour which are themselves heterogeneous, this assumption of mixing is rarely true [6–8]. Under these circumstances predicting disease spread is not straightforward. The importance of contact patterns in tracing the pattern of spread and in applying control measures has been extensively studied for sexually transmitted diseases, where there may be many

different types of contact ‘process’ and both spatial and temporal heterogeneity in contact pattern [9–11]. Whilst there has been considerable research to incorporate such heterogeneity in mean-field modelling approaches [12, 13], these approaches remain focused at the population level.

Individual-based approaches focus on the individuals in the population and their linkages to others with or without the disease. As such, individuals can be considered as vertices in a network, connected to each other through links called edges. An edge represents the relationship between two vertices, whether this relationship is a direct connection of a cable in a power grid, a neuron in a nervous system, a hyperlink of the worldwide web, or a joint authorship paper in a collaboration network [14]. In a disease context, an edge represents a contact between individuals in the susceptible population, and diseases spread through the whole population by means of these edges. The frequency and distribution patterns of connections amongst vertices are used to classify networks and there has been considerable interest in investigating how disease spread is determined by network

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characteristics. There are three basic patterns of connections between vertices in non-structured networks: random graphs, small-world networks and scale-free networks. Random networks consist of N vertices connected by M edges which are randomly chosen from the $N(N - 1)/2$ possible edges with probability P [15]. Small-world networks [16] are a class of random graphs which have a greater degree of local structure than one would expect from a random graph of the same size [17], whilst scale-free networks are a class of random graph where the frequency of the number of neighbours of the vertices is scale-invariant [18]. Scale-free networks have a few vertices with a very large number of neighbours, with the majority of vertices having few connections. It should be noted that these categories are not mutually exclusive; a network can have both scale-free and small-world properties. Lattice networks (e.g. [19]) have also been widely used to model spatial diffusion of a disease in a non-randomly mixing (structured) population.

It has been shown that in random graphs the rate of spread of disease is dependent on the distribution of contacts between the vertices [20], which approximates the results of epidemiological models based on assumptions of random mixing. Scale-free networks have no epidemic threshold for diseases [21, 22]. Diseases can spread in such networks even when the infection probability is exceedingly small. This contrasts strongly with the predictions of mean-field models where there is a precise, mathematically defined threshold of infected individuals [22], below which epidemics will not occur. Small-world networks also have a nonlinear distribution in the size of the neighbourhood of their vertices, but rather than a power relationship, the distribution is a delta function centred on the average number of neighbours [14]. In this type of network, the probability of an epidemic occurring coincides with the predictions of a random graph [23], but the velocity of disease spread is very different as a result of the local structure of interactions which are not found in random graphs [24].

Given that the dynamics of diseases in a network will depend upon the frequency of connections between individuals, processes which lead to the severing of these connections will, therefore, restrict or even halt the progress of the disease. The evaluation of the impacts of fragmentation of a network therefore has relevance in the field of practical disease control.

The aim of this paper is to investigate the dynamics of a real disease network. We focus on the 2001

outbreak of foot-and-mouth disease (FMD) in the United Kingdom as an example of a disease network characterized by heterogeneity both in space and in contact behaviour; and one in which it proved difficult to halt the progress of the epidemic, eventually resulting in the destruction of livestock on over 10 000 premises. We characterize the FMD network and compare it to theoretical networks used previously to investigate disease dynamics. We ask whether the nature of FMD, combined with the social structure of the UK farming community, resulted in a network that was resistant to the control measures imposed by the UK's Ministry of Agriculture by virtue of its topology. In addition, could an understanding of the likely topology of such diseases be of assistance in formulating plans for disease control in the future?

METHODS

Physical description of the FMD network

FMD is a virulent viral disease of domestic ungulates, and poses a serious threat to the livelihoods of livestock farmers due to its long incubation period and mild prodromal symptoms, which allows infected and infectious animals to remain undetected for up to 10 days post-infection [25].

