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Use and abuse of mathematical models: an illustration from the 2001 foot and mouth disease epidemic in the United Kingdom

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Summary

Foot and mouth disease (FMD) is a major threat, not only to countries whose economies rely on agricultural exports, but also to industrialised countries that maintain a healthy domestic livestock industry by eliminating major infectious diseases from their livestock populations. Traditional methods of controlling diseases such as FMD require the rapid detection and slaughter of infected animals, and any susceptible animals with which they may have been in contact, either directly or indirectly. During the 2001 epidemic of FMD in the United Kingdom (UK), this approach was supplemented by a culling policy driven by unvalidated predictive models. The epidemic and its control resulted in the death of approximately ten million animals, public disgust with the magnitude of the slaughter, and political resolve to adopt alternative options, notably including vaccination, to control any future epidemics. The UK experience provides a salutary warning of how models can be abused in the interests of scientific opportunism.

Keywords

Culling – Epidemiology – Foot and mouth disease – Infectivity – Mathematical model – Modelling – Slaughter – Stamping out – Transmission – United Kingdom – Virus spread.

Introduction

The epidemic of foot and mouth disease (FMD) in Europe in 2001 had a profound effect on the public perception of the consequences of a highly infectious disease spreading within an intensive livestock industry. Although there had been epidemics of classical swine fever in the Netherlands in 1997 (in which over ten million pigs were slaughtered) and in England in 2000 (in which 80,000 pigs were slaughtered), the nightly appearance on television of apparently healthy cattle and sheep being sacrificed to bring FMD under control horrified both rural and urban communities, and involved government at the highest level. In Britain, four million FMD-susceptible animals on 10,157 premises were slaughtered: 2,026 premises were declared infected, 4,762 premises were considered dangerous contacts of infected premises and 3,369 premises were located near to infected premises (2). A further 2.5 million animals were slaughtered for reasons of welfare, such as overcrowding and compromised nutrition. The official figure for the number of animals slaughtered was approximately 6.5 million, but when the total number of still-suckling lambs, calves and pigs that were slaughtered is included, the total could be as high as ten million (33). The financial cost of the FMD epidemic in

the United Kingdom (UK), that is, including Northern Ireland where four premises were infected, was over US$ 12 billion, including US$ 4.5 billion in losses sustained by the leisure and tourist industry (95). However, the social cost – evidenced by the many newspaper reports and submissions to the various inquiries conducted after the epidemic – could not be quantified. Significantly, the excessive slaughter ignored Coase's principle of welfare economics (108), which states that the actual costs of state intervention must be considered, rather than costs deriving from a theoretically coherent policy.

These costs, both financial and social, initiated political pressure to change national, European Union (EU) and international legislation and guidelines on controlling future epidemics of FMD to make vaccination more acceptable. The amount of slaughter that took place, particularly in the UK and the Netherlands, is no longer likely to be tolerated by the public. Following the epidemic, British, Dutch and EU politicians made it clear that alternative methods of control, although still allowing slaughter of clinically affected animals and their immediate contacts, must be adopted, without prolonged trade penalties (27).

The total number of animals slaughtered during the epidemic cannot be altered, and the public memory of the mounds of dead animals, funeral pyres and burial pits cannot be erased. Inevitably, the impression remains that a mass slaughter strategy for the control of FMD was inappropriate. But, if a large proportion of the slaughter was, in fact, unnecessary, driven by a policy based on poor science, would there be sufficient justification to replace the traditional approach (which accounted for only part of the slaughter) with one relying more heavily on vaccination? Moreover, should these traditional methods be superseded if the perception that they failed (4, 44) was false?

A major contributor to the slaughter was the novel, automatic pre-emptive culling of all susceptible animals on farms adjacent to infected premises, whether or not there was reasonable suspicion that the virus was present. The perceived merit of this action came from mathematical predictive models, which indicated that it was crucial to bring the epidemic under control. This paper reviews the characteristics, and therefore appropriateness, of the models used as guides to control the 2001 epidemic in the UK, and explores some alternative control methods, including the traditional ones that were applied.

Transmission and infectivity

To control an epidemic of FMD, it is essential to understand the mechanisms by which the FMD virus is being spread because, by breaking viral transmission, the virus is starved of susceptible animals and, after the removal of any persistently infected animals (formally referred to as 'carriers') (80), will soon disappear. While such a concept seems self-evident, and applies to infectious diseases in general, it was not applied during the UK epidemic in 2001. Instead, the tactic recommended by predictive models was to pre-emptively cull susceptible animals that the models themselves predicted to have a higher probability of becoming infected than others, though still not particularly high (e.g. ≤ 26%) (31). There was no requirement to assess the mechanisms and likelihood of transmission in real, individual, cases.

Animals infected with FMD virus produce virus in all their secretions and excretions, particularly in their breath (as aerosols) and in the secretions associated with ruptured vesicles on their feet and in their mouths. The most common route of infection of susceptible animals in an unrestrained epidemic is by contact with animals showing clinical signs, and transmission can occur by the respiratory or oral routes, or through skin abrasions. Transmission of FMD virus can also occur by the movement, and usually subsequent ingestion, of infected animal products, such as milk from an infected cow, or meat from an animal that is slaughtered while still infected with FMD virus (22). This is most probably how infection entered the UK: through illegally imported meat products, some of which were fed, uncooked, to pigs (2). Once in the UK, spread of the virus occurred by the movement of infected animals. The virus also spread mechanically, on the hands, clothes, equipment and vehicles of people who had contact with infected animals, and then, without adequate disinfection, had further contact with susceptible animals. After movement controls were imposed on livestock, this spread by fomites became the major source of transmission, with airborne spread being of minor importance (98).

Infected pigs produce up to 3,000 times more aerosol virus than cattle or sheep (22), but are considerably less susceptible to infection by the aerosol route (23). Apart from the seven characterised antigenically distinct serotypes of FMD virus, which also have their own serotype-specific epidemiological characteristics (58), strains within each serotype vary in their virulence, host specificity and their ability to spread as an aerosol – so much so, that each strain can be considered to be unique (12). There have been examples of long-distance spread of FMD virus as an aerosol. For instance, the outbreak on the Isle of Wight in the UK in 1981 was caused by virus spreading as a plume from an outbreak in pigs in Brittany, France, over a distance of 250 km (25). The atmospheric and topographical conditions required for this degree of spread have been well defined, and spread over land rarely exceeds 10 km (22). An additional important component in calculating the extent of aerosol transmission is the

strain of FMD virus involved, because the quantity of virus particles produced by infected animals varies considerably between strains (23). For example, distances over which the C Noville strain is infectious are likely to be up to 50 times greater than for a strain of the Pan-Asia topotype (a topotype is a group of genomically closely related strains of FMD virus; the Pan-Asia topotype is within the serotype O), for similar donor and recipient species (24, 91). Thus, it is impossible to generalise or predict the behaviour of an epidemic strain without first studying its epidemiology in associated epidemics – if this information is available – and conducting controlled experiments – if time permits.

