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Forecasting virus atmospherical dispersion. Studies with foot-and-mouth disease

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Abstract

Virus release from vaccine plants or, more frequently, from infected farms may spread, causing epidemics. One of the mechanisms by which virus dispersion can take place is transport by air. In these cases, a plume is formed downwind, similar to that found in gas releases. Although a number of authors have cited this qualitatively, very few papers have treated it in a quantitative way. In this article, a simulation code developed for risk analysis has been used to forecast the dispersion of airborne virus at distances up to 10 km. The possible deposition of virus particles has been considered, as well as some restrictive conditions influencing virus survival in the atmosphere. The results have been tested by comparison with real data corresponding to two epidemics of foot-and-mouth disease. The agreement is relatively good, although uncertainty arises from poor knowledge of the real virus excretion rate and of the exact meteorological conditions existing in the two aforementioned epidemics.

Keywords: Atmospherical dispersion; Virus speed

1. Introduction

Considerable effort has been devoted to the development of methods and calculation codes for the estimation and prediction of the effects of toxic clouds of heavy, neutral or light gases. However, few papers have been published on the atmospherical dispersion of virus clouds, although these phenomena have potential consequences which make them extremely important both socially and economically.

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Virus clouds may be formed as a result of a release from a plant in which vaccine is being produced. For example, an accidental release of foot-and-mouth disease (FMD) virus probably originated from the Danish State Veterinary Institute for Virus Research in 1966, where FMD vaccine had been produced without an adequate air-filtration system [1]. Other cases in Europe are not so clear but seem probable according to the results of several molecular epidemiology studies [2, 3]. However, virus spread may be simply due to the emission originated by infected persons or animals. For obvious reasons, this is relatively frequent in farms, with severe consequences on neighbouring farms. Diverse investigations of a number of outbreaks occurring in different countries have shown that atmospheric transport of virus is one of the main mechanisms by which epidemics of some diseases propagate, not only to relatively near premises, but also over large distances over sea and even from one country to another.

Several authors have studied this topic, and a few of them have modelled it, testing their predictions with the scarce data available. Taking into account the features of this type of emission, it seems that atmospheric dispersion of a virus could receive a treatment similar – with the appropriate modifications – to that usually applied to the dispersion of certain toxic gases. This would allow the application of powerful calculation tools developed in the field of risk analysis and loss prevention in processing industries. In this paper, such an application is made; the validity of the results has been tested by studying two real cases of FMD (Hampshire and Worcestershire, 1967) on which detailed information is available.

2. Importance of airborne virus

The spread of viral diseases between farms takes place by diverse mechanisms: movement of carrier animals, both domestic and wild, persons, vehicles, infected food, water or manure, and finally dispersion of airborne virus. To prevent disease, control measures on the movement of animals, persons and articles may be established; however, it is very difficult sometimes to avoid air contamination and, in these cases, it is impossible to control the dispersion and transport of the virus as a function of meteorological conditions.

FMD is an acute viral disease that affects cattle, pigs and sheep, producing fever, vesicles and ulcers on the feet and mammary glands and around the mouth. It is one of the most important diseases of animals, and several authors have remarked on the importance of airborne virus in the propagation of epidemics [4–8]; in some outbreaks this mechanism has been claimed to be responsible for up to the 80% of the spread. Other virus infections would also be capable of spreading by wind; more details may be found in a review by Sattar and Ijaz [9].

In the event of an outbreak, several emergency measures must be taken: rapid detection and diagnosis, restriction of the movements of persons and animals, and avoiding the spread of the disease by other means. The prediction of atmospherical transport of the virus is therefore very important for the control of the disease. Rapid

estimation of the dispersion may help considerably, through intensive surveillance, to reduce the spread of epidemics and in consequence economic losses.

There are two important differences between airborne virus dispersion and gas dispersion. The first one is that, in the case of a virus, the dose may be significant (i.e. the consequences may be important) at very large distances if the atmospherical conditions are favourable. On the other hand, viruses may lose activity in the course of time, depending on the environmental conditions.

Several examples of airborne virus dispersion – besides those described in this paper – which can be cited are: an outbreak of FMD in Northumberland (UK) in 1966 with a probable spread at distances up to 8 km [5]; another outbreak of FMD in Côtes-du-Nord (France) transmitted over the sea to Jersey (Channel Islands), at a distance of 105 km, in 1974 [7]; transmission over 100 km from the Kalvehave area (Denmark) to Skaane (Sweden). Atmospheric transmission of Aujeszky's disease has been reported in Yorkshire (UK) at distances up to 7.5 km [10] and in Denmark at distances up to 55 km [11].