During the 2001 outbreak of FMD in the United Kingdom, The Ministry of Agriculture, Fisheries and Food [MAFF, now the Department of Environment, Food and Rural Affairs (Defra)] and the Veterinary Laboratory Agency (VLA) traced the origin of all confirmed outbreaks of the disease, and these data were published on MAFF's website. As they became available, the first 239 infected premises (20 February–15 March) were resolved into a network (henceforth called the FMD network) consisting of $n = 248$ vertices (the 239 infected premises plus nine livestock markets). Edges between vertices were defined as being between an infected vertex and its traced source; thus, information about the flow of disease through the network was preserved. The size of the network (that is, the number of edges) was $M = 295$. Subsequent to the epidemic, data from all 2030 premises eventually infected became available to academic researchers; however, the source of infection was not positively identified for many of these data, and the resolution of a larger fully connected network has its own problems [26]. We have, therefore, concentrated on analysing the fully connected fragment of the first 248 cases, for which the data on contacts

were publicly available. This spans the period of the initial outbreak, the Ministry's immediate response to control the epidemic, and subsequent attempts to limit its spread.

Characteristics of the FMD network

Networks are characterized by the pattern of connections (*edges*) between individuals (*vertices*). This pattern describes the relationships between members of the network, and has important consequences on how a disease spreads through the network.

The frequency distribution of the number of connections to a vertex (called the *degree distribution*) is important in determining the infective potential of individuals because it quantifies the number of vertices with a given number of neighbours. However, the degree distribution gives no measure of the level of inter-connectedness of a vertex at the local scale. The *clustering* of a network (the proportion of the neighbours of the same vertex that are also adjacent to each other [17]) measures the level of local connectivity in a vertex's neighbourhood, which represents the tendency for individuals to form social or spatial groups which are more tightly connected with each other than with the rest of the network. The importance of a vertex in maintaining the connectivity of the whole network (rather than the connectivity of the local cluster) is its *significance* (the expected number of edges by which a local neighbourhood is separated in the absence of its central vertex [17]).

The level of connectivity in a network – as described by the degree, clustering and significance of its vertices – will affect the speed of the disease through the network. The disease velocity may be measured by the average shortest number of edges needed to be traversed to reach every other vertex in the network (this is called the *path length* of a vertex). Short path lengths indicate that an infected vertex is well-connected, and can infect the rest of the network in comparatively few time-steps.

Values for path length, clustering and significance were measured for each vertex in the FMD network. These three characteristics, along with the degree distribution, were compared with those expected from random, scale-free and small-world networks on theoretical grounds.

Fragmentation of the FMD network

The fragility of a network is the response observed when vertices are removed [27]. If the pattern of

connectivity in a network does not show substantial changes when vertices are randomly deleted, the network can be said to be 'error tolerant'. However, high error tolerance is usually coupled with a social or spatial structure in which a few vertices play a vital role in maintaining the network's connectivity. The existence of these key vertices means that the network's structure is vulnerable to targeted attacks [28], which can cause major changes to the coherence of the network and thereby impact on the speed and pattern of disease spread.

Fragmentation of the FMD network is the equivalent of removing individual markets (through a movement ban) or farms (through culling) from the disease network, either through total quarantine, vaccination, or culling of all susceptible livestock. The removal of a vertex from the network interrupts the spread of the disease, and may break the network up into one or more fragments. The size of the largest fragment following removal of a vertex therefore represents the largest possible size of the epidemic following this removal. The number of fragments resulting from the removal of each vertex is a measure of the relative importance of the vertex that was removed – premises which are highly connected are likely to leave behind multiple fragments of a network that are no longer capable of contracting the disease because they are no longer connected to the largest fragment.