The FMD virus strain that caused the epidemic in the UK in 2001, and later spread to France, the Netherlands, Northern Ireland and the Republic of Ireland, was one of a group called the Pan-Asia topotype (60). Strains of FMD virus can be individually identified by the nucleotide sequence of the gene that encodes the major structural protein, viral protein 1. The high mutation rate of the virus (20, 21) allows discrimination between strains separated by space or time, and the association of strains with sequences within 5% homology into epidemiologically meaningful clusters. The Pan-Asia topotype was first characterised in India in 1990, and has spread through most of Asia, including those Asian countries previously free of FMD, such as Japan (free since 1908), twice into South Korea (free since 1934), eastern Russia, Mongolia and Taipei China (Taipei China had been infected only two years previously with a pig-specific strain of FMD virus) (60). All these countries successfully eradicated outbreaks due to this strain by slaughtering affected and in-contact animals ('stamping out'), disinfection, controlling animal movements and, in South Korea, vaccination around the infected farms. None reported any significant aerosol spread of the virus between farms (78).

Trail of infection in the United Kingdom in 2001

The first infected farm in the UK epidemic was almost certainly a pig farm of approximately 500 pigs that were fed waste food. It was identified on 22 February, near Newcastle-upon-Tyne. The virus had probably been on the farm since the beginning of February, but the disease had not been reported by the farmer (1). If a worst-case scenario is assumed, and the infected pigs were producing aerosol virus in quantities consistent with infection due to the C Noville strain of serotype C (known to be produced in large quantities in pigs) (23), enough virus would have been generated for infection to have spread beyond the coasts of Denmark and Germany, assuming weather conditions in the previous three weeks were sufficiently compatible (70). Certainly, a large amount of virus would have covered the countryside surrounding the infected farm. Subsequent investigations showed that during that time, consistent with the epidemiology of strains in the Pan-Asia topotype, only ten farms in the area were likely to have been infected by long-distance aerosol, ranging in distance from 1 km to 9 km from the infected pig farm (39). Since infected pigs produce up to 3,000 times more aerosol virus than sheep or cattle, 500 infected pigs would be equivalent to 1,500,000 infected sheep or cattle. Yet, they had failed to spread an infectious dose of aerosol virus to more than ten nearby farms in a three-week period. An adjacent dairy farm remained uninfected throughout the whole epidemic. It was clear by the beginning of March that the epidemic strain was not spreading any significant distance as an aerosol, and the reduced aerosol output from pigs infected with this strain of FMD virus was soon confirmed experimentally (24).

Unfortunately, one of the farms infected by airborne aerosols from the pig farm was a mixed beef and sheep farm (39), and sheep from this farm that were incubating disease had been sent to market before the disease was recognised (36, 67). Sheep thus became the predominant species involved in the early spread of the epidemic. By the time the first case of FMD had been diagnosed, infections had already been spread by the movement of infected animals (mostly sheep) across the northern counties of England and as far south as Essex and Devon, so that 10 of the 12 geographic clusters that developed were infected (67). However, previous investigations in Italy during the 1993 outbreak, and in Greece during the 1994 and 1996 outbreaks, in which sheep were also affected, indicated that the spread and maintenance of FMD within sheep flocks was dissimilar to that in cattle and pig herds (59). Spread between sheep is often slow, and the virus may disappear before all the sheep are infected, as exemplified by low levels of seroconversion in infected flocks in Europe and North Africa (63, 65, 66). Thus, in 2001, after a complete national ban on the movement of FMD-susceptible animals was implemented on 23 February, there was no reason to assume that sheep would continue to drive the propagation of the epidemic. The low prevalence of infection within affected sheep flocks, and their low individual virus production (24), would make them a relatively low risk for spreading aerosol virus (probably to a maximum distance of less than 100 m) (24). Sheep would also pose a lower risk for spread by fomites, because less virus was being released into the environment.

So, how was the virus able to continue spreading after implementation of the national animal movement ban? Almost 80% of the virus spread in 2001 was classified as 'local' (36). However, without an explanation of how the spread had occurred, this really only reflected the difficulty of quickly and positively identifying sources of infection, due to the pressure on veterinary resources and the complexity of the real situation, where it is often

impossible to pinpoint actual sources and spread events. 'Local' spread (36, 81) was defined as having occurred if new infected premises were within 3 km of previously confirmed, infected premises, and if more than one possible conveyor of infection was identified. This does not, therefore, imply radial spread from infected premises (somewhat like the ripples that occur when a pebble is dropped into a pond) and bears little relation to the location of infected animals and actual contacts between them. Thus, it does not adequately describe the transmission mechanisms (e.g. direct contact between infected and susceptible animals, or short- or long-distance spread by fomites) at which control measures are targeted.

Possible mechanisms of FMD spread have been described above, and the only routes that seem likely, assuming compliance with the movement ban, would be:

– by very short-range (less than 100 m) aerosol transmission from affected flocks or herds until slaughter took place. However, where cattle were affected, these were predominantly housed until May, usually over 100 m from neighbouring herds;

– by mechanical carriage of the virus from infected to susceptible animals;

– through the movement of infected animal products, such as milk.

Subsequent analyses of field data revealed that only 5% of the premises contiguous to infected premises may have become infected by direct animal-to-animal aerosol transmission (49, 98), but that short- and long-distance spread by fomites was likely to be of major significance. Forty-three percent of infected premises in the Cumbria region of the UK (where over 40% of cases occurred) were more than 1.5 km away from the nearest infected premises that could have transmitted FMD virus to them (94). A significant proportion of affected premises in south-west Scotland were over 3 km from the nearest infected premises that could have transmitted infection to them, particularly during the last half of the epidemic (98). Spread by fomites was shown to be the main route of infection in the south-west of Scotland (98).

Control measures

Traditional measures

Before compulsory vaccination was introduced to mainland Europe in the 1960s, up to 30,000 outbreaks of FMD were reported each year. However, this was reduced to a few hundred a year within a decade, using a combination of vaccination, movement controls, biosecurity measures and co-operation between European countries, under the umbrella of the European Commission for the Control of FMD (57).

The duration of the 1967 to 1968 and 2001 UK epidemics was almost identical, and the epidemic curves showing the number of new affected farms per day were also very similar. Both epidemics began with the wide dissemination of infection. In 1967, this was due to the distribution of infected meat imported from South America, and in 2001 it was caused by the spread of infection among sheep at Longtown Market in north-west England, and their subsequent dispersal (14, 67, 88). Farming conditions in 1967 were different from conditions in 2001 and, in the 1967 to 1968 epidemic, predominantly pigs and cattle were involved. The lower aerosol production of the Pan-Asia O topotype of the virus should have made the 2001 epidemic easier to control than that of 1967 to 1968, as soon as effective movement restrictions and biosecurity were in place. A complete ban on the movement of FMD-susceptible animals was implemented on 23 February 2001. This differed from the 1967 epidemic, which had run for over three weeks before a national movement ban was enacted in England and Wales, and it was a further week before Scotland was included (13).