All these cases concern animal epidemics. Although releases of virus affecting persons seem also to have occurred, we have not found reliable information about this that can be included here.

Generally speaking, two different scenarios may be considered. Firstly, the airborne transport of virus over distances up to about 10 km is relatively common. Secondly, transport – sometimes over the sea – over much greater distances (up to 100 km) is less frequent. Although the prediction of virus dispersion is important in both cases, this paper is devoted to the first scenario.

3. Emission source

Virus is excreted for a period of four or five days, at a rate which depends on the species of animal (Table 1), the date of infection and the type of virus [12]. Emission can therefore be substituted by a certain flow rate of air, that corresponds to the respiratory rate of the infected animals, containing a given concentration of virus. The initial temperature of this air is at body temperature (approximately 38 °C), but in this work it has been supposed that it rapidly cools to room temperature.

Table 1
Emission rate of FMD virus (strain O_1) at the period of maximum excretion rate[a]

Source	Flow of infectious units (ID_{50}/min)	Respiratory rate (1/min)
Pig	4×10^3	25
Cattle	85	100
Sheep	66	10

[a] Taken from Ref. [12].

4. Dispersion of airborne virus

Virus is transported as an aerosol, with a certain granulometric distribution which is approximately as follows: 60% of particles with a diameter of approximately 6 μm, 25% in the range from 3 to 6 μm and 15% with a diameter less than 3 μm. In this size range, dispersion is indistinguishable from a gas, but the eventual deposition of these particles on the ground must be considered.

The deposition of particulate material from a gas cloud is a field of great uncertainty; it has been treated by Clancey [13]. The terminal velocity of a spherical particle (viscous flow) may be calculated from the force balance by applying Stokes' law:

$$u_t = \frac{d^2 g(\rho_s - \rho)}{18\mu}. \tag{1}$$

This expression is valid for Reynolds numbers $Re < 1$:

$$Re = \frac{d u_t \rho}{\mu}. \tag{2}$$

This equation really applies to spherical particles; for non-spherical particles, terminal velocity is reduced by a factor which depends on particle shape. For large particle diameters (10–1000 μm) and densities ranging between 1 and 5 g/cm^3, the following equation has been suggested:

$$\log_{10} u_t = 1.244 \log_{10} r - 0.536. \tag{3}$$

Generally, it is accepted that airborne virus is transported in particles with an average diameter of approximately 6 μm [6]. For this diameter, Eq. (1) gives a terminal velocity of 0.001 m/s, which corresponds to a Reynolds number of approximately 3.5×10^{-4}.

For particles with a diameter less than 20 μm or a terminal velocity less than 1 cm/s, the effects of turbulence are still more important than those due to gravity [13]. At these particle sizes, deposition to the ground is mostly not by gravitational settling. In the present case, taking into account the low value for terminal velocity, it can be accepted that due to the effect of atmospheric turbulence the deposition or transfer of virus to the ground is negligible. Therefore, the effect of deposition on the concentration in the plume can be neglected compared to the effect due to dispersion in the atmosphere.

Besides dry deposition, airborne particles may be separated from the atmosphere by wet deposition, i.e. by the action of falling raindrops. However, Chamberlain [14] has proved that only for submicron particles (which can be entrained by turbulence into the rain-forming layers) is wet deposition much greater than dry deposition. For 6 μm diameter particles wet deposition is negligible, especially for distances up to 10 km from the source. Although several authors [4] have concluded that there was an important relationship between rain and disease spread, it seems that this is essentially due to the fact that during precipitation very favourable conditions for airborne dispersion of virus exist: relative humidity is very high (and, therefore, virus activity

and potential infectivity is strong), the lower layers of the atmosphere are relatively stable and wind speed has adequate values for transporting virus particles.

Of course, all these considerations concern only the aforementioned 6 μm particles; virus particles surrounded by mucus or attached to dust may be deposited when the air is calm or may be washed out by rain.

As happens with a gas, the emission of virus will give rise to a plume in the direction of the wind, with a certain horizontal and vertical dispersion. The shape and concentration of the plume will be a function of wind velocity and of the meteorological conditions, especially atmospherical stability. Generally, the highest concentrations in the plume will be reached at evening and night, when the lower atmospherical layers are usually stable and vertical dispersion is considerably reduced; during the day, wind is usually stronger and greater turbulence exists.