The fragility of the FMD network was, therefore, analysed by deleting vertices and then recording the subsequent characteristics of the network: the size of the largest fragment; and the number of fragments. The FMD network was first converted into a directed network before this analysis was performed. In the directed FMD network, edges operated only in one direction, connecting a recipient of the disease to its source. This conversion did not affect the overall connectivity of the network, but did alter derived characteristics such as path length. The reason for this conversion was that we were only interested in the relationship between infected vertices and their subsequent infections, rather than considering the edge to be a two-way connection (which is unrealistic).

Vertex deletion was performed twice, using the methodology of Solé & Montoya [28]: vertices were removed either in a randomly chosen order (to test for error tolerance); or in order of their degree, beginning with the vertex with the highest number of neighbours (to test for attack vulnerability). Thus, deletion began with the vertex that had the most outward links

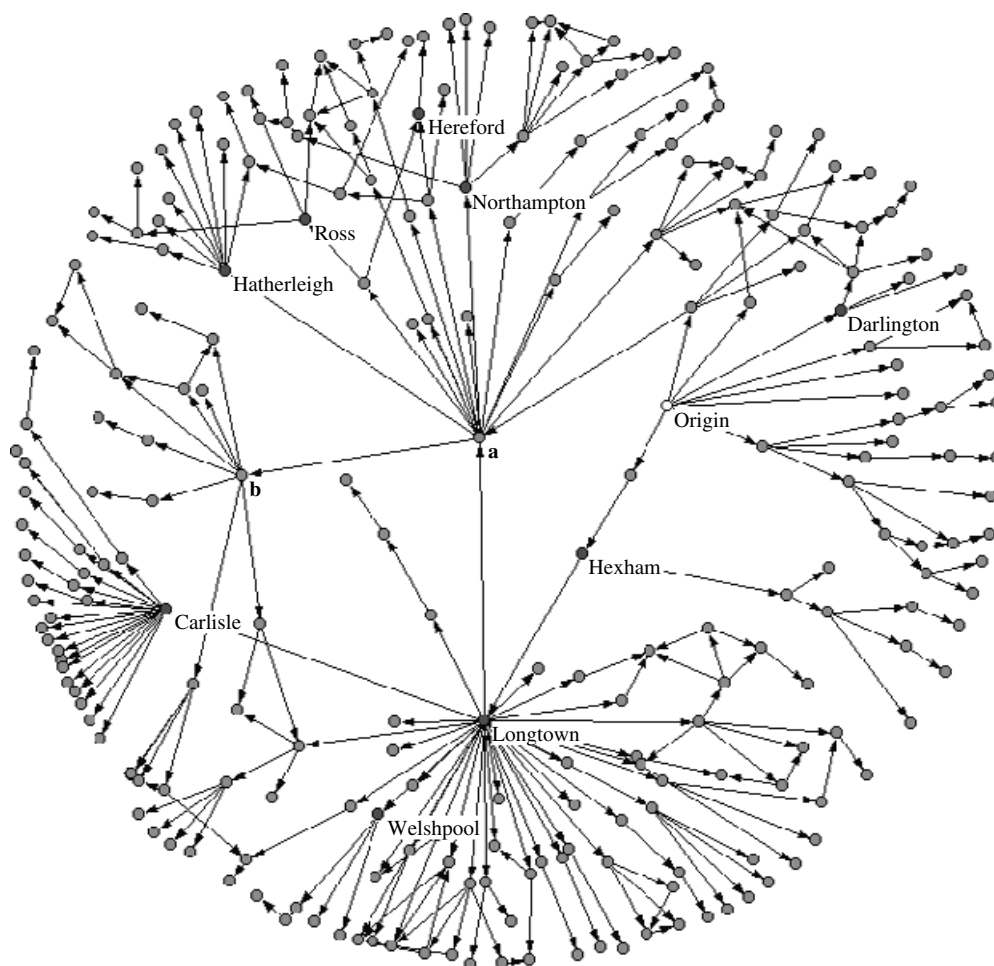


Fig. 1. The network of the 2001 foot-and-mouth disease epidemic (20 February–15 March). Vertices marked with a label are livestock markets, unmarked vertices are farms. Only confirmed infected premises are included. The origin of the epidemic in Northumberland is indicated by an open vertex. The vertices marked **a** and **b** are farms in Devon and Hereford respectively. Arrows indicate route of infection. (Image created in the Pajek program, <http://vlado.fmf.uni-lj.si/pub/networks/pajek/pajekman.htm>).