A key traditional control strategy is rapid detection and slaughter of all susceptible animals on infected premises, and identification of dangerous contacts (i.e. susceptible animals considered to have been exposed to infection) by tracing and veterinary assessment. Analyses have demonstrated the efficacy of traditional rapid slaughter on infected farms in controlling the 2001 epidemic (48, 97). Movements on and off infected premises during the previous 21 days, particularly of animals, people and vehicles, were traced in an effort to identify potential sources and the spread of infection. The danger and degree of exposure to infection of livestock on traced premises and premises next to infected premises was the subject of veterinary assessment. If it was judged that the likelihood of exposure to infection was high, the animals were classed as dangerous contacts, and all susceptible animals were slaughtered as soon as possible. If the judgement was that the likelihood of infection was low, but it could not be ruled out entirely, the premises were placed under restriction, the livestock isolated, and regular veterinary surveillance inspections were implemented.

Biosecurity measures were also taken to limit the spread of the disease. A 'Protection Zone', with a 3-km radius, and a 'Surveillance Zone', with a minimum radius of 10 km, were established around each infected farm, within which stringent movement restrictions were applied to animals, people and vehicles. Livestock within the zones were subject to veterinary surveillance. Later in the epidemic, larger 'Restricted Infected Areas' were defined, within which strict biosecurity measures and movement controls could be enforced.

During the 1967 to 1968 epidemic of FMD in the UK, in which 2,364 farms were clinically affected, 442,000 animals were slaughtered, compared with over four million in the 2001 epidemic (1). 'Traditional' slaughter was applied less aggressively in 1967 to 1968: dangerous contact animals were only slaughtered if they had arrived on a farm within 72 hours (h) of putative contact, otherwise they were isolated and subjected to regular clinical inspection (13). In 2001, all dangerous contacts were generally slaughtered. Also, in 1967 to 1968, following the tracing of dangerous contacts to Oswestry Market, the animals in the market (of which there were 3,299) were moved under licence to farms within the Protection Zone, and none developed the disease (13, 76). In 2001, sheep traced through certain markets were identified and slaughtered (102).

Model-driven control strategies

Early in the 2001 epidemic, policy decisions about the control programme were removed from the Ministry of Agriculture, Fisheries and Food (MAFF), now known as the Department for Environment, Food and Rural Affairs (DEFRA), and placed with the Cabinet Office Briefing Room (COBR) (also known as COBRA because it was Room A). Campbell and Lee (9) comment that: 'The incredible state of affairs in which a regulatory problem of livestock rearing and farm economics was thought to require a response by a government apparatus designed to deal with problems more akin to general insurrection has passed with little other than approving comment in the official reports'. (COBR was convened most recently in July 2005, in connection with the terrorist bombings in London.)

The COBR was advised by the Chief Scientific Adviser to the Government, who had established a 'Science Group' to help formulate this advice. The Science Group, which first met on 26 March, was dominated by four teams of modellers. To quote the then current MAFF Chief Scientific Adviser, David Shannon (89): '… A formally constituted scientific advisory committee would have looked considerably different'. One team had already, on 21 March, used the media to disseminate its dire predictions to the public on the eventual outcome of the epidemic (the course of which would include a general election), unless its advice was followed. The issue then assumed both technical and political dimensions, with the danger of scientists expressing 'convictions or opinions which (however scientifically founded) cannot in any way be identified with knowledge in the strict sense which science generally affords this term' (107).

The involvement of modelling with the control programme for the FMD epidemic was not part of the pre-arranged contingency plan, but came about in an ad hoc way. The 'Lessons to be Learned' inquiry report (2) describes how Sir John Krebs, Chairman of the Food Standards Agency, began speaking to a number of mathematical modellers in late February 2001, soon after the epidemic began. An ad hoc meeting, organised by Krebs, took place on 6 March. Mathematical modellers from Imperial College, London, Cambridge University and Edinburgh University gathered and discussed the data requirements for modelling the epidemic (45). Then MAFF supplied the data requested on 13 March and the groups of modellers began their analyses.

The 'Lessons to be Learned' inquiry report (2) states that the Imperial College group were furthest advanced at that stage and reported their initial findings to MAFF on 16 March. According to the report, the main advice at that point was that the delay between the reporting of the disease on premises and subsequent slaughter must be reduced. This advice simply reiterated longstanding doctrine (13), which has subsequently been confirmed by analysis of the field data (48, 98).

A meeting between the mathematical modellers, the Chief Scientific Adviser to the Government, the Chief Veterinary Officer and experts from the Institute of Animal Health and the Veterinary Laboratories Agency occurred on 21 March (101). Between 21 and 26 March, the modellers examined the potential effects of various culling policies. On 23 March, instructions (105) were issued to slaughter, as dangerous contacts, all FMD-susceptible stock (cattle, pigs, sheep/goats, llamas, alpacas) on premises that shared a boundary with an infected site that had been confirmed on or after 16 March. Following the first meeting of the FMD Science Group, on 26 March, instructions were issued to confirm disease on clinical signs alone, without awaiting laboratory confirmation for doubtful cases. In addition, all FMD-susceptible species on the infected premises were required to be slaughtered within 24 h of confirmation, and all susceptible animals on premises contiguous to an infected site, as well as on other farms designated as dangerous contacts, within 48 h (the '24/48 h' policy) (103). The classification of contiguous premises as dangerous contacts became automatic (i.e. not subject to veterinary assessment) on 29 March (104), (the 'pre-emptive contiguous cull') (100).

While such a policy might be practical (if not scientifically justifiable) if only a small number of farms are affected (as in the Republic of Ireland in 2001) (42), implementing this policy for the UK epidemic – in which the disease was spread over a wide geographical area – resulted in the destruction of many healthy animals and logistical problems of carcass disposal. Not surprisingly, the tourist industry was adversely affected by the images of cattle destruction and the movement restrictions for walkers in the countryside (8, 14, 29, 30). Mrs Wendy Vere, a West Country veterinarian, commented to the *Devon Independent*

Inquiry: 'Their idea was to control the disease by culling in contiguous farms. That is fine if you are sitting in front of a computer screen in London. However, it is different on the ground. A person in London will just see the numbers and will say that they have been taken out. That is why it was carnage by computer' (9, 84). This graphically exemplifies the isolation and abstraction of 'armchair epidemiology', whilst also poignantly highlighting the importance of personal involvement in disease control to gain complete insight into its impact. This concept is already well established in the social sciences, where feelings, responses and experience are considered necessary for a full understanding of reality (so-called 'experiential analysis') (75).

