

## Studies on the 1967–8 foot-and-mouth disease epidemic

### The relation of weather to the spread of disease

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#### SUMMARY

An analysis of the 1967–8 foot-and-mouth disease epidemic with reference to the initial spread, the origin of outbreaks more than 60 km. from the main epidemic area, the series of outbreaks near Worcester, a specific case history and the daily rate of spread of the epidemic, strongly suggests that the weather played a major part in the spread of disease. The two main factors involved in this type of spread are wind and precipitation. It is noted that after the epidemic had been checked, following anticyclonic weather, the association between the weather and the spread of disease was less apparent.

#### INTRODUCTION

Many possible means of spread of the virus of foot-and-mouth disease (FMD) are known or have been suggested. It is believed that the virus can be carried by any of the following agents: infected or incubating animals, bones or meat or milk from such animals, by fomites such as birds, insects, rats, hay, vehicles or people and by the wind.

During the 1967–8 epidemic in England and Wales, every outbreak was carefully investigated by the Government Veterinary Field Service (V.F.S.) to try to identify the most likely cause; however, the number of outbreaks for which a definite origin was eventually determined was very small—of the order of 5%. The source of infection for some 2200 outbreaks remained unaccounted for.

The possibility that foot-and-mouth disease virus may be disseminated by the wind was first put forward by Hardy & Milne (1938), who suggested that it was carried on particles and insects, and by McClean (1938), who suggested that it might be absorbed on aeroplankton yeasts. Experimental airborne infection over 10 m. has been demonstrated by Fogedby, Malmquist, Osteen & Johnson (1960). Hyslop (1965*b*) showed the presence of aerosols of FMD virus in air, coarse-filtered to remove dust, from loose boxes containing infected cattle; his work (1965*a*) also demonstrated that release of virus into the air occurred before the appearance of clinical symptoms, as well as during and after the appearance and healing of vesicles.

Some evidence has been advanced in favour of the possibility of airborne spread over distances up to 150 km. Fogedby *et al.* (1960) described how the disease appeared in Scandinavia after being present in northern Germany, and how it appeared to 'hop' from island to island in the Danish archipelago. Hurst (1968) has shown that many of the primary outbreaks which have occurred near the east and south coasts of England since 1937 can be attributed to wind-borne spread from sources on the Continent, although the first outbreak in the 1967-8 epidemic was not windborne. Most recently Smith & Hugh-Jones (1969) demonstrated that the wind has been one of the prime factors in the spread of FMD during previous epidemics in this country, and that most outbreaks due to wind spread were associated with rain. The present paper will demonstrate that during the 1967-8 epidemic the weather also played a very large part in the spread.

#### METHODS

##### *Disease data*

Some of the disease data were gathered during the period 5 Nov. 1967 to 14 Jan. 1968 by one of us (M.E.H.-J.), who interviewed farmers and veterinary officers in selected areas and maintained an index of map references of affected stock and premises. In July-August 1968 further information was abstracted from the V.F.S. files of all outbreaks; this included date of confirmation of FMD, the total number and species of animals reported diseased, number and species of animals in contact with affected stock, date and time of slaughter of the latter group, the number and species of stock affected each day until slaughter; whether stock were housed or grazing, type of housing and how long they had been in this accommodation before official confirmation of foot-and-mouth disease. Whilst it is agreed that this hearsay information is not as accurate as that obtained by individual interviews, the size of the epidemic made the latter impossible in every case. The information in the files was also used to estimate the most probable date of first clinical disease for each affected group, accepting that even in controlled circumstances it is an inspired guess. When an adequate herd history was available, the date was taken to be the earliest date on which any animal, later found to have lesions of foot-and-mouth disease, had been noted by the owner or his staff, his veterinary surgeon or visiting veterinary officer to be 'off colour', lame or suddenly producing less milk. Otherwise the date was estimated by the extent and condition of the recorded lesions and clinical record of the affected stock. It was assumed that, for cattle and pigs, symptoms occurred at the following intervals from onset of disease: temperature of 105°-106° F. (at onset), unruptured vesicles (4-6 hr.), recently ruptured vesicles (6-8 hr.), ruptured vesicles with ragged edges and strips of loose epithelium (12-18 hr.), separation of horn from coronary band and necrosis (24-36 hr.), granulation tissue (36 hr. or more). For sheep, however, the estimation of the age of clinical disease in a flock using this method is extremely unreliable. When a farm had two or more outbreaks, widely separated in time or space, they were regarded as separate outbreaks. The period during which an outbreak was active as a source of infection was taken to be from the date of initial disease to

the date of slaughter; however, this may not be correct, not only because of doubts about the former date, but also because it is believed that animals may produce substantial quantities of virus before clinical disease appears (Burrows, 1968).

### *Meteorological data*

Hourly weather records from the Meteorological Office at R.A.F. Shawbury, situated 8 miles north of Shrewsbury, were considered to be adequately representative of the general weather conditions over the epidemic area as a whole and were used in most of the analyses. Data from the Pershore meteorological stations were used in the analyses of the small epidemic near Worcester.

### *Detailed analysis*

Detailed studies were made of the spread during the initial and final stages of the epidemic, and in Worcestershire. In these studies an attempt was made to determine the most likely source of each individual outbreak, taking into account wind directions, rain, proximity to the supposed source and variations in the incubation period. Various indications (M. E. Hugh-Jones, unpublished work) led us to believe that during this epidemic the incubation period lay between 4 and 14 days, with a mode at about 8 days. The minimum interfarm disease interval seemed to be 3 days; because of the high viability of the virus strain in this epidemic, a maximum interval was difficult to estimate owing to the delay by susceptible animals in finding an infective dose of virus.

This type of analysis was not practicable for most of the epidemic because of the multiplicity of possible sources of each outbreak.