(i.e. the vertex which passed the disease on to the greatest number of other vertices).

Effect of MAFF control policies on network structure

The National Movement Ban (NMB) came into force at 17:00 hours on 22 February 2001, preventing the movement of livestock between premises in an attempt to limit spread of disease. The livestock markets were closed as a result of this ban.

To examine the effects of the NMB on the topology of the FMD network, each vertex was categorized as to whether it was infected before or subsequent to the NMB. Infection date was estimated by the VLA to be either 5 days prior to the earliest appearance of lesions, or the date of known contact with the infection. The period of infection (from estimated infection

date to slaughter) was calculated for each vertex, along with the number of subsequent premises infected (i.e. the 'out degree' of the vertex).

RESULTS

Physical description of the FMD network

A visualization of the FMD network is shown in Figure 1. The origin of the outbreak in Northumberland was identified on 22 February 2001 after tracing back the first case from an abattoir in Essex. By the time precautions to prevent further spread were in place, infected animals from the origin had already made their way to Hexham (10 February), Darlington (12 February), Longtown (14–24 February) and Carlisle (16 February) livestock

Table 1. A comparison of network metrics calculated for the FMD network with the theoretical expectations of these metrics for random graphs, small-world networks and scale-free networks

	FMD network	Random graphs	Small-world networks	Scale-free networks
Degree distribution	Approximates a power distribution	Poisson distribution	Delta distribution	Power distribution
Average path length	$D=5.18$	$D=6.39$	$D=6.39$	$D=3.23$
Average clustering	$C=0.19$	$C=0.01$	$0.60 \leq C \leq 0.67$	$C=0.04$
Average significance	$S=70.87$	$S \approx D$	Varies, but tends to be low	Can be very high

Unless otherwise stated, it is assumed that these networks have the same basic parameters of the FMD network, namely $N=248$ vertices, and average degree $K=2.37$. The theoretical values for the three networks were derived from formulae given in Albert & Barabási [14] and Watts [17].

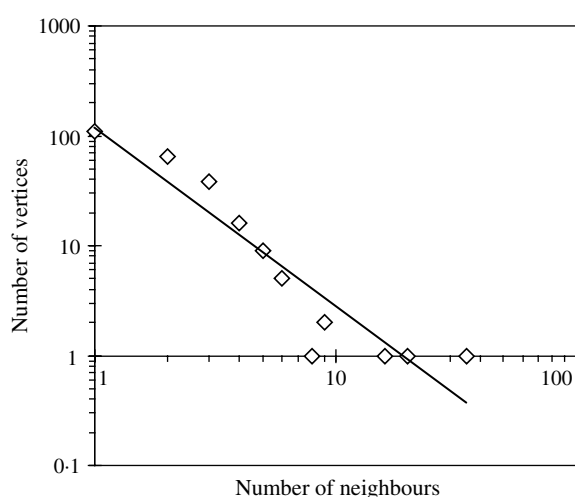


Fig. 2. The degree distribution of the FMD network. The fitted line has the following equation: $y=118.5x^{-1.6}$, $R^2=0.87$.

markets. By 20 February, cattle bought at Longtown market had been transported to Devon, and from there to Hereford, Northampton and Ross.

Subsequent to the 239 cases (plus nine markets) included in this network, the disease spread to 2030 premises in the United Kingdom; and a total of 10 157 farms, markets and abattoirs were culled due to being contiguous to known infected premises or having known contact with one. An estimated 6 456 000 sheep, cattle and pigs were slaughtered before the epidemic had run its course [29].