The peak in the number of confirmed new outbreaks occurred on approximately 29 March. If, as calculated by Woolhouse (112), the time between infection entering premises and those premises being declared infected was approximately eight days, then the new infections peaked on 21 March, almost a week before the 24/48 h policy was introduced (45). This is consistent with the conclusions of analyses of the epidemic (37, 44, 98), but conflicts with the notion that pre-emptive contiguous culling was an essential component of effective control, as proposed by various mathematical models (3, 41, 56, 109). In fact, the epidemic peak preceded the start of pre-emptive contiguous culling. Significantly, more than two weeks after the epidemic peaked, models failed to identify the time at which the epidemic was under control (86). Subsequently, modelling groups conceded that the epidemic was coming under control faster than the models had predicted (87), and that model projections of the course of the epidemic might have been 'over pessimistic' (85). For example, Ferguson *et al.* (31) estimated that, in the Cumbria, Dumfries and Galloway area, 79% of 5,000 farms would be infected (3,950 infected premises) by 28 March, assuming the model parameters remained unchanged from the status quo. The actual result in the main epidemic focus in North Cumbria (where, significantly, the automatic pre-emptive contiguous cull could not be implemented due to resource constraints) was that 24% of the 2,684 farms within the 3-km Protection Zone became infected and, over all, 50% were depopulated, leaving approximately 50% of premises with livestock (94).

Modelling in perspective

Modelling and scientific method

The disparity between the course of the 2001 epidemic and the model predictions demands an explanation. The numerical output of models has an air of intellectual superiority (noting that: '…mathematized theory in science is rarely so pellucid or so rigorous that its significance and bearing can be grasped immediately by distant readers' [74]), while also seeming entirely appropriate in a society where numbers can '… reassure by appearing to extend control, precision and knowledge beyond their real limits… wrong numbers, one might add, are worst of all because all numbers pose as true' (11). Numbers, therefore, may convey an illusion of certainty and security that is not warranted (43); for example, because of the use of whatever numerical data are available, regardless of their relevance and quality (38).

A model constitutes a theory, and a predictive model is therefore only a theoretical projection. This is clearly illustrated by the different conclusions generated by different models addressing the same issue (6, 26). A theory cannot be formally proved in the sense that propositions in logic and mathematics can. The degree of confidence in a theory depends on several factors (15), the most relevant of which are 'testedness' and 'refutedness'. To be well tested, a theory should provide predictions of what would happen in a variety of different circumstances. If these predictions have been extensively tested over a wide range of conditions (either in the field or experimentally), then the theory can be called well tested. 'Refutedness', as viewed by Bertrand Russell, refers to 'external confirmation': the theory must not contradict empirical facts. The degree of confidence in the 2001 predictive models is therefore low because they were not widely tested, and their conclusions (e.g. that pre-emptive contiguous culling was necessary to control the epidemic) have been refuted. Moreover, there are constraints on testedness in any case, because of the rarity of FMD epidemics, and the genetic plasticity of the organism, which can result in strain variation with consequent changes in the transmission characteristics of the virus. Predicting a chance, long-distance transmission event on the virus-contaminated hands of an unsuspecting stock-owner would also be impossible, other than to say it might occur. But researchers would be unable to specify where or when; and clearly this would vary, both over time in one epidemic, and between different epidemics. Additionally, models generated to assist in the control of a specific epidemic are 'tactical' rather than 'strategic' (46), and this further limits their testedness.

Appropriateness of model use in 2001

The stages in model-building are well documented (17, 68, 93). The model requires appropriate input parameters that accurately reflect the behaviour of the system that is being modelled. It must then be seeded with data. The level of understanding of the system, and the quality of the available data, determine the appropriate application of the model (Table I). Prediction should only be attempted if both are good. Finally, the model needs to be validated, to

Table I
Appropriate use of models in the context of epidemiological knowledge and data quality (47, 93, 96)

Epidemiological knowledge	Data quality and quantity	
	Poor	**Good**
Poor	Exploration of hypotheses	Hypothesis testing
Good	Simplified representation of past events, and guarded use for prediction of future events	Detailed representation of past events, and prediction of future events

establish if it behaves like the actual biological system that the model is designed to mirror. This should be undertaken by assessing the model against data not used in its construction (92).

The 2001 predictive models were constructed in an environment of poor-quality data (e.g. they used out-of-date census data for stock levels), and poor epidemiological knowledge (e.g. the transmission characteristics of the virus strain, and the distribution of the initially infected farms, were unknown). Therefore, their use as predictive tools was inappropriate.

An area of uncertain epidemiological knowledge that was crucial to the modelling was that of the source and spread linkages between infected premises. Modellers (31, 56, 110) used MAFF contact-tracing data to estimate key model parameters, including:

– the distance between source-infected premises and spread-infected premises

– the onset of infectivity in relation to the infection date and reporting date

– changes in the level of infectivity over time.

These parameters would have been crucial to model predictions about the necessity of a pre-emptive contiguous cull, as discussed below, but the tracing data (provided to the modellers on 13 March – just three weeks into the epidemic) were incomplete. For example, as the result of careful work during and after the epidemic, Mansley *et al.* (67) identified approximately 115 premises that were possibly already infected (through infected animals from Hexham and Longtown Markets) when the movement ban was imposed on 23 February. This information was not available to the modellers in March when they were advising on policy. In fact, it was thought at the time that, during the first week of the epidemic (20 to 26 February), only seven premises were infected (2). This would have had a profound effect on their calculations of R_0, the basic reproductive number (19), because the appearance of so many infected premises within a short period of time would have been assumed to result from continuing rapid propagation of the epidemic. Consequently, the modellers would have over-estimated the number of premises infected just after the movement ban (44). These early calculations provided the support for the announcement on 21 March by one of the Imperial College team that the epidemic was not under control (4). Furthermore, the model of Ferguson *et al.* (31) did not include separate species, and so modelled all farms with the same 'homogenised' species, even though virus output varies substantially between species.

There must have been many infected premises for which the source was either wrongly identified, identified simply as 'local', or identified as unknown. A definite source of infection was established for relatively few of the infected premises in 2001. According to Gibbens and Wilesmith (37), out of a total of 2,026 infected premises, a definite source was only identified for 101 (5%). In the absence of a definite source of infection, it was common to attribute the source to the nearest possible candidate infected premises (110) – a naïve exercise. One result of such inaccuracy in assigning the correct sources to infected premises appears to have been that the models incorporated parameters that conflicted with the known biology of the virus (as described earlier).

These conflicts centred around the periods between infection, the onset of infectivity and the onset of clinical signs (after which the case could be reported). Specifically, the models represented farms as becoming infectious one (32), three (31) or four (56) days after infection. These estimates allowed time for the disease to spread (in the models) before the appearance of clinical signs and reporting the disease. For example, in one model, animals become infectious five days before clinical signs appeared (56). These timings are at variance with the well-known variability in the incubation periods of infectious diseases (73, 82, 83), and ignore the effects of both species and the number of animals in determining virus output.