### *Statistical analysis*

This analysis was an attempt to relate the amount of spread at different stages of the epidemic to meteorological and other possible influences. It was necessary to calculate for each day an estimate of the 'rate of spread'; this was defined as:

$$\frac{\text{Apparent number of outbreaks resulting from spread on day } D}{\text{Source strength on day } D}$$

The denominator was calculated by the same method as was used by Smith & Hugh-Jones (1969), using outbreaks in cattle and pigs. Outbreaks in flocks of sheep (which formed less than 5% of the total number of outbreaks) were omitted because of the frequent doubts about their initial disease dates, and also because infected flocks, for reasons little understood, do not appear frequently to infect stock grazing adjoining fields. The numerator depends on the length of the incubation period, and we took it to be the mean number of outbreaks with initial disease on days  $D+7$ ,  $D+8$ ,  $D+9$ , again omitting sheep outbreaks. For the period from 15 Jan. onwards, because of the small number of outbreaks, 5-day mean values were calculated. For the first 11 days of the epidemic it was possible to estimate the rate of spread a little more accurately. The resulting estimates are included in Fig. 6.

## THE INITIAL SPREAD

The first known outbreak and that with the longest history of clinical disease was Bryn Farm at Nant Mawr, 5 miles to the south of Oswestry in Shropshire. Briefly, one sow was lame on 21 Oct. 1967, a second on 23 Oct., a third sow and eleven store pigs were ailing on 25 Oct. and by slaughter the next day a total of 28 pigs were found to be diseased. The farms next affected in the area were two neighbours within  $\frac{1}{2}$  km. and 2 km. with 12 and 14 affected cattle on 27 Oct. It is possible that there were other unrecognized affected stock in the area before or concurrent with the Bryn Farm outbreak, but this can be ignored as it does not affect the following argument.

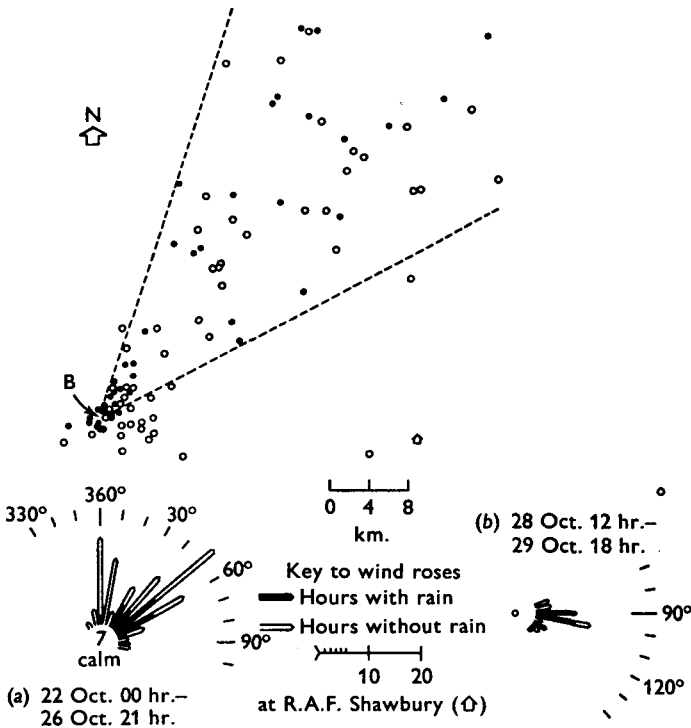


Fig. 1. Distribution of FMD outbreaks during the periods 21 Oct.–1 Nov. 1967 (dots), and 2–5 Nov. 1967 (open circles).

Figure 1 shows (dots) the positions of all the outbreaks in the country up to 1 Nov., with the exception of one at Carnforth, Lancashire. It can be seen that 31 outbreaks lay within a limited sector (between discontinuous lines), and 11 outbreaks were outside this sector but less than 2 km. from Bryn Farm, the vertex. Only two outbreaks were outside the sector and more than 2 km. from Bryn Farm; one of these was 3 km. to the WSW, the other was the Carnforth outbreak 150 km. away on bearing  $010^\circ$ .

Figure 1a shows that during the period 22 Oct. 00 hr. to 26 Oct. 21 hr. the wind blew predominantly towards bearings  $360^\circ$ – $070^\circ$ . This range of directions contains the sector in which the outbreaks occurred. Most of the outbreaks outside the sector were so near to the source that temporary or local variations of wind, or

winds on 21 Oct. which included calm spells, could have been associated with them. It rained frequently during the period 22–26 Oct. It is not possible to associate any of the outbreaks with any specific type of weather, although there is some suggestion that spread was confined to the directions in which the rainy winds blew. However this association may be partly due to other differences such as variations in the strength of the source; winds blowing towards the north did not occur after 13.00 hr. on 25 Oct., and since the number of known clinically affected animals at the source gradually increased, more spread could have been expected towards the end of the period than at the beginning.

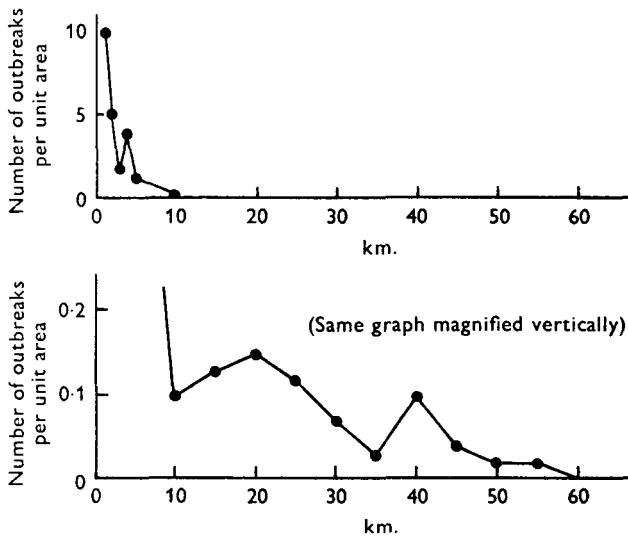


Fig. 2. Number of outbreaks, 21 Oct.–1 Nov., per unit area, plotted against distance from the first outbreak. Only farms which lay between the dotted lines of Fig. 1 are included.

Figure 2 shows the number of farms per unit area affected at different distances downwind in the sector delineated in Fig. 1. The distribution shows a decrease approximately with the square of the distance from the vertex, conforming to the concentration pattern which would result from windborne spread of material from a point source.

