14.8.6.

SPREAD OF FOOT-AND-MOUTH DISEASE

A Review prepared for the Australian Meat Research Corporation

by

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EXECUTIVE SUMMARY

1. The route of infection is important in determining the way in which FMD virus might be spread between animals. The amount of virus needed to produce infection by the oral route is many times that required for infection by inhalation. The amount of animal tissue or fluids which would need to be consumed to initiate infection are shown in Table 18 (page 74). For pigs, consumption of about 1 gram of lymph node, bone marrow or skin could be enough to produce infection in the worst-case scenario. For both cattle and pigs, the ingestion of 1 ml of saliva could cause FMD.

2. Wind-borne aerosol spread of FMD virus is important in Europe and Great Britain, primarily due to the mild climatic conditions. Most recent studies on the potential spread of FMD virus have been based on the 1967/68 outbreak which occurred in the United Kingdom. It is believed by some authors that the strain of FMDV involved in this outbreak was unusual and that it had an increased ability to survive in aerosols.

3. A crude analysis of climatic data suggests that optimum climatic conditions for the survival and dispersal of aerosols of FMD virus may only occur in a limited number of areas, and only at limited times, in Australia. It is likely that other methods of spread may be more important in the epidemiology of FMD in Australia.

4. It is apparent that in many countries the major method of spread of FMD is through livestock movements. Where stocking densities are high and the associated marketing systems involve large-scale animal movements there is increased risks for the spread of FMD. It is suggested that this may also occur in Australia, especially where climatic conditions are not conducive to aerosol spread of FMD virus. A study of the livestock movement patterns, particularly of sheep, in Australia may aid in the control of FMD and other diseases.

5. The species of animal is important in the spread of FMD virus by aerosols as pigs produce about 30 times more virus than either sheep or cattle from their respiratory tract. Pigs are rarely found to be carriers of FMD virus after recovery from the disease. Sheep appear to give a variable clinical response to infection with FMDV with a significant proportion of animals failing to develop lesions after infection.

6. The role of sheep in the epidemiology of FMD may be important in Australia. Conflicting evidence is available on the importance of this species in the spread of FMD. Sheep may be infected with FMDV, but show no clinical signs of FMD. This may allow the undetected infection of large numbers of animals within a flock, providing a large reservoir of virus which may be in aerosol form and sufficient to infect nearby susceptible flocks and herds. The movement of infected sheep, showing no clinical signs of FMD, during an FMD outbreak would also be a means of spread of FMD and highlights the need for strict movement controls to inhibit the spread of FMD.

7. FMD virus may be excreted by animals before they exhibit clinical signs, by animals which become infected but fail to develop clinical signs (e.g. sheep) and by animals after they have recovered from clinical FMD. Transmission of FMDV from animals in the first 2 categories to susceptible animals, resulting in FMD has been demonstrated. Natural transmission of infection from recovered carriers to susceptible animals has been difficult to demonstrate, especially for extended periods after recovery from clinical FMD. Pathogenicity tests of carrier virus strains of FMD virus present in oesophago-pharyngeal fluid samples showed that carrier virus was attenuated for the species of the carrier animals but retained its pathogenicity for other species.

8. The occurrence of recovered carrier animals, and the period of time during which FMDV continues to be shed from the oesophago-pharyngeal area is species-related. Cattle tend to shed FMDV for longer periods than sheep. Pigs rarely become carriers of FMDV.

9. Epidemiological evidence suggests that there may be little likelihood of carrier virus spreading in a vaccinated population under natural conditions. However, when animals are vaccinated in the face of an outbreak there is a chance that carrier animals will occur. This depends upon the period which elapses between vaccination and infection with animals vaccinated as little as 3 days prior to exposure to infection being protected against clinical FMD but not against infection resulting in sub-clinical infections. Vaccinated animals may become carriers and have the potential to be infectious for up to 30 days after vaccination. There is no simple and reliable method of detecting such carrier animals, except culture of oesophageal fluids to detect FMD virus, at the present time.