Not only did the models simulate a very early onset of infectivity, but that infectivity was modelled as immediately maximal and constant until the slaughter of the animals on the infected premises, implying that all animals were simultaneously infected. For example, Ferguson *et al.* (31): 'assumed constant infectiousness from three days after infection until slaughter (for an average of eight infectious days)', and, in a later model-based analysis, the same authors assumed constant infectiousness from the day after a farm is infected to the day its animals are culled (32). This ignores the well-established phenomenon of the intra-herd epidemic, where some animals in a herd may be infected up to a month after initial cases, depending on stock location and farm structure (52). Clear evidence of

the occurrence of an intra-herd epidemic on an infected site is provided by the clinical picture of the pig farm infected at the start of the epidemic (1). Here, pigs were found with a range of lesions, indicating that infection began in a few pigs (those with the oldest lesions), then spread to others over a period of time. The presence of seropositive animals with no lesions suggested even older infection. Work on dairy farms in Saudi Arabia (52) and in experimental infections (51) indicates that within-farm prevalence increases over time, and so the amount of virus being shed will also increase in the first few days of a clinical infection on a farm. Further evidence was also provided by field analysis of the 1967 to 1968 epidemic, where the herd serial interval was more than twice the mean incubation period, indicating that at least two cycles of infection (the second amplifying the first) were necessary before farms became infectious (50). This would suggest that the infectivity of an infected farm increases over time. It is clear that infected premises could not be equally infectious throughout the course of infection because, initially, only one or two animals would be infected. Moreover, particularly on cattle and pig farms, levels of infection would increase as the number of animals infected at a given time increased. Yet the possibility of an intra-herd epidemic was specifically excluded from the predictive models produced during the 2001 epidemic. Keeling *et al.* commented that (56): 'There has been some speculation about the role and existence of a within-farm epidemic. Clearly, if initially just one animal was infected, then there should be a build up of the within-farm epidemic over time and hence an increase in the farm's infectivity. However, there is no evidence for such a build up from the data – the rate at which secondary cases are generated is approximately constant throughout the infectious period. This may be due to the aggregated nature of infection, such that many livestock on a farm get infected at any one time.'

One important effect of simulating an epidemic where a rapid onset of infectivity to maximum levels occurs, resulting in a high proportion of infection spread before clinical signs appear, is that the value of rapid culling of the infected premises would be underestimated, because diseases with these particular characteristics require some form of pre-emptive action to bring them under control (34). It appears that the models did underestimate the true value of rapid culling of infected premises, because retrospective analyses have demonstrated the key role of culling speed in controlling the epidemic (48, 49, 97), whereas the predictive models advised that rapid culling alone would fail to control FMD (31, 56).

The models of Ferguson *et al.* (31, 32) and Keeling *et al.* (55, 56) addressed the spread of disease in a population of farms using 'black box' probabilities of infection, without attempting to model the actual mechanisms of disease spread (93). Central to the 'black box' approach is the 'spatial kernel', a probability construct that describes the probability of infection as a function of distance between infectious and susceptible farms. Construction of the spatial kernel also depended on the MAFF contact-tracing data referred to above. Ferguson *et al.* (31) state: 'contact tracing for all FMD-affected farms has produced unique data on the spatial scale of disease transmission, clearly demonstrating that farms closest to index cases of FMD are at greatest risk of infection … We estimate that farms 0.5 km, 1 km and 1.5 km away from a single farm affected by FMD would have probabilities 0.26, 0.06 and 0.02, respectively, of becoming infected.'

Modelling disease spread in this way, especially with spatial kernels that are heavily weighted towards short-distance spread, tends to produce simulations that show 'centripetal' spread of disease – that is, disease spreading radially and over short-distance increments from the initial seeded infection. See, for example, the animated simulation provided by Keeling *et al.* (55) in supplementary material to a recent paper. This pattern of spread is at variance with reality. A 2005 DEFRA project report (18) describes the spatial pattern of FMD spread in north Cumbria, subsequent to initial seeding, in which a high proportion of cases occur, with no possible source of infection within 1.5 km, in the first four or five weeks, rapidly expanding the confluent 3-km Protection Zone to almost its maximum extent before the majority of later cases 'filled in' the gaps between old infected premises. Disease that truly spreads centripetally could logically be tackled by pre-emptive culling of contiguous premises, but it is hard to see how this approach, rigorously applied from when the first case was confirmed, could have successfully prevented the rapid scattering of early cases that occurred in north Cumbria in reality.

As discussed above, and as the modellers themselves commented (32, 56), the tracing data, especially that provided early in the epidemic, would be biased towards short-distance transmission. Ferguson *et al.* (32) indicate that this could have been the case when writing about an analysis conducted later in the epidemic. 'The newly estimated spatial kernel differed significantly from that previously derived from the infectious contacts identified by DEFRA (MAFF), with considerably more long-distance transmission events being predicted.… The median distance of the newly estimated kernel is about 4 km, suggesting that most transmission probably occurred through the movement of animals, personnel or vehicles, rather than through animal contact or wind-borne spread.' Keeling *et al.* (56) also recognised that the tracing data could be biased and that this could be critical to the models they produced: 'it is crucial to quantify the spatial infection kernel or, at least, relative contributions of local and non-local spread …The contact tracing is probably biased towards short-distance infection, which may cause a similar bias in the transmission kernel.' In addition,

retrospective analyses of the field data have shown that a significant proportion of infected premises were further than 3 km from a possible source of infection (94, 98). Taylor *et al.* (94) indicate that the risk of infection faced by premises that did not neighbour an infected farm was sufficient to allow the tail of the epidemic in the area south of Penrith to continue to propagate, despite pre-emptive contiguous culling.

The significance of this is that a model with an unrealistically narrow kernel (i.e. where most disease transmission is over short distances) would tend to overestimate the efficiency of a local pre-emptive culling policy (i.e. culling premises contiguous to infected premises). In a paper describing an adapted version of their 2001 model to guide vaccination, Keeling *et al.* (55) admit: 'in terms of the total number of farms affected by the outbreak, wide diffuse kernels would mean that contiguous premises culling is an inefficient strategy as the infection is far less localised on the neighbouring contiguous farms'.

A further simplification in the models was the use of constant (throughout the duration of the epidemic) transmission parameters (31, 56). Such models are unable to include the possible disease control effect of improvements in biosecurity (e.g. the Restricted Infected Area regulations applied late in the epidemic) and were therefore limited to modelling control policies based on culling and/or vaccination only. Thus, these models were incomplete in an important area of decision-making for control policy.

Ferguson *et al.* (32) later modelled the epidemic using a model that allowed transmission rates to vary over time and concluded that changes in culling policies explained less than 50% of the observed variation in transmission rates, which in turn indicated that effective movement restrictions and rigorously maintained biosecurity were equally vital in reducing disease spread. This would suggest that the role of the contiguous cull in controlling the epidemic was less crucial than proposed by the earlier model.