The mean bearing of the 19 outbreaks within the sector and more than 10 km. from Bryn Farm was 039°. The mean surface wind direction (21 Oct. 23 hr. to 26 Oct. 22 hr. based on hourly values) was 034°, and the mean 2000 ft. wind direction (22 Oct. 00 hr. to 26 Oct. 21 hr. based on 3-hourly values) was 068°. Thus the direction of spread was closer to the surface wind than to the 2000 ft. wind. Pasquill (1961) gives a formula for calculating the position of the axis of a plume of windborne material at distances of 10–100 km. downwind of a source:

Bearing of axis =

$$\frac{1}{2}(\text{mean surface wind bearing} + \text{mean 2000 ft. wind bearing}) - 10^\circ.$$

In the present example the formula yields the value 041°, close to the mean

bearing (039°) of the outbreaks. The height at which transport took place was somewhat above the surface, but not nearly as high as 2000 ft.

The outbreaks which first showed disease during the period 2–5 Nov. are also shown in Fig. 1 (open circles). Most of these were in the same sector as the earlier outbreaks, and while some of them may have had the same source, others may have been the result of spread from the earlier outbreaks; so individual analysis is of little value. However, of particular interest are the 12 which occurred more than 2 km. from Bryn Farm in a sector to the east of Nant Mawr; 10 were in a cluster within 8 km., the other two were respectively 27 km. and 57 km. away. No previous outbreaks had occurred in this region, and this suggests that these 12 must have become infected at a later date than the earlier outbreaks. If the minimum possible incubation period is assumed to be 4 days, it follows that infection would have occurred in the period 27–29 Oct. During this period there were within 2 km. of Bryn Farm three farms with diseased cattle and one with diseased sheep. From 12.00 hr. on 28 to 18.00 hr. on 29 Oct. the wind blew with rain mainly towards bearings 090°–100° (*b*, Fig. 1) with short spells on either side of this range. The 12 outbreaks were centred on these directions, and in particular the two outlying outbreaks both lay on a bearing of 099° from one of the farms affected on 29 Oct. and at distances comparable to those reached by the previous wave of infection.

It may therefore be concluded that most of the initial spread in the 1967–8 epidemic can be attributed to wind.

#### LONG DISTANCE SPREAD

There were thirteen outbreaks (approximately 0.5% of the total) which were at least 60 km. from any possible source and for which no fomite was identified as being responsible for the spread (see Table 1). There may have been some further

Table 1. *Outbreaks which were probably at least 60 km. from their source*

Area	Approximate distance from main infected area* (km.)	Date of initial disease	If wind-spread, probable time when spread occurred
Carnforth	150	29. x.	22. x.–25. x.
Darley Dale	70	6. xi.	22. x.–1. xi.
Blackburn	60	9. xi.	1. xi.
Leicester	70	18. xi.	9. xi.–14. xi.
N.W. Gloucestershire	90	21. xi.	15. xi.–16. xi.
Usk	110	23. xi.	15. xi.–16. xi.
North Hykeham	110	7. xii.	27. xi.–30. xi.
Castle Bytham	120	8. xii.	27. xi.–30. xi.
Ledbury	80	16. xii.	6. xii.–12. xii.
N. Herefordshire	60	22. xii.	16. xii.–17. xii.
Darley Dale	60	29. xii.	21. xii.–24. xii.
Coventry	60	9. i.	30. xii.–3. i.
Market Harborough	80	13. i.	3. i.–6. i.

\* Cheshire–North Shropshire.

outbreaks, on the fringe of the main infected area, which resulted from spread over distances of 60 km. or more, but these are difficult to identify. These outbreaks may have been caused by wind or some other mechanism, while some may have been primaries resulting from an untraced or hypothetical source. Mead's work (1968) suggests that spread by birds must be considered unlikely.

In each case there was a wind blowing with rain from the main infected area at a suitable time. The distribution of distances (for what it is worth, being based on such a small data sample) conforms well with a wind hypothesis.

A description of the circumstances of two of these outbreaks at North Hykeham and Castle Bytham is of interest. Diseases first occurred in Lincolnshire on 7 Dec. at North Hykeham, just south of Lincoln, in 37 heifers and a bull grazing a 10-acre field without cover or shelter. By slaughter at 11 hr. on 9 Dec. every animal in the herd was affected. It is possible that one animal may have been infected a few days previously and infected the others, but clinical disease appeared in all animals within only 35 hr., which is extraordinarily fast for spread within a grazing herd, the owner had been regularly inspecting them thrice each day, and a thorough examination of the slaughtered animals revealed none with healing lesions. It would therefore seem probable that virus was deposited on the field or feeding trough and infected all the animals nearly simultaneously. A thorough tracing by veterinary officers revealed no contacts with the main epidemic nor any other suspected sources of infection. The nearest outbreak at this time was 70 km. to the west and the main area 110 km.; however, winds blew from the west throughout the period 27–30 Nov., with intermittent rain each day.

On 8 Dec. one housed cow out of 26 was affected at Castle Bytham, 44 km. south of North Hykeham; there had been no previous outbreaks in the area.

It is not unlikely that these two outbreaks were extreme instances of airborne spread and intermittent rain deposition. We have noted that the furthest outbreaks in such circumstances often occur in a single line or front at right angles to the probable direction of spread.

#### *The Worcester sub-epidemic*

Between 14 Nov. and 6 Dec. 38 outbreaks occurred in a small area of Worcestershire just east of Worcester. Infection was introduced to three farms in this area in skim milk and there were no outbreaks in adjoining areas which can reasonably be supposed to have been the result of long distance spread from the main epidemic; the area can therefore be treated in isolation.

On 10 and 13 Nov. 500 gallons of skim milk were delivered to farm 1, 800 gallons to farm 2, and 500 gallons to farm 3. Other outbreaks elsewhere at the same time also traced to this skim milk indicate that the 10 Nov. delivery was most likely to have been infectious. At farm 1 the skim milk was fed to, among others, two sows and their 19 8-week-old offspring in a pen in an open-fronted shed. One sow was noticed to be 'poorly' on 14 Nov. At slaughter on 16 Nov. both sows and three young were clinically affected, and the next day 9 of the remaining 15 were affected. As a result of this outbreak, regular inspections were made of the other two farms which had received milk from the same consignment. At farm 2



4/50 store pigs (10–12 weeks old) in an open-fronted pen and fed on skim milk, were first noted to be ill by the owner at mid-day 17 Nov., with a total of 14 clinically affected at 17 hr. when slaughtered. The first pig noted to be ill at farm 3 was a newly weaned pig in a litter of eleven 10-week-old stores on 18 Nov. By slaughter the next day two more were affected. The store pigs in an adjoining fattening house fed the same skim milk were apparently healthy at slaughter.