10. The nucleotide sequence of strains of FMD virus may evolve over a period of time to become specific to a geographical area. This is probably due to a complex interaction of climatic conditions, vaccination effect on immune status of animals, animal movement and species present in an area. It is known that the strain of FMD virus affects its production and survival in aerosols. Whether this is reflected in the nucleotide sequence is not known. It could be postulated that an FMD virus strain which has become 'adapted' to a particular geographic area may produce a different epidemiological pattern when transferred to another area. The implications of these findings are that FMD virus strains may behave differently, epidemiologically, in Australia than in their country of origin. The presence of a animal population which has had little, or no, contact with FMD viruses may also influence the epidemiology of FMD in Australia.

11. Previous assessment of the possibility of transmission between domestic animals and native fauna, in Australia, was primarily based on the excretion of virus in lesions. More recent research suggests that the respiratory route is the primary portal of entry of FMD virus. While there is, in general, little close contact between native Australian fauna and domestic stock there could arise in certain locations sufficient concentrations of native fauna and domestic stock to allow aerosol transmission of FMD. This aspect probably needs to be further explored.

12. Concurrent infection with bovine enteroviruses has been reported to inhibit the multiplication of FMD virus in cattle, causing a latent period before FMD virus causes infection and clinical signs.

RECOMMENDATIONS

1. A small preliminary investigation of the possibility of windborne spread of FMD in Australia was carried out by the Bureau of Meteorology in 1982. Relative humidity and winds were tabulated for a small number of coastal sites. This aspect requires further study in Australia with a retrospective study of climatic data to determine, for representative areas of Australia, the probability of sustained wind-borne spread of FMDV during each month of the year. The probability of wind-borne spread of FMDV will influence the planning and utilisation of resources for any FMD outbreak in Australia.

2. Buik (1979) has analysed cattle movements in Australia and noted that they can be used to identify key points in disease transmission such as major source regions of non-slaughter cattle, major recipient regions and the principal sale/purchase points through which the cattle move. Calculation of a contact index is suggested which could provide a weighting, in terms of probability of being infected, for all properties within a region. Sanson *et al.* (1993) examined animal movements in a small area of New Zealand to determine the size of control areas needed to contain the spread of FMD. Analyses similar to these studies need to be carried out for representative areas in Australia to determine whether zoning can be implemented and to investigate the extent of movement controls which would be necessary to control the spread of FMD. There is particular need to document livestock movement patterns in Australia, and sources of information on such patterns, in advance of any outbreak so that basic information is available to rapidly implement analyses similar to those of Sanson *et al.* (1993). The movement patterns of sheep may be particularly important because of the possibility that they may be infected without showing clinical signs and their movement may result in the spread of FMDV to other farms.

3. The movement of animal products within Australia is extensive and may represent a potential source of FMDV during an outbreak. There is a need to assess the risk that FMDV may be spread by animal product movement within Australia. This would need to take into account the survival of FMDV in dairy products and meat and the likelihood of it reaching susceptible livestock. In addition, the handling of animal products originating from an outbreak area needs to be examined. For example, the studies examined in this review suggest that normal pasteurisation of milk may not be sufficient to eliminate FMDV from dairy products, and special processing may be needed to make these products safe. In addition, the economic implications of restricting the movement of animal products needs examination.

4. The role of sheep in the spread of FMD needs further study. Many authors have observed that sheep often do not develop clinical signs after infection with FMDV but shed virus which may infect other susceptible animals. It has been suggested that FMD may spread slower, or not at all, in sheep raising areas. However, it is difficult to assess the significance of some of the observations. Further study of the role of sheep in the epidemiology of FMD is required, preferably in countries where there are major sheep enterprises.