The model that Ferguson *et al.* (31) presented to the Science Group in late March probably had the most influence on early policy decisions (93), specifically, the introduction of the pre-emptive contiguous culling policy. However, this model, and the Keeling *et al.* model (56) that was used to corroborate it, were assigned parameters that could not help but favour that policy, based on field tracing data that should have been viewed with caution. The models were highly sensitive to the accuracy of this information, in that these data determined the degree of disease transmission to be simulated before clinical signs and the distance over which the majority of transmission took place. Despite the fact that the modellers seemed to be aware of these issues, the models were used as strong support for the implementation of the contiguous cull.

The authors of this paper argue that the models were not fit for the purpose of predicting the course of the epidemic and the effects of control measures. The models also remain unvalidated. Their use in predicting the effects of control strategies was therefore imprudent.

In retrospect, very little of value was added to the FMD control policy by the use of predictive models. The latter therefore failed the most pragmatic 'litmus test': namely, usefulness (40; Hugh-Jones, quoted by 79). The key question for any model is whether decisions made with it are more correct than those made without it (17). However, the consequences of following the recommendations of these models were severe: economically, in terms of cost to the country; socially, in terms of misery and even suicides among those involved in the slaughter programme; and scientifically, in the abuse of predictive models, and their possible ultimate adverse effects on disease control policy in the future (see below).

In his description of the value of models during the 2001 epidemic (111), Woolhouse emphasised that, 'mathematical models should be one of the tools available to policy-makers', but that they are not, 'a substitute for experience and expertise in the control of FMD'; a view shared by Kao (54) when he said, 'all theoretical models are only one aspect to providing good scientific advice'.

During the 2001 epidemic, MAFF/DEFRA were using another model, InterSpread, on a daily basis to monitor the progress of the epidemic (71), but this model was not prominent in the decision-making process that led to the contiguous cull. The model was run to give regularly updated predictions of the overall size, duration and spatial extent of the epidemic. As with the other models, assumptions about the start of the infectious period were needed. Morris *et al.* (72) mention that, in InterSpread, infectivity starts on or just before clinical signs appear, stops when control measures are completed (i.e. the end of slaughter), and varies according to both the stage of disease and control measures. Both these characteristics (onset of infectivity and variability of infectivity over time) therefore differ considerably from those of the other models. InterSpread is a very detailed simulation model which attempts to represent the transmission of disease by specific contact routes, rather than using the 'black box' transmission kernel, and also includes the effects of differences between species on disease transmission, as well as other farm-level factors. This model therefore includes many epidemiological parameters and control strategy definitions. Most of these parameters were assigned values that were based on data from the 1967 to 1968 epidemic and a review of the literature, although the parameter governing the tendency for airborne spread was

reduced, to reflect knowledge about the behaviour of the 2001 epidemic.

When used to assess different control options early in the epidemic, InterSpread predicted optimal control of the epidemic if slaughter was achieved on infected premises within 24 h, and an average of between 1.1 and 1.4 premises were pre-emptively culled per infected set of premises (72) – a far lower level of pre-emptive culling than automatic contiguous culling. It should be noted that InterSpread uses assumptions about the sensitivity and specificity of the identification of dangerous contacts that simulate 'veterinary assessment', rather than selection based on the 'proximity of premises' to infected premises.

As InterSpread explicitly models different spread mechanisms, such as: movement of animals, airborne spread, spread by milk tankers and spread by other vehicles, it could be used by veterinary epidemiologists as an interactive tool to assist in understanding the field situation. When the real situation varied from the modelled situation, adjustments could be made to the modelled transmission mechanisms to understand what may be happening in the field. In fact, InterSpread modelling conclusions coincided with field observations that the continued spread of disease during the epidemic tail south of Penrith was largely being mediated by the movement of people or animals. This provided support for the introduction of improved biosecurity measures in the area (Restricted Infected Areas, in which movement and cleansing and disinfection of farm traffic were intensively targeted by the authorities), which finally brought the epidemic to a close.

In a lecture to the Royal College of Veterinary Surgeons (reported in the *Veterinary Record*, 26 July 2003), Lord May acknowledged that the use of mathematical models during the 2001 FMD epidemic had created controversy (nothing new, when mathematics 'invades' hitherto non-quantitative domains) (90), but suggested it was based on a lack of mutual understanding between veterinarians and modellers (69). But Lord May was not present during the Science Group meetings, in which models based on inadequate and inaccurate information were being used to formulate recommendations to the Prime Minister's Office to initiate changes in the FMD control policy. This was despite the fact that alternative, tried and tested strategies were being proposed by experienced veterinarians from the FMD World Reference Laboratory and MAFF. The 'Lessons to be Learned' inquiry reported that it was: 'unable to find a clear account of decision-making around that time' (2), highlighting problems within the FMD Science Group. The group was criticised as being a 'modelling sub-committee' although experts from other scientific disciplines were present. At times there were polarised views within the group but no mechanism for handling such conflict (2). It is not necessary to be mathematically literate to appreciate that no model will produce the right output when fed the wrong input. In the future, care should be taken to ensure that lessons are learned – a bad model is like a bad x-ray because it invariably results in erroneous conclusions and a wrong course of action.

It is inevitable that modellers will seek to improve their models. However, they tend to focus their attention on tractable issues of 'uncertainty' (that is, focusing on influential parameters whose probabilities are not known). This is common to scientific research, which thus ignores parameters that are less amenable to investigation (61). The net result is that, as uncertainty is decreased, 'ignorance' (essentially: not knowing what one does not know), which is a measure of the completeness and value of knowledge, increases (116). This apparent paradox therefore acts as a cautionary warning over the use of ever-more-detailed models as policy guides. Moreover, modelling will never present the full picture because, 'rarely if ever is a mechanism proposed that would account for all observed cases of disease, or all effects of all risk factors, measured and unmeasured. Background "noise", in the form of unaccounted-for effects and interactions, would easily obliterate any pattern sought for by the investigator' (77).

Alternative approaches to control

Recent analyses of the epidemic data have indicated that there was no significant relationship between the use of contiguous culling and the spread of infection (48, 97). Doubts have also been raised about the legality of the cull (10), where intervention 'occurred beyond formal legal doctrine' (62). This concern undoubtedly contributed to the speed with which new legislation, the Animal Health Act, 2002, for England and Wales (99), was enacted after the epidemic. The Act provides extended power to carry out any slaughter deemed necessary to control disease, dubbed by some as: 'the power to panic' (10).

Note, however, the consequences of such power. In 2001, only 65% of the 2,026 'infected' premises were confirmed positive by diagnostic analyses of the samples submitted to the laboratory at the Institute for Animal Health, Pirbright: 1.3 million animals were slaughtered on infected premises; 1.5 million animals were slaughtered on contact farms; and 1.2 million animals were slaughtered on adjacent premises, some of which were also considered dangerous contacts (33). On these figures alone, approximately three million healthy animals were slaughtered to control the epidemic, even allowing for the possibility that another 12% of the

infected premises were actually infected (i.e. 77% of the declared infected premises).