Topological description of the FMD network

The degree distribution of the FMD network can be seen in Figure 2. It can be seen that the distribution has a clear power-law relationship between the

number of nodes with a given neighbourhood size. A total of 70% of the vertices in the network have only 40% of the overall number of edges; whereas the most highly connected 2% of the farms collectively have 15% of the connections.

The average path length, clustering coefficient, and average significance of the FMD network are shown in Table 1 along with expected ranges of these parameters for the random, scale-free and small-world networks.

The FMD network was most topologically similar to the scale-free network of the same order in terms of degree distribution and significance. The only distinguishing feature was the lack of clustering in the scale-free network that was quite prominent in the FMD network. On average, 19% of a vertex's neighbours in this network were also neighbours. This clustering is a result of more than one farm in a neighbourhood being a possible source of the disease of an infected farm; which is a consequence of the densely packed UK farming landscape.

The degree distribution of the FMD network (Fig. 2) most closely follows a power law distribution in that the majority of premises have a single neighbour responsible for passing on the disease; with a few premises acting as 'hubs' and having a large number of neighbours to whom the disease was transmitted. The hubs in the FMD network can be clearly identified in Figure 1 as the livestock markets at Longtown, Carlisle and Hatherleigh, and the two farms marked **a** and **b**.

Fragmentation of the FMD network

The effects of random and selective fragmentation of the FMD network on the size of the largest fragment

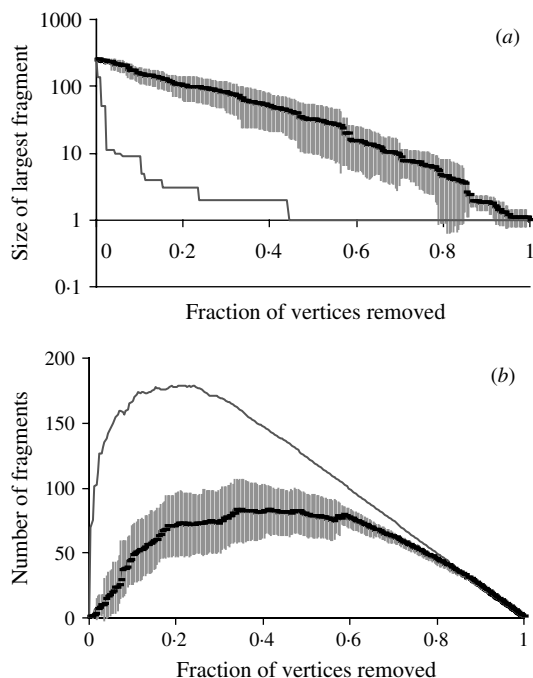


Fig. 3. The effect of fragmentation on FMD network. (a) Size of the largest fragment. (b) Total number of fragments. Standard deviations are shown as grey bars for random deletion ($n = 10$). —, Random; - - -, selective.

and the average size of the other fragments can be seen in Figure 3. When vertices were selectively removed (Fig. 3a), there was more than a tenfold drop in the size of the largest network fragment following the removal of only 2% of the vertices. This level of fragmentation left a total of 112 network fragments (Fig. 3b). For random deletion of vertices there was no such threshold at which the size of the largest remaining fragment dramatically declined (Fig. 3a); instead there was a steady decline in the total epidemic size. At the same time, the number of fragments did not vary greatly (Fig. 3b), implying that the random removal of vertices did not further fragment the network to any great extent. Each farm randomly removed from the network, on average, prevents a very few farms from contracting the disease.