5. The role of vaccination in combating an outbreak of FMD in Australia needs to examined in view of the potential for the development of carrier animals and the present difficulties of differentiating infected animals from vaccinates. Some analyses of the use of vaccination when FMD has been introduced into a fully susceptible population have demonstrated that, once the disease has reached a certain incidence, vaccination is the most cost-effective option. Most of these models have not included in their analysis the costs of loss of export markets. There would be merit in using or adapting existing models to examine when and where vaccination might be used to aid in the eradication of FMD in Australia.

6. Early studies by Australian workers indicated that there was a wide range of susceptibility to infection with FMDV between Australian fauna. The assessment of the possibility of transmission between domestic animals and native fauna was primarily based on the excretion of virus in lesions, although in one experiment native fauna and domestic animals were run in close contact. It was believed at the time of the work that lesion fluids were the most likely source of virus for FMDV transmission. Later work suggests that the respiratory route is the primary portal of entry of FMDV. While there is, in general, little close contact between native Australian fauna and domestic stock there could arise in certain locations sufficient concentrations of native fauna and domestic stock to allow aerosol transmission of FMD. This is an aspect that probably needs to be further explored.

7. A further assessment of the possible role of feral pigs in the maintenance and spread of FMD in Australia is warranted because of the amount of resources that would be needed to control this disease in feral pig populations. Anecdotal evidence suggests that the role of feral pigs in the epidemiology of FMD is minimal in Europe and South America. While the ecology of feral pigs in Europe is possibly different to that in Australia it would be useful to identify countries where their ecology is similar to Australia. Their association with FMD in those countries could be further investigated and an assessment of their importance in the epidemiology of FMD made.

8. New Zealand authorities have conducted a risk analysis of the importation of various diseases into their country by travellers and in postal items which is based on traffic flows from various countries, the prevalence of the disease in the country, the risks of spread in New Zealand and the economic costs to the country. Australia should adapt or modify such an analysis to quantify the risks of introducing FMD on a country by country basis by travellers and other means. Such an analysis would be useful in targeting animal quarantine inspections of traveller's luggage and would also provide some indication of the strains of FMDV likely to be imported into this country.

8. It has been observed that concurrent infection with bovine enteroviruses may mask the clinical signs of FMD and the multiplication of FMDV in cattle. While bovine enteroviruses are present in Australia, little is known of the prevalence of such infections. Further studies on bovine enteroviruses might be of use in determining whether masked infection could represent a problem in the diagnosis of FMD.

CONTENTS

ACRONYMS

AUSVETPLAN. . .	Australian Veterinary Emergency Plan
BTEC	Brucellosis and Tuberculosis Eradication Campaign
FMD	Foot and Mouth Disease
FMDV.	Foot and Mouth Disease Virus
ID_{50}	Infectious Dose (50%) - the dose of an infectious agent which when given to a group of animals produces infection in 50% of the group
MID_{50}	Mouse Infectious Dose (50%) - the dose of an infectious agent which produces infection in 50% of mice given the agent
pfu	Plaque forming unit - a virus particle which has the ability to form a plaque in a tissue culture
$TCID_{50}$	Tissue Culture Infectious Dose (50%) - the dose of an infectious agent which infects at least 50% of the cells in a tissue culture

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SPREAD OF FOOT-AND-MOUTH DISEASE

INTRODUCTION

Australia has been free of Foot-and-Mouth disease (FMD) for more than 100 years but the economic effects on the country following an outbreak would be devastating. Planning for the eradication of any potential FMD outbreak has a high profile with the initiation of AUSVETPLAN. To adequately plan for the eradication of any disease, information is needed on the methods and likelihood of its spread. Much of the published information available about primary outbreaks of Foot-and-Mouth disease, i.e., those occurring in a fully susceptible population, originates from Great Britain where climatic and farming conditions are considerably different to those in Australia. Other information comes from areas where Foot-and-Mouth disease is endemic or where vaccination is used to control the disease. These situations are different to that where infection is introduced into a completely susceptible population. This review is an attempt to assess the possible methods of spread of Foot-and-Mouth disease in Australia and to indicate where further information is needed to facilitate control of this disease in the event of an outbreak in Australia.