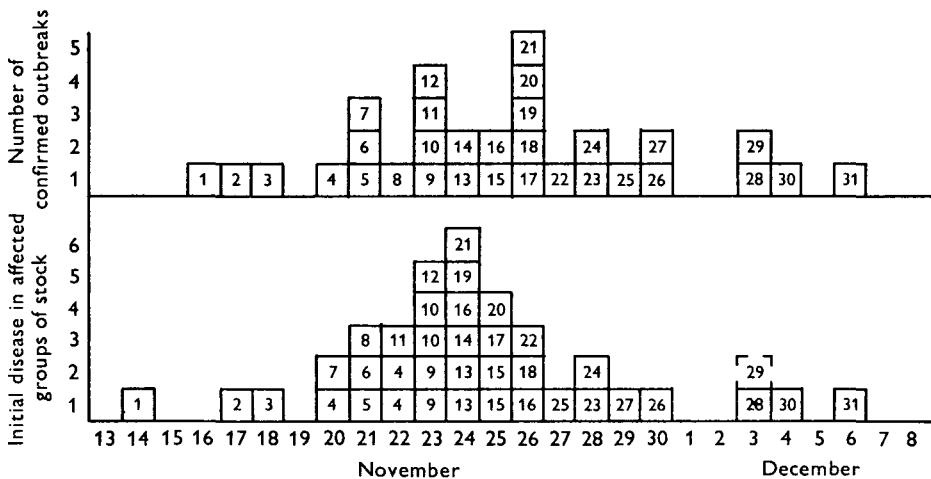


Fig. 3. Dates of the confirmation and the estimated first appearance of disease in herds and flocks in the Worcester sub-epidemic. The internal spread of disease on two farms, 19 and 21, has been disregarded.

Thorough tracing of bulk tankers and milk lorries by veterinary officers could not implicate milk in any of the subsequent spread in this area. Nor could any mechanical, human or animal vehicle be blamed, except on one farm (19), where infection was probably spread internally by a shepherd tending two flocks, and another (21), where outlying stock were brought into contact with the housed home stock. On some farms clinical disease appeared in different parts of the farm coincidentally or had developed by the time of slaughter. If these outbreaks are re-plotted by date of initial disease in different affected herds or flocks instead of confirmation date of disease on the farm, it can be seen that most of these outbreaks occurred within 13 days of the last pig killed on farm 1 on 17 Nov. (Fig. 3). The majority of outbreaks formed a cluster about farm 1 (Fig. 4). A careful tracing, counting and plotting of all the herds and flocks in the area showed that there were more than adequate numbers of susceptible stock on nearby and more distant farms for the epidemic to have spread further and especially from farms 2 and 3.

During the period 14–17 Nov., when farm 1 was infected, the winds varied in direction and there was rain at times while the wind was blowing towards the east (see Appendix for weather summary). The subsequent outbreaks about farm 1 were distributed in accordance with the weather during this period. In contrast, farm 2 was probably an active source on 17 Nov. and farm 3 on 18–19 Nov. for farms 18, 25 and possibly 29; this was a time of dry weather with light easterly winds, and



a relative absence of new outbreaks. During 20–24 Nov., when there was the maximum number of outbreaks, adequate numbers of animals available in spite of the slaughter of ‘dangerous contacts’, and dry anticyclonic weather with calm or light winds, little if any spread occurred; if and when spread did occur at this time (it is difficult to decide whether the individual outbreaks 23, 24 and 26, represent new infections or normal but long incubation periods), it was in the nature of infilling and not centrifugal dispersal. On the farms where FMD was diagnosed, virtually all groups of stock were affected by the time of slaughter and

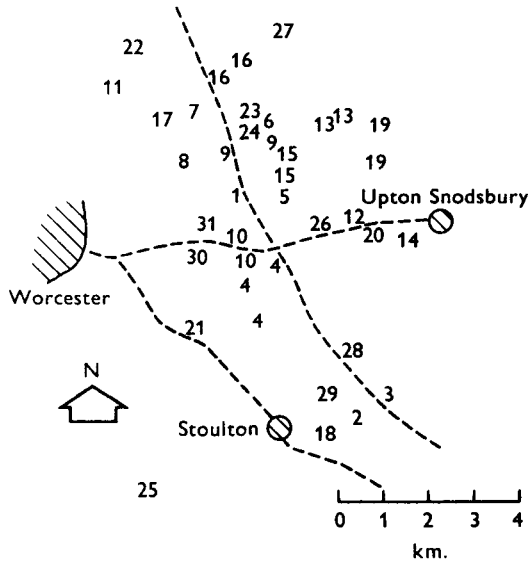


Fig. 4. Outbreaks in the Worcester area. The outbreak number for a farm is repeated for as many separate groups of stock as were clinically affected on the farm and indicates their positions.

it is only at the margins that unaffected groups were common, allowing for slaughter limiting the expression of disease. The commonest groups which apparently escaped disease were sheep flocks: it is notoriously difficult to diagnose FMD in sheep flocks. With the reappearance of wet weather, further outbreaks occurred. During 3 hr. of the early morning of 27 Nov., 1 mm. of rain fell with the wind blowing due north. Disease appeared in cattle at farms 30 and 31, 3 km. north of 20 affected cattle at farm 21, and at farm 28 adjacent to farms 18 and 29. So many sheep were affected at farm 29 that it was impossible to ascertain the initial date of disease in the flock. The earlier period of light rain on 25 Nov. fell with the wind blowing towards areas without stock and when these hypothetical ‘source’ farms had fewer diseased stock.

The study of this sub-epidemic was carried out during the epidemic and agrees with Henderson’s results (1969). However we are not as confident as he as to the source of infection of some of the later outbreaks.

Although foot-and-mouth disease is extremely infectious, this was a very localized epidemic which affected virtually all the stock in the original dispersal

and stopped in spite of the nearby availability of susceptible stock. This check occurred coincidentally with the anticyclone.

*A specific history*

This case history is presented to demonstrate a specific example of an aerial, and possibly aerosol, spread of infection between two herds.