Taking into account the low virus concentration in the air emitted from the source, it can be accepted that the emission is a neutral gas (with the same density as air). Therefore, it may be accepted that the plume follows a Gaussian distribution; in this case, and supposing that the emission source is at ground level, the concentration at a certain point will be given by the following expression:

$$C(x,y,z) = \frac{Q}{\pi\sigma_y\sigma_z u}\exp\left[-\frac{1}{2}\left(\frac{y^2}{\sigma_y^2}+\frac{z^2}{\sigma_z^2}\right)\right] \tag{4}$$

which, for $z = 0$ (concentration at ground level), takes the following form:

$$C(x,y,0) = \frac{Q}{\pi\sigma_y\sigma_z u}\exp\left[-\frac{1}{2}\left(\frac{y}{\sigma_y}\right)^2\right]. \tag{5}$$

Finally, if the receptor point is located on the plume centerline directly downwind, the expression reduces to

$$C(x,0,0) = \frac{Q}{\pi\sigma_y\sigma_z u}. \tag{6}$$

One important parameter is atmospherical humidity, which has a strong influence on virus survival once emitted. In order to keep virus active, the relative humidity must be higher than 60%; in these conditions, virus can be active for many hours, but at lower humidities it becomes rapidly inactivated. In dry climates, therefore, humidity constitutes an important limitation to the atmospherical propagation of epidemics. Furthermore, bright days imply the action of ultraviolet light on the virus, which may be killed.

5. Simulation

The calculations have been performed using the ALOHA 5.05 code [15]. ALOHA (Areal Locations of Hazardous Atmospheres) was developed by the National Oceanic and Atmospheric Administration (NOAA, USA) as part of a hazard assessment package called CAMEO. It can be applied to gases which are neutrally buoyant or

heavier than air, and is essentially intended for accidental spills rather than for low-level chronic releases. This code had been previously tested, its results being in close agreement with those obtained from other simulation programes [16].

ALOHA allows the selection of a given atmospherical stability class (A, B, C, D, E or F) and may include the existence of a thermal inversion. Values for wind speed and ground roughness can be introduced, as well as the degree of cloud cover, relative humidity and air temperature. Source strength may take into account the eventual source height respect to ground level (except for heavy gases). The model does not incorporate effects of particulates, and should be used with caution at very low wind speeds, with very stable atmospheric conditions, where there are wind shifts or terrain effects (which are not taken into account in the calculations), and with concentration fluctuations close to the source.

The existence of obstacles such as buildings, trees, etc., can create disturbances which considerably alter the shape of the plume, and can sometimes have a protective effect; in one of the cases studied (Worcestershire), one farm was protected from infection by a wood 270 m wide of trees 12–15 m high. To avoid the difficulties inherent in these factors, only cases corresponding to dispersion over open country have been considered here, with a roughness length $z_0 = 0.03$ m.

Only outdoor concentrations have been calculated. The possibility of significantly lower doses due to the sheltering of animals in buildings following the emergency alarm has not been taken into account for three reasons. Firstly, the ventilation rate of buildings – a requirement for ascertaining indoor concentrations – is not known in most cases. Secondly, in some cases (for example, Worcestershire 1967) it has been observed that the only result of bringing in cattle immediately was to delay the infection for 1 or 2 days. Finally, it must be realized that in the case of epidemics the periods over which the source is emitting are much longer than in the case of toxic gas releases; although there is a certain variation in wind direction during the day, if a predominant direction is maintained the plume may cover a given location for many hours and allow indoor concentrations to reach values much closer to the outdoor levels.

This type of simulation would be merely speculative if the results were not compared with some experimental data. Here, the validation of calculated values has been achieved through comparison with two cases on which detailed information is available. Both cases are epidemics of FMD. Several characteristics of this virus make it very suitable to be transmitted by air: (a) infected animals – especially pigs – excrete high doses of virus; (b) animals can be infected with very low doses, and (c) virus loses very little activity in a wide range of meteorological conditions; in the following calculations it has been supposed that the loss of activity was negligible.