SOURCES/PRODUCTION OF VIRUS

Much of the data available on excretion of Foot-and-Mouth disease virus (FMDV) has been obtained from animals which have been artificially infected by intravenous, intradermal, intradermolingual, intramammary inoculations or intranasal instillation. In many instances this produces virus titres in secretions somewhat higher than those observed in natural outbreaks. For example, Hyde *et al.* (1975) infected milking cows by intramammary and intravenous inoculation of FMDV and produced virus titres of approximately $10^{7.0}$ $\log_{10}$ pfu/ml in milk, but Hedger and Dawson (1970) reported virus titres in milk from FMD-infected cows in a disease outbreak which were about 100 fold less than these values.

An attempt has been made in this review to gather information on virus excretion from natural cases or from animals infected by contact with infected animals, rather than the data derived from animals infected by parenteral inoculation.

(a). Virus excretion

The major sources of FMDV in infected animals are excretions such as (i). oesophago-pharyngeal fluids which produce aerosols during normal respiration, coughing or sneezing; (ii). saliva; (iii). milk; (iv). faeces; (v). urine; (vi). semen; and (vii) vaginal discharge. Other sources which, while not excretions, contain virus and may be the means of virus spread include muscle, bone marrow and lymph nodes; ova and blood.

(i). Oesophageal- Pharyngeal Fluid or Respiratory Tract Aerosols

Sellers *et al.*, (1977) found FMDV in the air of looseboxes in which animals previously exposed to infection with FMD were housed. FMDV was first observed at 30 minutes to 22 hours after exposure and then subsequently between 2 and 7 days after exposure. The first period probably represents virus shed mechanically from the respiratory tract and from the coats of animals exposed. However, the virus detected at 22 hours might represent a limited virus multiplication. The second period of virus excretion results from multiplication of FMDV in cells of the respiratory tract. The authors noted that, after exposure to infected animals, unvaccinated sheep may excrete airborne virus but fail to develop FMD lesions, or develop only mild lesions. Such animals could spread infection and it has been recommended that movement of animals should be restricted for 21 days after any possible contact with FMD infection.