Farmer A and Farmer B share what was once one set of farm buildings (Fig. 5). Both farmers kept dairy cattle in these buildings, but as far as could be ascertained had taken reasonable precautions to isolate themselves and their stock from each other. There was no common airspace, such as a loft or eave spaces between the two herds, and the brick wall dividing the two cowsheds was about ten inches

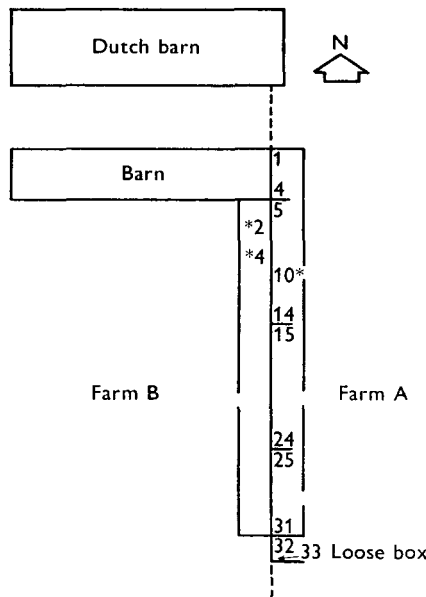


Fig. 5. Farms A and B showing standings (numbered). Asterisks indicate first animals affected on each farm.

thick with slots every 15 ft. and 8 ft. from the floor. These slots, 6 in.  $\times$  12 in.  $\times$  10 in., were filled with rubble and bricks except for the slot next to Farmer A's standing 10, which was filled with hay only.

On 16 Nov. at 07 hr. Farmer A noticed that a 6-year-old in-milk Friesian cow in standing 10 was dull, shivering, hunched up, salivating slightly with a temperature of 102.2° F. but eating normally. By 10 hr. her temperature had risen to 105.5° F. and he telephoned the nearest Foot-and-Mouth Disease Centre. A veterinary officer visited the farm at 11 hr. and found the cow to be 'off-colour', depressed, shivering, lifting her feet, with a temperature of 106° F. but without vesicles in the mouth or on the udder. Not convinced, but suspecting FMD, he served on the farm a formal restricting order and re-examined the cow at 17 hr., when the cow had a temperature of 105° F. but was otherwise no different and picking at her food. He saw her again the next morning at 10 hr., when she had

a temperature at 103° F., was much 'brighter', feeding, and without any vesicles in the mouth or on the feet and udder. Because of her habit of over-eating the officer diagnosed the condition as laminitis and removed the restriction. Later that morning, 17 Nov., Farmer A moved her into standing 33 in the loose box and put a cow from the loose box in her standing.

At about 22 hr. on 18 Nov. Farmer A noticed that five cows about standing 10 had become uneasy, and the next morning his veterinary surgeon diagnosed foot-and-mouth disease. At mid-day on 19 Nov. the cows in the following standings had unruptured or recently ruptured vesicles in the foot or mouth, cows 1, 4, 5, 9 to 14, 17, 19, 24, 27, 33, and cows in standings 20, 21 and 30 had temperatures over 104·5° F. The cow in standing 33 had healing lesions in her mouth and extensive separation at the coronary bands of her feet.

Farmer B's 27 milking cows were slaughtered as dangerous contacts on 20 Nov. at 15 hr. The cows in standing 2 and 4 opposite to Farmer A's standings 6 and 10, were found to have high temperatures and vesicles. The cow in standing 4 was opposite the hay-filled slot.

The hay-filled slot above standing 10 would have acted as a coarse filter and allowed through an aerosol, but probably not dust, drawn through as a result of any minor pressure differences between the two air spaces. It is highly possible that Farmer B's cows were infected as a result of aerosol transmission through this slot.

### *Epidemic pattern*

If we assume for the moment that all areas respond with equal numbers of outbreaks to equal amounts of virus deposited in them, we may use the daily 'rates of spread' as defined under Methods, to study the behaviour of the epidemic. The spread from Bryn Farm (most of which took place on 25 and 26 Oct.) resulted in 0·7 secondaries per unit source, much higher than at any later period of the epidemic. The spread then settled down to a general figure near 0·3 secondaries per unit source until mid-November. There was then a rapid drop in the rate of spread to 0·1 secondaries per unit source, as the epidemic passed its 'peak' (the maximum daily totals of confirmed cases were during 21–26 Nov., the spread which caused them being therefore about 15 Nov.). After a small rise in the rate of spread to about 0·17 in early December, it gradually decreased to below 0·1 by the end of the month. Throughout November and December there were large fluctuations with no clear general figure, but the details for these months must be treated with caution because of the small samples involved.

Comparison of the rate of spread with wind speed shows evidence of association (Fig. 6). In particular, the rate of spread was exceptionally high on 25–26 Oct., and on both these days the wind speed also was higher than on most days of the epidemic and was also constant in direction. Other marked similarities between the two graphs are the peaks on 5–6 Nov., the troughs about 9 Nov. and the peaks about 15 January. There were, however, many variations in the rate of spread which were not associated with similar variations in wind speed. It is of interest to note that from late October to mid-November maxima of wind speed occurred

about every sixth day. This interval is similar to that between infection and viraemia, if we assume a modal incubation period for clinical disease of 7–9 days.

The most important similarities between the graphs of rate of spread and precipitation duration were the coincidence of the steep drop in the rate of spread in mid-November with the start of the first dry spell of the epidemic, and also the