Figure 4 shows the effect on disease spread resulting from fragmentation. There were only eight vertices (3.2%) in the FMD network (other than the origin of infection) that, if they had been removed, would have prevented more than 10% of subsequent infections each. These eight vertices had extremely high individual significance, which was responsible for a high average significance for the network as a whole (Table 1). The removal of any one of only three of

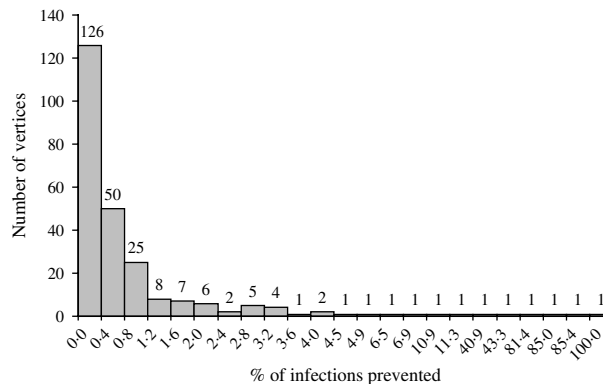


Fig. 4. The effect of vertex removal from the directed FMD network on preventing subsequent infections. There is one node (the origin) whose removal would prevent 100% of the 247 other infections. There are 126 vertices whose removal would have no effect, as they do not pass the disease on to other vertices in the first 24 days of the epidemic.

these vertices would have stopped over 80% of the infected premises, and halted the epidemic in its tracks. These three vital vertices were: Hexham market, Longtown market, and the farm in Devon which is marked **a** in Figure 1.

Effect of MAFF control policies on network structure

The effects of the NMB on the network structure are shown in Table 2. Approximately half of the FMD network was infected by the time that the NMB came into force, although there were only 15 cases showing signs of the disease. The NMB reduced the average period of infection by ~5 days; and the proportion of infected premises (IPs) which caused more than one other premise to become infected dropped from 69 to 39% following the ban.

However, two of the three largest hubs in the FMD network were reported as IPs after the date of the ban, having been already infected by moving livestock. This suggests that the effects of the NMB would only be seen after all IPs infected before the ban had been reported.

DISCUSSION

What has a *post-hoc* analysis of the contact pattern told us about the 2001 FMD epidemic? The topological analysis of the FMD network reveals a heterogeneous pattern of contacts, which relates to different stages of the epidemic. First, the process of long distance movement of sheep by livestock dealers

Table 2. *The effects of the national movement ban (NMB) on the FMD network*

	Before NMB	After NMB
Total number of cases reported	15	233
Total number of cases infected	119	129
Average number of days between infection to slaughter (standard deviation)	15.8 ± 5.7	10.6 ± 2.4
Largest number of infections per source	19, 8, 5	35, 14, 8
Proportion of reported sources responsible for > 1 subsequent infection	0.69	0.39

These data apply only to the 248 vertices of the FMD network. Infection and slaughter dates were provided from the FMD Data Archive (<http://defra.gov.uk>), and collected by Defra and the VLA.

[30] meant that a few vertices (i.e. the markets) monopolized a large proportion of the total edges in the network. This global heterogeneity can be seen in the FMD network, characterized by an approximation of a scale-free degree distribution. This meant that these markets dominated the network and were major features determining disease spread. By the time the presence of FMD was disclosed on 20 February (based on only two cases), it has been estimated that 57 premises in 16 counties had already been infected [31], including the six hubs identified in the FMD network. Another 62 cases are estimated to have been infected before movement restrictions on livestock came into force on 23 February [31]. Subsequent to the movement restrictions there were no livestock markets and animal transport was prohibited; and contagious diffusion of the disease to nearby farms lead to the development of low-degree vertices in the network.

Secondly, whilst our network consisted of only the first 3 weeks of a 7-month epidemic, more than 80% of the total infectious events occurring subsequent to the livestock movement restrictions arose within 3 km of their source [30], suggesting that the further progression of the epidemic beyond the first 239 cases was through contagious spread between farms, either short-range wind-borne spread of the virus across field boundaries between herds of livestock, or through physical contact. The number of cases arising from each infectious premises in the later stage of the disease was low relative to earlier phases in the epidemic. This pattern of contagious spread was represented in the network as clustering, and the high level of clustering observed in the FMD network is a feature not seen in randomly mixing populations which have a homogeneous pattern of connectance.