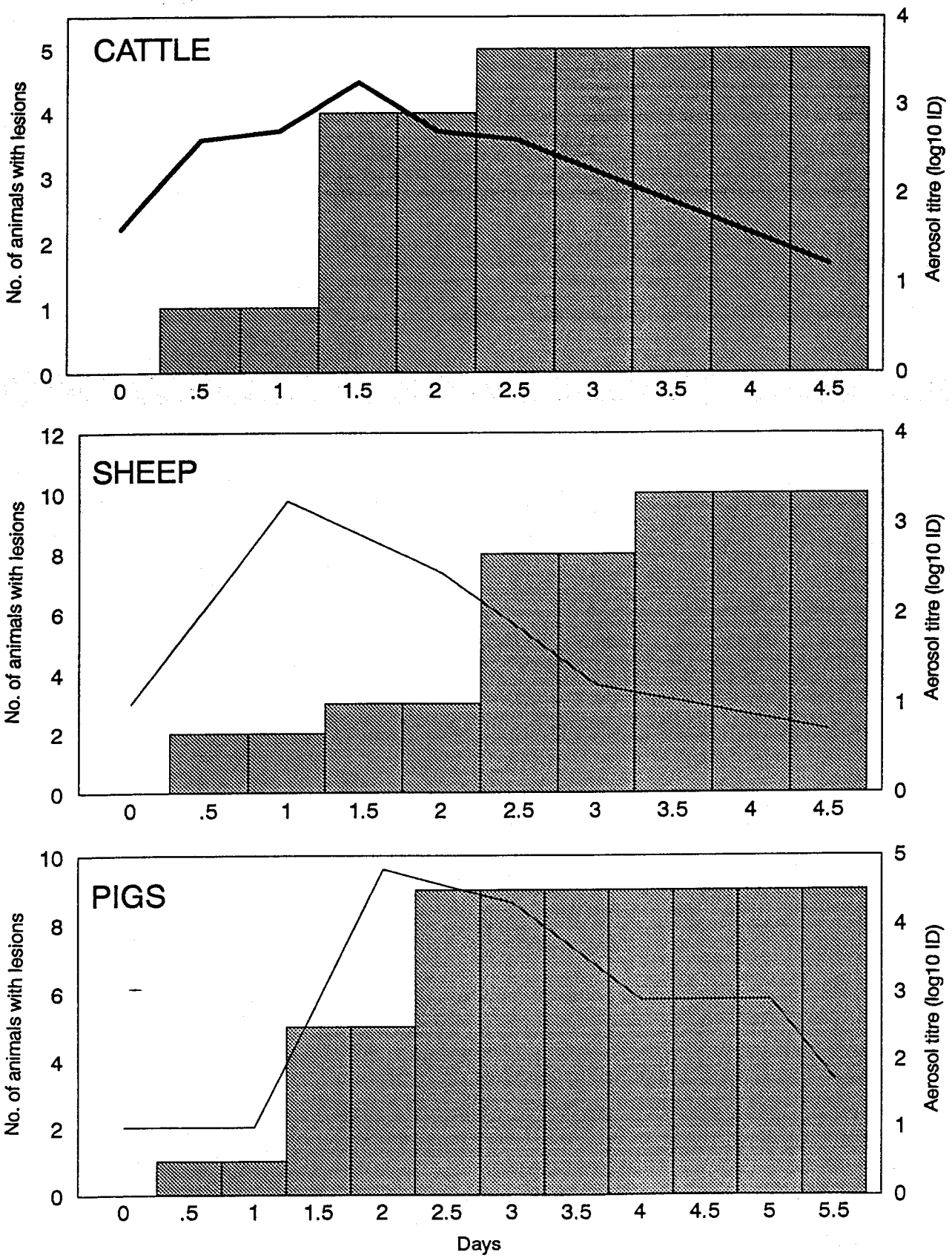
It has been found that the primary site of multiplication of FMDV in animals is the pharyngeal area. It also appears that FMDV infection persists in this site for prolonged periods in sheep and cattle after they have recovered from the disease. Earlier workers reported on the presence of FMDV in oesophago-pharyngeal fluids which they collected with a probang instrument. Since the realisation that virus aerosols produced by infected animals is probably the most important source of infecting virus for other animals attempts have been made to measure virus output from animals. These measurements are made by running an air sampler in the vicinity of infected animals, usually held in loose-boxes, for defined periods and counting the number of virus particles captured during the period. This is usually reported as the total output of virus from the infected animals. However, this does not take into account the fact that the sample is really a measure of the virus concentration produced by the infected animals in their immediate environment. The aerosol data reported in this review has been calculated from various studies as the number of virus particles trapped per cubic metre of air sampled, in an attempt to give a measure of the concentration of FMDV in air produced by infected animals. Because the concentration of virus in the air will depend to some extent on the volume of the air

space surrounding the animals and the ventilation of the area any figures can only be an estimate. In addition, the values obtained for virus concentrations in the air of loose-boxes containing infected animals will probably be higher than those in the air surrounding animals in open spaces. Fitting masks to animals to collect samples may provide a better estimate of virus produced in respired air but is difficult to implement.