Farm restrictions, clinical surveillance and testing

The implementation of the pre-emptive contiguous cull policy meant that premises were declared infected from diagnosis on clinical signs alone, without laboratory confirmation. Clinical FMD in sheep can be very mild (59), and is easily confused with other causes of mouth ulceration or lameness (16). Not only were sheep flocks (and some cattle herds) being culled due to mistaken clinical diagnoses (as indicated by negative laboratory results when samples were taken), but the animals on adjacent farms were also slaughtered under the provision of the 48-h contiguous cull policy. The diagnostic tests used in the laboratories were extremely sensitive and specific for showing evidence of infection with FMD virus (114), and, assuming those animals suspected of FMD were the animals from which the samples were collected, and the samples were kept cool and submitted to the laboratory within 48 h (which they were), these results would give a very good indication of which flocks and herds had been infected. There was no compelling reason why the slaughter, particularly of sheep, which have low virus excretion rates (59), could not have been delayed until laboratory results were available. In some instances, laboratory results showed that the FMD virus had actually been in a flock for some considerable time before slaughter, and yet spread had not occurred to other farms (1). Therefore, in many suspect flocks awaiting the results of testing, the sheep could easily have been isolated and restricted at a distance from neighbouring animals and farms, to reduce the risk of potential virus spread (should the virus, in fact, have been present).

Veterinary-assessed dangerous contact culling

The value of using veterinary judgement in deciding which premises were at high risk of having been infected (i.e. incubating disease), and therefore which ones to cull – rather than using an automatic contiguous culling regime – was assessed from the results of the control programme in Cumbria (49). These results showed that automatic contiguous culling was unnecessary (49), and could be replaced by applying basic epidemiological principles to decide the risk of exposure to infection. Analysis of data in south-west Scotland also indicated the efficiency of veterinary assessment in detecting infection, while also failing to detect infection on any automatically contiguously culled premises, although many of these (most of which had the indicator species, cattle, on them) were slaughtered beyond the median incubation period of infection in relation to the time when adjacent premises could have infected them (97).

Control by vaccination

The reality of the 2001 epidemic of FMD in the UK was that more than six million animals were slaughtered to control disease and maintain animal welfare. In the Netherlands FMD epidemic, 60,000 animals (predominantly cattle) were slaughtered to help control the epidemic, and a further 200,000 FMD-susceptible animals, which had been vaccinated in the area surrounding the main focus of the epidemic, were also slaughtered to help quickly re-establish international trading status. The UK did not use vaccination to assist in controlling the epidemic, although vaccination programmes were planned, vaccination teams were trained and 50,000 doses of vaccine were ordered and ready for use in Cumbria. There was considerable discussion about potential vaccine use, but the issue was where to use it, since it was not initially clear where the disease was distributed, and there was concern that there would be no market for milk or meat from vaccinated cattle – even though deboned meat from vaccinated cattle in South America had been sold in the UK for 50 years. The MAFF officers acknowledged that the use of vaccination would primarily be to help relieve the limited resources then available, and that any vaccinated animals would later be slaughtered. Not unreasonably, if the animals were to be slaughtered after the epidemic, and if there was likely to be a problem selling the milk (some supermarket chains had indicated their unwillingness to sell milk from vaccinated cattle), farmers preferred their compensation during the epidemic, rather than later.

The World Organisation for Animal Health (OIE) defines guidelines for bilateral trade agreements involving live animals and animal products, to reduce the spread of diseases between countries, in particular, highly infectious diseases such as FMD (115). The OIE also advises the World Trade Organization on animal-related trade disputes. The OIE *International Animal Health Code* at the time of the epidemic specified that a country previously free of FMD, which had used vaccination to help control an epidemic, and which had not subsequently slaughtered all the vaccinated animals, could not re-apply for FMD-free status until 12 months after the last use of vaccination (115). If all the vaccinated animals were slaughtered, an application could be made three months after the slaughter of the last vaccinated animal, together with evidence that the virus had been eliminated (115). These conditions were based on the possibility that vaccinated cattle and sheep that had contact with live FMD virus during the epidemic could become persistently infected, and therefore cause fresh outbreaks.

The political repercussions of the 2001 FMD epidemic in Europe manifested themselves in a meeting in Brussels in December 2001, sponsored by the British and Dutch governments and the EU. At this meeting (27), Ministers

from both countries made it very clear that the slaughter that had taken place to control the FMD epidemics was no longer acceptable and alternative policies were required, notwithstanding the fact that much of the slaughter resulted from the nugatory pre-emptive cull, which need not – indeed, should not – be repeated in the future. Nevertheless, attention then focused on vaccination. Considerable publicity was given to a serological test that would distinguish animals that tested positive for antibodies against FMD virus following infection, from those that were positive following vaccination. In this way, persistently infected animals could be identified.

The test identified antibodies to the non-structural proteins (NSP) of FMD virus, in particular 3ABC (64). The vaccine against FMD is an inactivated preparation, and there is no viral replication or expression of the NSP, therefore few or no antibodies are made to these proteins. An infected and recovered animal would have NSP antibodies and thus be easily identifiable.

What the new test did not fully address was that vaccinated cattle and sheep that come into contact with live FMD virus can become persistently infected, without showing clinical signs or producing detectable antibodies to NSP (19). However, the use of high-potency vaccines may reduce the development of persistent infections (7).

The test had also not been validated to the standards set by the OIE for any species. However, in 2002, the OIE agreed to change the requirements for re-establishing FMD-free status: to six months after the last vaccination if the vaccinated animals were not slaughtered, and after the use of the NSP test to show that the FMD virus had been eradicated from the affected country. Questions arose from delegates of OIE Member Countries, when this was presented to the OIE International Committee in May 2002, because little was known of this test and its diagnostic sensitivity and specificity, particularly in vaccinated, persistently infected animals. Nevertheless, the political pressure for change was overwhelming.

Following the OIE decision, the EU also changed its Directive on measures for the control of FMD. No longer is vaccination considered the last resort, and considerable reliance is being placed on the use of the NSP test to mitigate its consequences. However, in spite of political assurances that using the NSP test will overcome the potential dangers of persistently infected animals, the new EU Directive states that, following a declaration of FMD freedom in a Member State: 'the dispatch from one Member State to another Member State of susceptible species vaccinated against FMD shall be prohibited'. It is probable that, if vaccination is used within specific zones within a country, free movement of vaccinated animals throughout that country would also be prohibited (28).