Finally, the clustering was not uniformly spread through the FMD network, as indicated by the high

significance, with some premises having very few connections and others very many. The clusters of contiguous farms in the UK farming landscape resulted in many infected premises having more than one possible source of infection, as suspected sources of infection could not be distinguished from actual sources of infection in the contact data. This suggests that the velocity of the disease would not be greatly affected by control measures which restricted the means of transmission on a local level, unless these measures were swift and total, simultaneously removing a source of infection and hence all of its possible future contacts.

The relevance of this analysis for disease control is constrained by the fact that the FMD network consists of infected premises only; that is, the network does not exist until the epidemic has occurred. The farming network – that is, the network of all premises, is likely to consist of a structured network of clustered communities, joined by the occasional long-distance link to livestock markets. However, when planning control strategies, the knowledge that the epidemic network could take the form of a locally clustered, globally heterogeneous network is important, as this structure will affect the efficacy of strategies to limit the spread of the disease and the extent of subsequent epidemics.

The strategy of vertex removal taken by MAFF during the 2001 epidemic was to implement the 24/48 policy on 29 March, aimed at preventing further transmission from infected premises. Two characteristics of the FMD network – the high significance and the power-law degree distribution – indicated that the network also displayed a pattern of global heterogeneity, in that some regions of the network had much larger neighbourhoods and consequentially were of much higher significance, than other regions of the network. This global heterogeneity resulted in

tolerance to random deletion of vertices, but high susceptibility to selective deletion – ‘error-tolerant’ and ‘attack vulnerable’ respectively [32], and should have responded well to a strategy which removed infected premises swiftly from the network. However, operational difficulties in conducting the livestock cull meant that 59% of infected premises took more than 24 h to cull, and 68% of contiguous premises and dangerous contacts took more than 48 h for complete slaughter; and the removal and disposal of the corpses took even longer [29]. Additionally, livestock can excrete virus particles for up to 5 days (in cattle) and 10 days (in pigs) before becoming symptomatic [33]; and the removal of a vertex in a network *after* it has transmitted the disease is irrelevant to the epidemic network, as the edge has already been formed. A removed farm vertex has been culled, and a removed market vertex has been closed; in either case, no animals are present to excrete virus particles. These factors combine to make the MAFF culling policy analogous to a random deletion strategy, which may eventually result in disease eradication, but is far less effective than a strategy based on selective vertex removal. While it is clear that the 24/48 policy had a major impact on the decline of the epidemic [34], the problems in its execution resulted in a larger epidemic than that predicted if the policy had been easier to implement. The MAFF NMB – closing livestock markets and stopping all movement of livestock – was analogous to a selective removal strategy; except that it is clear with hindsight that not all hubs in the network are markets. Figure 1 shows that two farms in particular, one in Devon and one in Hereford, were important hubs. Prioritization in culling vertices which have the potential to cause a high number of subsequent infections would have continued this concentration on selective removal; however, such a strategy is only possible with knowledge of the network structure of the farming landscape.

In scale-free networks, the epidemic threshold for Susceptible–Infected–Susceptible epidemiological spread is zero [22], i.e. a single infected individual will always produce an epidemic, even if the rate of spread of the disease is very small. This result has been confirmed for Susceptible–Infected–Removed epidemics [35], although these authors also indicate that this result is due to the infinite variance in the vertex-connectivity distribution of these infinite-sized scale-free networks. They suggest that, as heterogeneity is usually low in networks describing disease

transmission, these results may be less appropriate for diseases spread by social contact. However, in the case of the FMD network, it is clear that the global heterogeneity (variance) is very high, much like a scale-free network. Consequently, while the epidemic threshold will not be zero (because the FMD network is finite in size), it is expected to be considerably lower than a randomly mixing network. Rapid spread of the disease is, therefore, an inevitable consequence of an outbreak of FMD in the current UK farming system unless all infected vertices can be simultaneously cured, which is practically impossible without a total vaccination policy. However, the epidemic threshold can be restored if sufficient highly connected vertices over a certain size are cured [36]. Under this strategy the rate of disease spread will not exceed the threshold, and an epidemic will not occur. Additionally, the more successful that the policy is in curing significant vertices, the fewer cures are needed [36].