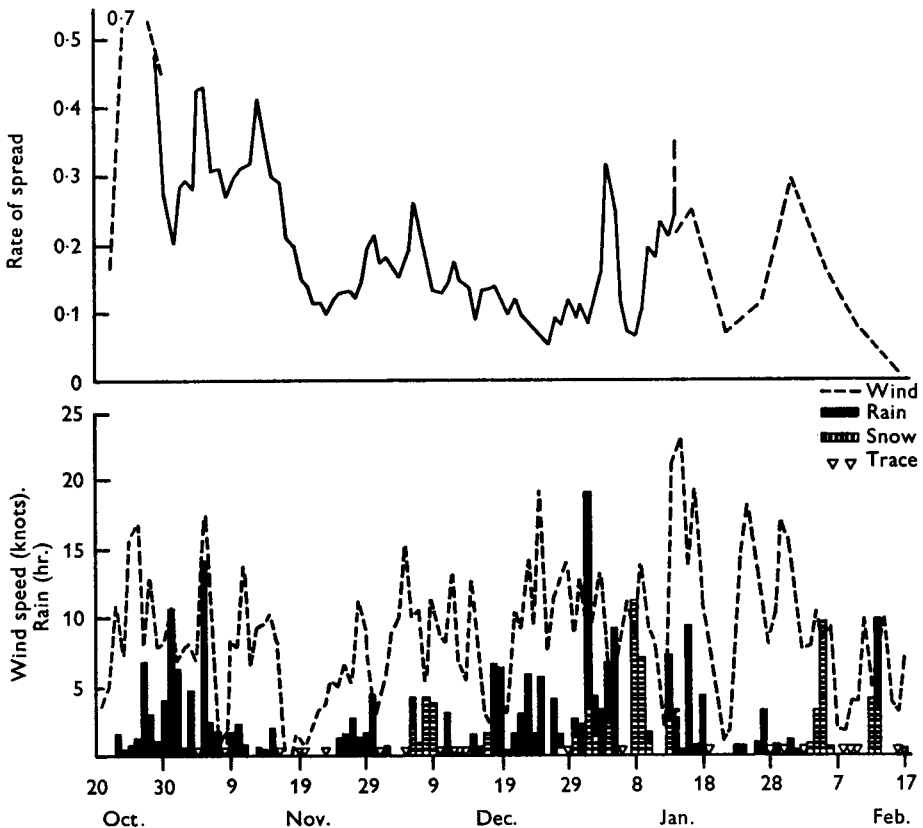


Fig. 6. The daily rates of spread of disease, mean wind speed (knots) and hours of precipitation during the period 21 Oct. 1967–17 Feb. 1968.

coincidence of the subsequent rise in the rate of spread with the return of rain towards the end of November. After this dry spell, the three highest peaks in the rate of spread were associated with three periods of snow—6–9 Dec., 8–10 Jan., and 3–5 Feb.; snow is known to be a highly efficient interceptor of airborne particles. However, while the snow in early December coincided with the peak, the other snow periods appear to have been somewhat later than the corresponding peaks. The association with hours of precipitation was not always close; for example, while the wet spell in early November was accompanied by a high rate of spread, a nearly equally wet spell (with similar average wind speeds) during Christmas week was accompanied by the lowest rate of spread of the epidemic. During the period 30 Oct.–20 Nov., a time of greatest spread, there was an average of 2.3 hr. rain per day; 21 Nov.–31 Dec. during least spread, 1.6 hr. rain per day;

1 Jan.–13 Feb. during increasing spread there were 2·6 hr. rain per day. Precipitation duration and amount were highly correlated, and so a comparison with amount would yield similar results.

There is a closer association between the daily rates of spread and the weather variables (01–24 hr.) during the period 30 Oct.–20 Nov. than at any other time of the epidemic. The correlation coefficients are:

Factors	1	2	3	4	
1 Rate of spread	1	—	—	—	
2 Rain duration (hr.)	0·20	1	—	—	
3 Mean wind speed (kts)	0·53	0·57	1	—	
4 Mean wind speed during rain (kts)	0·38	0·58	0·85	1	
5 24-hour variation of wind direction (°)	0·00	0·24	0·16	0·16	1

(5 % Significance level, 0·40)

This table demonstrates two points, that the weather variables are inter-related, and that the mean daily speed more than the wind blowing only during periods of rain is most closely associated quantitatively with the daily rate of spread. This is not incompatible with the previous results associating the spread of disease with rain-bearing winds because the weather factors are closely inter-related and because this quantitative analysis is non-spatial. The correlation coefficients for the rest of the epidemic were insignificant.

There was a lower rate of spread after the peak than before. We have seen this diminished response to the weather in all other foot-and-mouth disease epidemics in the U.K. that we have studied.

*Effect of housing stock*

It was usual practice to advise only farmers contiguous to outbreaks to house as many stock as they could and to withdraw the remainder into the middle of the farm; presumably it was thought that most spread occurred over only short distances, and this procedure would therefore protect the animals. However, there is no evidence to suggest that housed animals were any less at risk (Wright, 1969; Henderson, 1969; M. E. Hugh-Jones, unpublished results). During the 1967–8 epidemic all farmers were advised to house their stock; this advice together with the normal seasonal trend towards housing of stock meant that the proportion of animals grazing decreased rapidly until about 20 Nov., after which date it remained fairly constant (Fig. 7). Housing of stock will greatly decrease the air-flow over them.

*Effect of slaughter*

As all epidemics appear to spread up to the peak in spite of the slaughtering of diseased stock, and in a more limited manner afterwards, slaughter cannot be regarded as an absolutely effective control. If it does have an effect after the peak, which is unclear before, the nature of this control must change. Until 26 Nov. more herds of pigs and cattle, in which disease had been diagnosed, were being slaughtered the day after confirmation than on the confirmation day, taking the

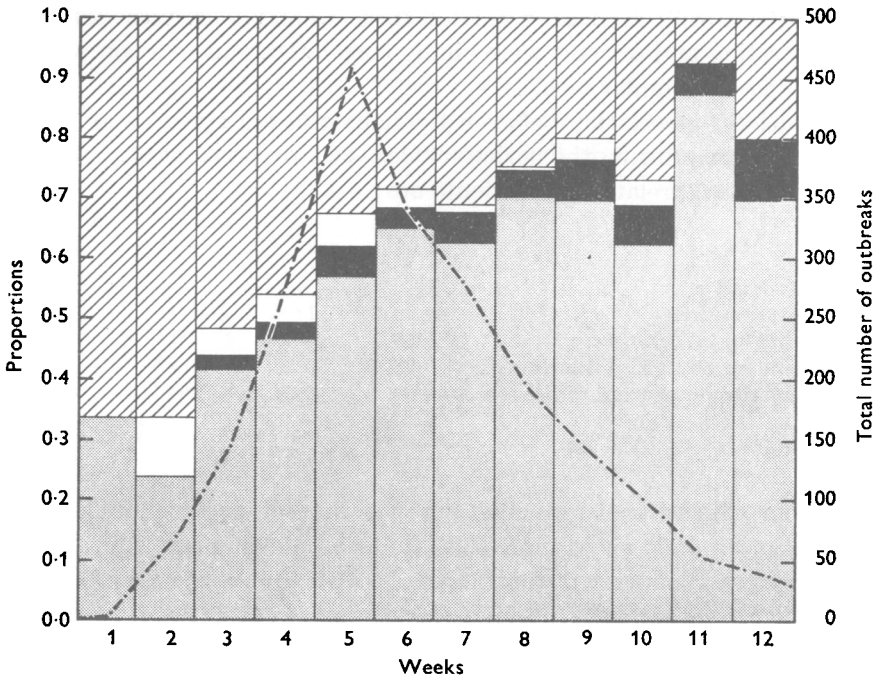


Fig. 7. Diagram showing proportions of infected herds of cattle housed (dotted), yarded (black) and grazing (diagonal lines) during each week of the epidemic; unrecorded or complex patterns of husbandry are blank. The graph of the number of outbreaks each week is shown for comparison.