5.1. Epidemic in Hampshire (UK), 1967

A complete analysis published by Sellers and Forman [17] has been taken as the main data source for this study. Between 6 January and 3 February 1967, the disease was confirmed on 29 farms; 170 cattle, 285 pigs and 4 sheep developed FMD; a slaughter policy was applied and 2774 cattle, 414 sheep, 4708 pigs and 6 goats were

Table 2
Distances from infected farms, dates of lessions and incubation periods[a]

Suggested origin	Infected farm	Distance (km)	Estimated earliest date of lessions	Period of 4 to 10 day incubation period
Abattoir	2	6	5 January	26 December–1 January
Abattoir	5	2.5	8 January	29 December–4 January
Abattoir	11	3	8 January	29 December–4 January
Abattoir	12	10	9 January	30 December–5 January
1	12	1.5	9 January	30 December–5 January

[a] Taken from Ref. [17].

Fig. 1. Map of the area involved (Hampshire, 1967) showing rivers, 60 m contours, towns and farms. Cross-hatching indicates built-up areas. A: abattoir; P: Portsmouth (taken from Ref. [17]).

slaughtered. The disease appeared initially in pigs fed on infected meat, and the virus was subsequently disseminated from the local abattoir. According to the analysis developed by Sellers and Forman, for this study four farms were selected (number 2, 5, 11 and 12, respectively; Table 2) in which airborne virus had been probably the only mechanism of infection (Fig. 1).

Taking into account the date of detection of the disease, the incubation period (4–10 days for cattle between farms) and the direction of the wind, the days on which virus transport could have taken place were selected (Table 3).

Table 3
Wind speed during the epidemic[a]

Date	Possible sources and farms downwind	Hours of wind	
		> 2.5 m/s	> 5 m/s
31 December	Abattoir → 2, 5, 11, 12	24	22
1 January	Abattoir → 2, 5, 11, 12	24	14
5 January	1 → 12	18	11
6 January	1 → 12	13	3

[a] Taken from Ref. [17].

By considering the information on meteorological conditions and on the source, the following values were used for the simulation:

(a) *Atmospherical stability class*: D
Wind speed: 5 m/s (this parameter has also been varied from 5 to 7 m/s)
Relative humidity: 70%
Cloud cover: 70%
Air temperature: 4 °C
Ground roughness: 3 cm (open country)
Release flow rate: 100 l/min (this parameter has also been varied from 10 to 1000 l/min)
Release temperature: 15 °C
Release concentration: 160 ID_{50}/l[a].

(b) The same values as for (a), but with the following changes:
Atmospherical stability class: E
Wind speed: 2.5 m/s
Cloud cover: 50%.

A typical plume has been plotted in Fig. 2, where the isopleths corresponding to two concentrations can be seen. As commented before, these data correspond to the outdoor concentration. The indoor concentration (i.e. the concentration inside the building where cattle receiving the dose were located) would probably be lower, being a function of building air exchanges per hour.

The distance at which a given concentration is reached is a function – maintaining all the remaining variables constant – of wind velocity and of the release flow rate (release flow rate is, of course, a function of the number of animals infected at the source). Fig. 3 shows this variation for two different wind velocities; the concentration selected was 7×10^{-3} ID_{50}/m^3, which would give rise to a dose of 1 ID_{50} (the minimum required to cause infection in a cow) for a period of 24h.

Fig. 4 shows the variation of concentration at a given point as a function of the release flow rate; the variation follows a linear trend, as could be inferred from the form of Eq. (3). Finally, Fig. 5 shows the variation of the concentration at a given

[a] ID_{50} is defined as the dose of virus which will infect 50% of test animals.