Mathematical models were a prominent part of the Government’s strategy for fighting the FMD epidemic of 2001. There have been previous studies which modelled the spread of this disease [25, 37, 38]. However, since clinical studies conclusively showed that the virus can be transported by air currents as well as direct contact between individuals [39, 40] the assumption of wind-borne transmission dominated the design and operation of monitoring and control programmes for the disease in the United Kingdom [41–43]. The models of Ferguson et al. [44, 45], Keeling et al. [46] and Morris et al. [47] were used to inform MAFF policy while the 2001 outbreak was ongoing. The ‘Ferguson model’ [44, 45] was a mass-action model that assumed homogeneous mixing, but used moment closure to approximate neighbourhood effects. Both the ‘Keeling model’ [46] and the ‘Morris model’ [47] included spatial information about the location of all British farms. A further model [48] ignored long-distance movements and concentrated on contagious spread between neighbours. All three models used to advise the UK government came to the same three conclusions [49]: (i) that the disease had exceeded the epidemic threshold number of cases; (ii) that stamping out infected premises and dangerous contacts was unlikely to reduce the basic reproductive rate of the disease quickly; (iii) that the preventative slaughter of contiguous premises would bring the reproductive rate of the disease below the epidemic threshold. MAFF introduced a ‘24/48’ policy on 29 March [34], where culling of animals on

infected premises and dangerous contacts would take place within 24 h, and on contiguous premises and known contacts within 48 h.

From our analysis of the FMD network, and based on the assumption that contagious spread was the route of transmission following the first 3 weeks of the disease, it is apparent that the strategy of reactive culling of diseased animals at infected premises (and their dangerous contacts) was not the most efficient strategy for controlling the outbreak. At least in the early weeks of the epidemic, covered by these analyses, 39% of premises gave rise to two or more subsequent infections, indicating that the disease continued to spread. The FMD network, by virtue of its pattern of contacts, was vulnerable to management policy directed at livestock markets acting as hubs (such as a movement ban); however, by the time the disease had become apparent, these hubs were infected and had already infected other premises. The construction of epidemic trees has revealed that if the NMB had come into force just 2 days earlier, the epidemic would have been half its eventual size [26]. The NMB prevented the formation of yet more hubs, but the ones which existed continued to spread the disease locally, as evidenced by the appearance of large hubs subsequent to the NMB (Table 2). Swift treatment of these premises would have stopped the epidemic from forming, but livestock with FMD excrete infectious virus particles before displaying clinical signs of the disease, so the identification of such premises proved impossible; and the epidemic of 2030 cases was the result.

Mathematical models have a useful role to play in the control of an epidemic while it occurs. In the case of the 2001 FMD epidemic, models were updated as the disease progressed, and highlighted the need to act quickly and devote more resources to epidemiologists from central government. They proved a useful tool for policy makers in the midst of the epidemic in predicting likely future disease dynamic and the economic impact of proposed actions. However, Taylor [50] recommends that mathematical models of epidemiology are most appropriately used in 'peacetime'; in retrospective analyses to inform model development, contingency planning for future epidemics, resource planning and the targeting of surveillance in priority areas. The results of this paper support these recommendations, and suggest that the most effective strategies would revolve around the neutralization of those premises such as livestock markets and dealers who act like hubs. On a more

general note, this study emphasizes the need to understand the contact pattern of susceptible populations before embarking on any strategy for disease control, which means that populations at risk from disease need to be characterized topologically before an outbreak occurs.

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