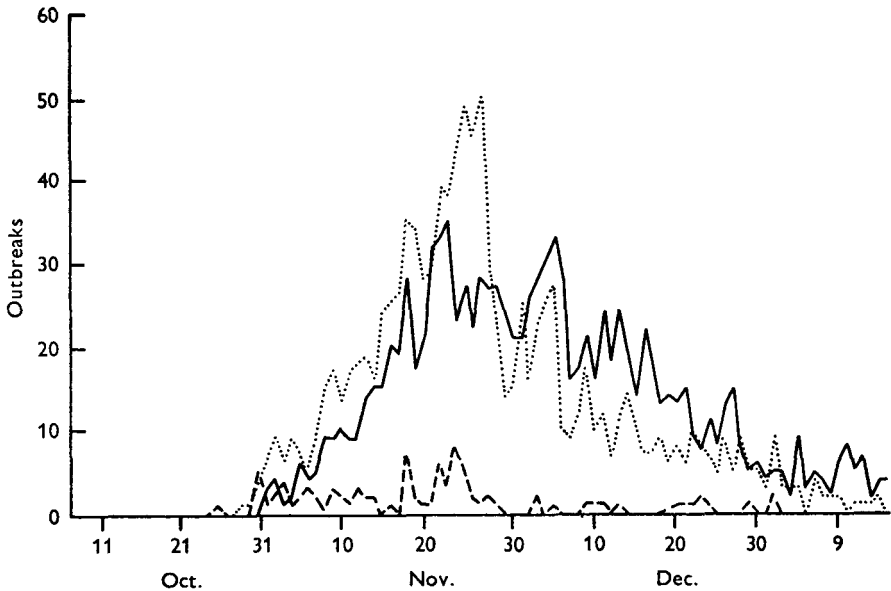


Fig. 8. Numbers of herds of cattle and pigs with confirmed FMD divided into three sets for each day: those which were slaughtered on the same day as confirmation, those slaughtered the next day, and those slaughtered more than one day later. —, Same day; ---, > one day; · · · ·, next day.



slaughter date to be the day on which animals in contact with the affected stock were killed. After this date the position was reversed (Fig. 8). During a 20-day period following 26 Nov. the average was 26.6 outbreaks in which the animals were killed on the same day as confirmation. The anticyclone in mid-November was associated with a reduction in the number of outbreaks to below 52 outbreaks per day; under these conditions more than 50% of outbreaks were slaughtered on the same day as confirmation. This is in contrast to the early part of the epidemic when only a minority were being slaughtered on the day diagnosis was made.

#### DISCUSSION

The evidence from this epidemic clearly suggests that windborne spread does occur, and it is now necessary to put forward a hypothesis to explain the mechanism. This rests on several assumptions, which appear reasonable but await experimental verification. The virus must first get into the air in sufficient quantities to survive extensive dilution; it must be transported in the air; it must be brought out of suspension; it must remain sufficiently viable and concentrated during these processes for a susceptible animal to find it and be able to demonstrate disease symptoms.

Emission from an affected animal can occur by breathing, sneezing or salivation from entire or ruptured vesicles, or by splash from falling infective urine, milk or faeces. The greatest emission usually occurs just before lesions appear but it can occur several days before (Burrows, 1968). Some animals, especially sheep, will show no obvious signs of disease, and therefore can act as a 'silent' source of infection. The relative rates of emission into air by different species is not known but appears to be in the following descending order: pigs, cattle, sheep (R. Sellers, personal communication). Sheep appear to be poor emitters because stock near to a sheep outbreak are not often diseased, but this may be only an apparent effect due to the fact that adjacent groups are usually also sheep, which although becoming infected may miss being diagnosed.

The virus when emitted is probably contained in droplet nuclei of various sizes. If spread is to occur farther than to adjacent animals the virus must remain airborne for sufficiently long to travel the required distance; this is possible if it is attached to or incorporated in particles of suitable size. Particles of diameter greater than about  $20 \mu$  will be deposited by gravity within a few hundred metres of the source, although if the wind is strong they will be carried farther before being deposited. Smaller particles, on the other hand, will show little tendency to fall, and although there will be some deposition due to turbulent transfer, a large number could be carried very great distances. An increased wind speed would be expected to increase the number of the smaller particles by assisting in the dehydration and breaking-up of the larger droplets; this, together with the greater distance of travel of the larger drops, would account for the observation that high wind speeds were associated with increased spread. It would follow that the housing of infected animals would reduce the dissemination of virus as a result of decreasing the speed of air movement around the animals.



During the course of the spread the infective material will diffuse horizontally and vertically; formulae for calculating the distribution of particles of various sizes downwind of a source have been published by Pasquill (1961). The amount of diffusion is proportional to the time of travel, rather than the distance, hence if the wind is strong and constant in direction there will be a greater concentration of particles at any given distance, especially along the axis of the plume. The viability of virus aerosols in different conditions of pH, air pollution and ultra-violet radiation is not known, although they would all be expected to have an effect. The ability to produce recrudescences of disease on restocked premises up to 4 months after previous infections suggests that the virus is effectively stable in field conditions and that loss of virus occurs more through erosion than through reduced viability.