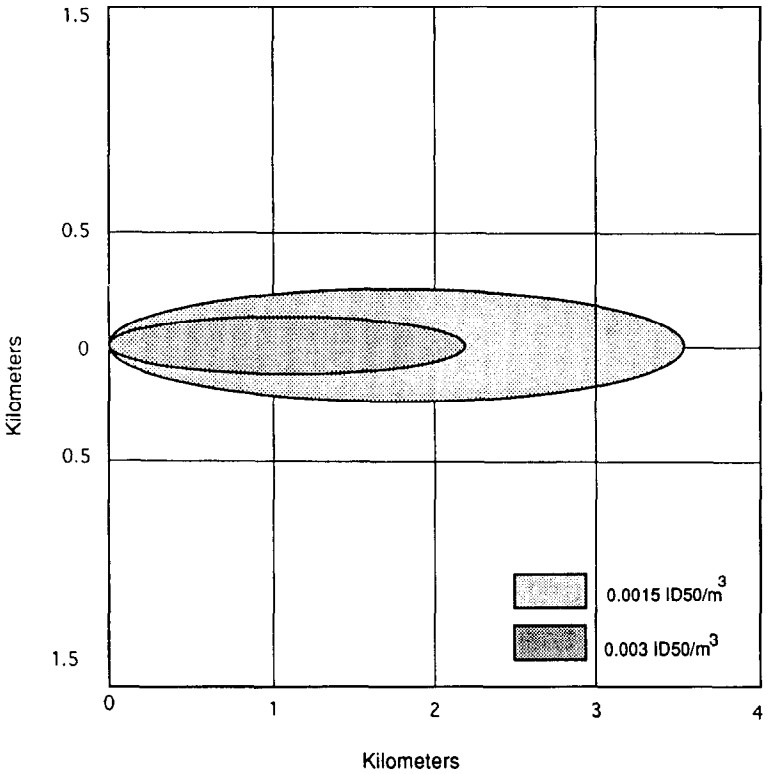

Fig. 2. Two different isopleths of the virus plume (release flow rate: 100 l/min; atmospheric stability class: D; wind velocity: 5 m/s).

distance from the source as a function of wind velocity; as the velocity increases the turbulence in the atmosphere also increases and, therefore, the dispersion is greater; this means that the plume is diluted and the concentration decreases. The variation has been plotted for three atmospherical stability classes (D, E and F) in the range of wind velocities in which they can exist.

The doses estimated for farms 2, 5, 11 and 12 have been summarized in Table 4. According to these results, farms 5 and 11 would have actually been infected by airborne virus. Farm 2 received a lower dose, but infection could also have been expected. However, farm 12 (for which emissions from the abbatoir and from farm 1 have been taken into account) could not have been infected by this mechanism, as the dose was clearly insufficient. These doses have been estimated assuming an excretion rate of 5.8×10^6 ID_{50} per day per infected pig and 1.22×10^5 ID_{50} per day per infected cow. However, some authors [6, 18] accept an excretion rate 17 times greater for pigs (i.e., 10^8 ID_{50} per day). If these values are assumed, the doses in Table 4 should also be multiplied by 17. These new values have been included in parentheses; according to these new values, all farms, including farm 12, were infected by airborne virus.