Conclusions

Epidemics of most infectious diseases are subject to mandatory control for which regulatory legislation has been passed. Governmental involvement in disease control, and controversies surrounding it, have a long history, dating back in the UK to the mid-19th Century, when the relaxation of trade restrictions and ensuing epidemics of FMD, sheep pox and, notably, rinderpest (cattle plague), saw a change in favour of veterinary policing of the country (113). Now, as then, urgent decisions may need to be taken when facts are uncertain (35). Such decisions are based on received scientific wisdom, which may be either central to regulatory mechanisms (the 'technocratic model'; 107) or subordinate to political considerations (the 'Weberian decisionist mode'; 106). In either case, the value of the facts must be judged, and scientific experts must be accountable, not only to government ministers but also to other experts. To date, this has not occurred in the context of the 2001 epidemic.

Modelling should only be countenanced if veterinarians and scientists agree that the design of the model and the information used to generate its results are correct (and plausible, from the known biology of the disease). Otherwise, models: 'become exercises in mathematical sophistry' (96). Moreover, the rift between the models and the practical reality of implementation may be so huge as to make the models irrelevant (5). Significantly, Michael Osterholm, Director of the Center for Infectious Disease Research and Policy, University of Minnesota, has commented: 'In 30 years in public health, I've never seen any statistical modelling that had any impact on public health' (26). The most appropriate use of models is as inter-epidemic tools, to aid retrospective analysis of real epidemics to gain an understanding of their behaviour. Hypothetical scenarios can then be modelled to develop insights into the relative merits of different strategies in different situations. In this way, decision-makers can be provided with *a priori* supporting guidelines, used in conjunction with veterinary wisdom and experience – not as a substitute for them.

The use of models during epidemics should be restricted to monitoring the epidemic and aiding short-term fine adjustments to strategies. Comparing real behaviour to 'expected' (model-generated) behaviour could alert epidemiologists to unexpected circumstances in the field, which could then be targeted for action. Models may also be useful to carry out limited 'what-if' simulations, to assess risks associated with various developments of the epidemic, so that appropriate contingencies could be made in resource planning. During epidemics, models can usefully support the requisition of resources needed for well-tried control measures, by graphically demonstrating

the possible development of an epidemic. However, the utility of predictive models as tactical decision support tools is limited by the innate unpredictability of disease spread between farms. With particular reference to deciding on the use of ring vaccination, James and Rushton (53) draw the following conclusion:

'The progress of an outbreak of FMD is extremely difficult to predict in the early stages of the disease. The course of an outbreak can be critically affected by minor and inherently unpredictable events, such as a single livestock movement. For this reason, predictive disease models, which depend on statistical probabilities of transmission, have not met with much success in predicting the spread of FMD from herd to herd, and still less the impact of control measures. Given these constraints on predicting the impact of ring vaccination on the progress and extent of an outbreak, it is difficult to envisage an economic analysis that would guide decisions on the possible use of ring vaccination. This leads to the rather unsatisfactory conclusion that, in most cases, the impact of using or not using ring vaccination is essentially unpredictable. By the time that it becomes apparent that ring vaccination would have been justified, it is likely to be too late to use this method of control.'

The consequences of the 2001 European FMD epidemic will probably not be restricted to Europe. There would likely be considerable pressure to use vaccination in other FMD-free countries affected by an epidemic. It is becoming more obvious, even to those to whom it was not obvious at the time (as analysis of the 2001 epidemic continues), that the slaughter that took place was grossly excessive. However, traditional methods of control (rapid slaughter of animals on infected premises, and veterinary assessment of dangerous contacts before slaughter – but excluding automatic pre-emptive slaughter of animals on farms that are merely cartographically contiguous to infected premises) have been shown to be effective, without the need for the draconian slaughter that occurred in 2001. Moreover, the small percentage of farms in 2001 likely to have been infected 'across the fence' (a non-preventable route), in contrast to the majority of farms in which infection entered 'through the gate' (a route susceptible to blocking by movement controls and biosecurity), suggests that, in FMD epidemics caused by a virus with strain characteristics similar to that which caused the 2001 epidemic, a suitable aphorism for control is 'prevent – not pre-empt'.

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Utilisation et abus des modèles mathématiques : l'exemple de l'épidémie de fièvre aphteuse de 2001 au Royaume-Uni

R.P. Kitching, M.V. Thrusfield & N.M. Taylor

Résumé

La fièvre aphteuse représente une grave menace, non seulement pour les pays dont l'économie dépend des exportations agricoles, mais aussi pour les pays industrialisés qui préservent la santé de leur élevage national en éliminant les principales maladies infectieuses dans leurs populations animales. Les méthodes traditionnelles de lutte contre les maladies comme la fièvre aphteuse nécessitent la détection et l'abattage rapides des animaux infectés et de tous les animaux sensibles avec lesquels ils peuvent avoir été en contact, soit directement soit indirectement. Pendant l'épidémie de fièvre aphteuse de 2001 au Royaume-Uni, cette approche a été completée par une politique d'abattage sanitaire fondée sur des modèles prédictifs qui n'avaient pas été validés. Ainsi, l'épidémie et les mesures de lutte ont eu pour conséquences la mort d'environ 10 millions d'animaux, les protestations du public devant l'ampleur du massacre et la décision prise par les autorités d'adopter d'autres options, notamment la vaccination, pour lutter contre les futures épidémies. L'expérience du Royaume-Uni nous donne un avertissement salutaire sur l'abus que l'on peut faire des modèles si l'on pratique l'opportunisme scientifique.

Mots-clés

Abattage – Abattage sanitaire – Abattage sanitaire total – Épidémiologie – Fièvre aphteuse – Infectiosité – Modèle mathématique – Modélisation – Propagation du virus – Royaume-Uni – Transmission.

■

La epidemia de fiebre aftosa de 2001 en el Reino Unido como ejemplo de uso y abuso de modelos matemáticos

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Resumen

La fiebre aftosa constituye una grave amenaza, no sólo para países cuya economía depende básicamente de las exportaciones agropecuarias, sino también para los países industrializados que protegen la salud de su cabaña ganadera manteniéndola libre de las principales enfermedades infecciosas. Los métodos tradicionales de lucha contra dolencias como la fiebre aftosa exigen detectar y sacrificar con rapidez a los animales infectados y a cualquier otro animal sensible al patógeno que haya estado en contacto, directo o indirecto, con ellos. Durante la epidemia de fiebre aftosa que asoló el Reino Unido en 2001, este procedimiento se acompañó de medidas de sacrificio sanitario que respondían a modelos predictivos no validados. La epidemia y la aplicación de dichas medidas se saldaron con la muerte de unos 10 millones de animales, cosa que suscitó el horror ciudadano ante la magnitud de la hecatombe y condujo a la firme decisión política de utilizar en el futuro métodos alternativos, que comprendieran en especial la vacunación, para controlar toda epidemia. La experiencia británica constituye una saludable advertencia contra el uso incorrecto de modelos en beneficio del oportunismo científico.

Palabras clave

Elaboración de modelos – Epidemiología – Fiebre aftosa – Infecciosidad – Modelo matemático – Propagación de virus – Reino Unido – Sacrificio – Sacrificio sanitario – Sacrificio sanitario total – Transmisión.

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