Deposition in wet and dry weather of particles of a suitable diameter has been described by Chamberlain (1955). In dry weather the amount of deposition would be expected to be comparatively small and localized. This is supported by the evidence from the 1967–8 epidemic; the statistical analysis showed a marked reduction in the rate of spread during the anticyclonic spell in November, and during this same spell the spread in the Worcester epidemic was not great and was limited to a radius of 4 km. On another occasion (during the final phase in April) a group of outbreaks could be attributed to spread from a nearby source during an anticyclonic spell. In both the latter examples the outbreaks occurred in clusters rather than plumes; this would be expected from spread in light and variable winds characteristic of anticyclonic weather.

When precipitation falls through a plume it increases the number of 4–20  $\mu$  diameter particles deposited and neutralizes the dilution due to vertical dispersal. We would therefore expect a greater amount of deposition during rainy than during dry weather, especially beyond about 4 km., where dry deposition appears to be small; this is supported by the evidence we have presented. If the precipitation is intermittent, the deposition will be correspondingly greater where the precipitation does fall because particles will not have been washed out previously; on such occasions, especially with strong winds maintaining concentrations over longer distances, some isolated distant spread can be expected. It thus appears likely that the most distant spread must be associated with strong winds and intermittent rain; the limited evidence available to us supports this. Snow is more efficient than rain at washing out particles; the peaks in the rate of spread (Fig. 6) associated with the periods of snow would agree with this.

The precipitation will deposit particles on grazing and fodder crops (Kindyyakov, 1940), and will also produce a ground level aerosol capable of blowing into buildings. Dry deposition will have similar effects. The virus then has to come into contact with susceptible animals; if it does not, then no outbreaks will occur. The number of outbreaks depends very much on precisely where the virus happens to fall, and may not be closely related to the amount of virus released.

It is not clear whether the deposition on the ground or the ground-level aerosol is the more likely to produce infection, but it would have been expected that housed stock would have been at least partially protected from infection. However, the

evidence suggests that no significant protection was afforded by housing the animals.

*Termination of the epidemic*

Although much of the behaviour of the epidemic has been accounted for in terms of the weather, several features remain unexplained, the most important being that, after the peak, the epidemic appeared to be less controlled by the weather and continued to decrease in intensity even under what appeared to be suitable weather conditions for a high rate of spread. Previous epidemics also showed these characteristics (Smith & Hugh-Jones, 1969), and it is necessary to look for an explanation. Several suggestions may be made:

(a) By analogy with the spread of an infectious disease in a limited population, the decreasing availability of susceptible animals through slaughter, which is not dissimilar in its effect to the development of immunity, and the expansion of the infected area into less densely stocked regions must certainly play a part in reducing the rate of spread during the course of an epidemic. This depopulation occurred in discrete areas, which would still have received virus but, without susceptible stock, would be unable either to demonstrate its reception or to disseminate it further. The total depopulation in Cheshire was about 30%. The continued appearance of disease about later outbreaks would suggest that the development of immunity, without apparent disease, does not occur or is easily overcome. The importance of this hypothesis has still to be assessed.

(b) Housing. It has been suggested that a smaller quantity of virus is taken from a housed infected herd than from a similar number of grazing animals. As was seen in Fig. 7, over 60% of infected animals were grazing during the early part of the epidemic, but the proportion decreased steadily until the fifth week, from which time only around 30% were grazing. It is suggested that this played a part in lowering the rate of spread after mid-Nov. It also offers an explanation of why the rate of spread was closely correlated with wind speed only during the early part of the epidemic—variations in outdoor wind speeds have comparatively little influence on indoor airflow. However, it should be borne in mind that the bringing together of animals will favour increased spread within the group and thus produce a more intense source; also that cow-houses are not normally hermetically sealed, and concentrated clouds of virus may be released from an infected cow-house when doors and windows are opened.

(c) Slaughter. It is obvious that the sooner an infected herd is slaughtered, the less spread there will be from it. It is not simply the slaughter of animals with clinical symptoms that is important; if slaughter is delayed then many of the apparently healthy animals incubating disease as a result of spread within the group may reach their period of greatest emission. It has been seen (Fig. 8) that up to 26 Nov. more herds were being slaughtered the day after confirmation than on the same day, and a number were left for more than 1 day; after that date the average delay was much reduced. It is suggested that this delay in slaughtering would have contributed to the comparatively high rate of spread during the first half of the epidemic.

*Precautions*

The conclusion that wind is responsible for most of the spread of foot-and-mouth disease does not make us very optimistic about finding means of limiting the spread. Nevertheless, knowledge of the wind directions which prevailed while farms were infective could be helpful in showing the areas in which search for fresh outbreaks should be concentrated. If the weather was anticyclonic, attention should be concentrated on nearby farms; if it was wet, then the epidemiologist in the field should be prepared for the possibility of outbreaks several tens of kilometres downwind. The housing of all animals in and downwind of an infected area is probably a wise precaution, but while this may help to reduce further spread it is unlikely to prevent those animals themselves from contracting the disease. Any fodder exposed to the weather must be regarded as a potential risk, whether grazed directly or carried into housed animals. However the most effective means of reducing the spread would undoubtedly continue to be the early detection of symptoms and the immediate slaughter of affected and in-contact animals.

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## APPENDIX

The weather at Pershore meteorological station 6 miles to the south east of the infected area, during the period when most of the spread occurred, can be summarized as follows:

14 Nov. 06 hr.–15 Nov. 18 hr. Downwind bearing 40–80°, mean speed 10 knots. Dry except for slight rain at the beginning and end of the period. (The wind had been on these bearings for the two previous days with traces of rain.)

15 Nov. 18 hr.–15 Nov. 23 hr. Wind veered from 80° to 150° bearing, mean speed 14 knots, 2·1 mm. rain.

15 Nov. 23 hr.–16 Nov. 15 hr. Downwind bearing 150–180°, mean speed 112 knots, 0·2 mm. rain during first hour, otherwise dry.

16 Nov. 15 hr.–17 Nov. 21 hr. Wind calm or less than 6 knots from an easterly point.

Thereafter up to 25 Nov. 00 hr. the wind was always from the east, less than 10 knots, often calm with no rain except for a trace on 20 Nov. From 25 Nov. 00 hr.–29 Nov. 03 hr. the wind was from the west, never more than 10 knots and included twelve consecutive calm hours, with 0·1 mm. rain falling about mid-day 25 Nov. Between 03 hr. and 06 hr. on 27 Nov. 1 mm. of rain fell while the wind blew on a downwind bearing of 360°. Thereafter the wind continued to blow from the west.