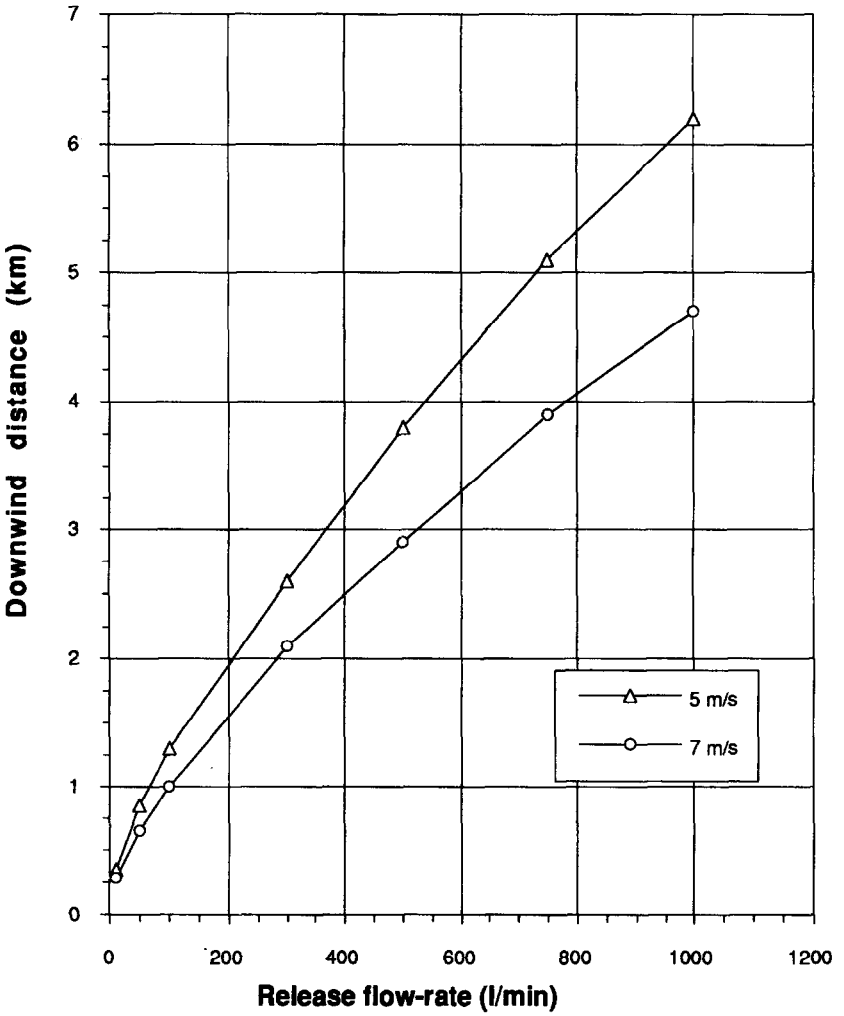

Fig. 3. Variation of the downwind distance at which a given concentration is reached ($c = 7 \times 10^{-3}$ ID_{50}/m^3) as a function of release flow rate, for two values of wind velocity (atmospheric stability class D).

5.2. *Epidemic in Worcestershire, 1967*

The epidemic appeared in three pig farms (on 15 November in the first one), originating probably from a tanker-load of skimmed milk. Information taken from the article by Henderson [19] has been used for this work.

The disease spread out radially, finally involving 39 farms. The following were selected for testing the simulation: farms number 6, 10, 14, 16 and 20 (15 November) and farms 4 and 11 (15 and 16 November) (see Fig. 6). The data on distances, wind velocity, etc., can be seen in Table 5.

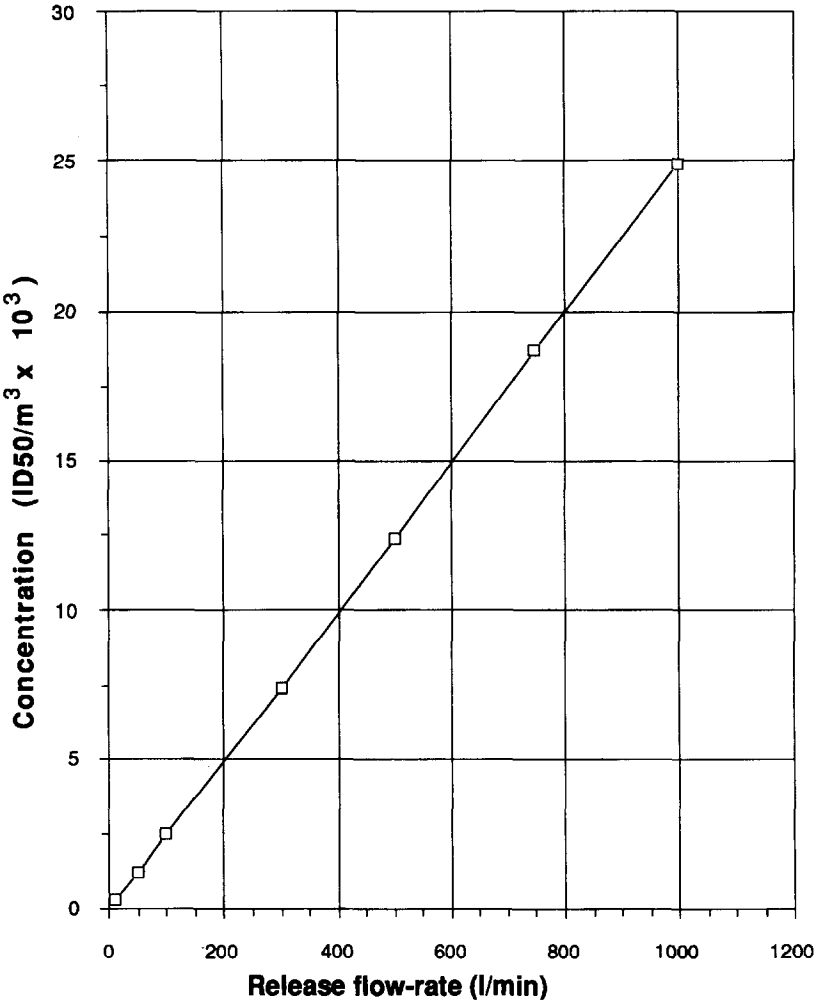

Fig. 4. Dependence of plume concentration at a given point (on the plume centerline downwind, at 2.5 km from the source) on the release flow rate (atmospheric stability class D; wind velocity: 5 m/s).

On 15 November a light breeze from the SW veered between lines AB and CD (Fig. 6) from 00.01 until 20.00 h (wind velocity: 0.5–1.5 m/s). From 21 to 24 h the wind blew from the NW between lines EF and GH (wind velocity: approximately 5 m/s). On 16 November, a light breeze came from the NW and N (between lines AB and CD) from 00.01 to 17.00 h, with a velocity of 0.5–3 m/s. After this, the aforementioned farms were infected. Taking into account all the measures taken by the farmers and the restrictions in the movements of persons, vehicles, etc., between farms, it was concluded that the disease spread to these farms from farm 1 by airborne virus.

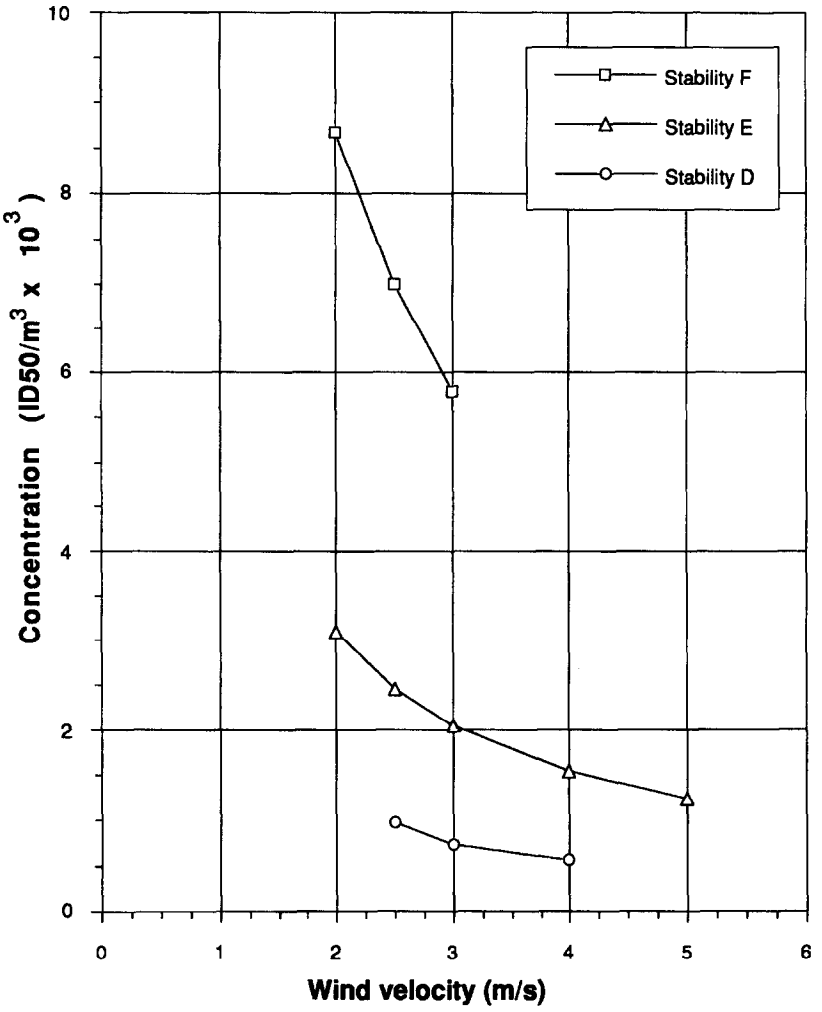

Fig. 5. Influence of wind velocity on virus concentration at a given distance downwind (2 km) for different atmospheric stability classes.

Table 4
Estimated doses, ID_{50}

Farm	Atmospherical stability class D	Atmospherical stability class E
2	0.33 (5.7)	0.8 (13.6)
5	1.2 (20.4)	2.5 (43)
11	0.92 (15.6)	2.0 (34)
12	0.15 (2.5)	0.38 (6.5)

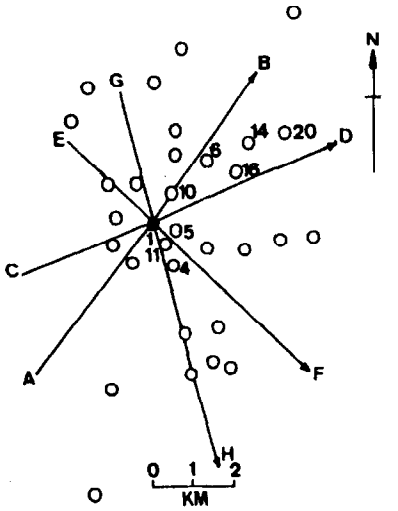

Fig. 6. Distribution of farms (Worcestershire, 1967) and wind direction (taken from Ref. [19]).

Table 5
Information concerning the selected farms[a]

Infected farm	Distance to origin (km)	Hours of wind	Wind velocity (m/s)
6	2	20	0.5–1.5
10	0.9	20	0.5–1.5
14	3.2	20	0.5–1.5
16	2.5	20	0.5–1.5
20	4	20	0.5–1.5
4	1.1	3	5
4	1.1	16	0.5–3
11	0.5	3	5
11	0.5	16	0.5–3

[a] Taken from Ref. [19]

The following conditions were taken for the simulation:

15 November

Farms 6, 10, 14, 16, 20:
Wind velocity: 1 m/s
Time: 20 h
Cloud cover: 90%
Release flow rate: 60 l/min.
Release concentration: 1.6×10^4 ID_{50}/min
Other parameters: as for the Hampshire case (a).

Table 6
Estimated doses for atmospherical stability class E

Farm	ID_{50}
4	2.1 (36)
6	1.3 (23)
10	4.9 (84)
11	8.8 (150)
14	0.5 (9)
16	0.9 (16)
20	0.3 (4.5)

Farms 5, 13, 15, 21:
Wind velocity: 5 m/s
Time: 1 h
Other parameters: as for farms 6, 10, 14, 16, 20.
Farms 4 *and* 11:
Time: 3 h
Other parameters: as for farms 5, 13, 15, 21.

16 November
Farms 4 *and* 11:
Wind velocity: 2 m/s
Time: 16 h
Release flow rate: 70 l/min
Release concentration: 2.4×10^4 ID_{50}/min
Other parameters: as for 15 November.

The predominating extremely low wind velocity rules out the existence of atmospherical stability class D and even E or F for most of the time. However, although it is a rather limiting condition, calculations have been done for class E (for a period of 11 h during the night). The results obtained can be seen in Table 6; according to them, airborne virus would have been responsible for infection in farms 4, 6, 10 and 11, and possibly also 16. The doses for the rest are too low. The doses obtained for farms 5, 13, 21 and 15 are extremely low (approximately 0.01 ID_{50}) and would not justify any infection; these farms were subjected to the action of airborne virus for a period of only 1 h, according to the information available, and this makes infection difficult. Again, all these values could be multiplied by a factor of 17 if the criterion accepted by some authors concerning the virus excretion rate is applied here (values in parantheses in Table 6).

6. Conclusions

The results obtained from application of the simulation code show relatively close agreement with the data taken from the literature concerning two epidemic cases of FMD. The main problem from the point of view of testing lies in the uncertainty of

some crucial data required for the calculations and in the incomplete information on the conditions corresponding to the reference cases used for the validation. The final doses forecasted by the simulation code depend heavily on the virus excretion rate, which can vary significantly; for example, for the case of FMD the excretion rate suggested by different authors ranges between $10^{4.8}$ ID_{50} and $10^{8.6}$ ID_{50} per day per infected pig. This may significantly modify the results for the prediction of eventual infection in a given situation. Furthermore, comparison of the predictions of the behaviour of disease spread in two real cases revealed certain difficulties due to lack of accurate and complete information on the local meteorological conditions during the outbreak. Finally, further uncertainty in the data from real cases arises from the fact that no absolute certainty can exist, for a given location, that only airborne virus had been responsible for the infection.

Nevertheless, this work has proved that the models usually applied to the prediction of the evolution of toxic clouds can also be applied to the dispersion of virus plumes. This is important for real-time analysis, during an outbreak (especially during the initial stage and the incubation period), of the affected area. In such cases, the aforementioned lack of information would not be a difficulty, as exact meteorological information could be gathered. This would be a powerful aid for the establishment of emergency measures.

Nomenclature

C	virus concentration
d	particle diameter, m
g	accelceration due to gravity, m s^{-2}
Q	mass release rate, kg s^{-1}
r	radius of the particle, μm
Re	Reynolds number
u	wind velocity at a height of 10 m, m s^{-1}
u_t	terminal velocity of a particle, m s^{-1}
x	distance downwind from the source, m
y	distance crosswind from the source, m
z	height over ground level, m
μ	air viscosity, kg m^{-1} s^{-1}
ρ	air density, kg m^{-3}
ρ_s	particle density, kg m^{-3}
σ_x	dispersion coefficient in the downwind direction, m
σ_y	dispersion coefficient in the crosswind direction, m
σ_z	dispersion coefficient in the vertical direction, m

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