Much of the early work measured the titre of virus in oesophago-pharyngeal fluids rather than actual virus output from animals. Some of this work is presented here to demonstrate the appearance of FMDV in the pharyngeal area from which it would be aerosolised and excreted in the breath. Sellers *et al.* (1969) exposed 4 steers to pigs infected with the $O_{1\ BFS\ 1860}$ strain of FMDV and measured the amount of virus in their oesophago-pharyngeal fluids from the third day after contact with the pigs. Virus titres ranged from $10^{1.9}$ to $10^{7.4}$ pfu per sample during the collection period which continued until the eighth day post-contact. Lesions were first detected on the fifth, sixth and seventh day post-contact. In cattle exposed by indirect contact to pigs infected with FMDV type $O_{1\ BFS\ 1860}$, i.e. donor and susceptible animals shared the same airspace but were not in direct contact, virus was found in the pharyngeal region 1 day before the appearance of lesions (Burrows *et al.*, 1971). Virus titres at this time ranged from $10^{3.5}$ to $10^{5.5}$ pfu/sample with an average of $>10^{4.5}$ pfu/sample. When the donor animals were infected with FMDV type $A_{22\ IRAQ\ 24/64}$ the virus titres in the pharynx of susceptible cattle ranged from $>10^{3.8}$ to $10^{4.2}$ pfu/sample one day before lesions were observed. Cattle exposed by contact with donor steers infected with FMDV type $O_{1\ BFS\ 1860}$ had virus present in their pharyngeal region for periods of up to 5 days before lesions occurred (see Table 1 and Figure 1). Virus titres in oesophago-pharyngeal fluids ranged from $10^{1.9}$ to $10^{5.5}$ pfu/sample with a mean of $10^{3.6}$ pfu/sample (Burrows, 1968a).

Since the 1967/68 FMD outbreak in the United Kingdom when evidence was obtained on the possibility of airborne spread of FMDV a number of researchers have measured FMDV aerosol outputs from cattle. For example, Donaldson *et al.* (1970) reported that in cattle, the maximum recovery of FMDV in air was obtained when lesions developed and became generalised. When cattle were infected by the inoculation of FMDV types O_1 and O_2 into their tongue epithelium it was estimated that they subsequently excreted a total of $3x10^4$ $TCID_{50}$ per animal over a 4 day period. Maximum virus recovery occurred 41 hours after inoculation when lesions had generalised (Sellers and Parker, 1969). If the data are expressed in terms of the aerosol created by these animals then aerosol clouds containing 6, 8, 26, 7 and 5 $TCID_{50}$ per cubic metre were present at 17, 22, 41, 46 and 55 hours post-inoculation respectively.

Figure1. Aerosol excretion of FMD virus - Adapted from Donaldson (1978)



Mean amounts of virus recovered per animal in loose boxes containing infected animals
(Air samples colloected for 60 minutes at a sampling rate of 1000 litres/min)

No. animals

aerosol concentration

Table 1. Appearance of FMDV in pharynx in relation to appearance of clinical lesions

Days after entering infected area	CATTLE		SHEEP		PIGS	
	Cumulative Number of animals with:					
	Clinical lesions	Virus in pharynx	Clinical lesions	Virus in pharynx	Clinical lesions	Virus pharynx
1 - 2	0	0	0	0	0	0
3	0	6	0	0	0	0
4	0	10	0	0	0	0
5	3	12	0	3	0	3
6	8		0	7	0	8
7	10		0	8	1	9
8	12		4	8	1	10
9			7	8	3	
10			8	8	5	
11			9	8	6	
12				8	8	
13				9	9	
Total Number of animals	12		9		10	

Sellers *et al.* (1971) reported that cattle infected with the $O_{1\ BFS\ 1860}$ FMDV strain produced aerosol concentrations of 7 $TCID_{50}$ per cubic metre in the loose-box in which they were held (Sellers *et al.*, 1971). In another experiment, Donaldson *et al.* (1970) examined the aerosols produced by cattle after tongue inoculation, intramuscular inoculation and contact with infected cattle using various strains of FMDV (Table 2).

In sheep, the maximum amount of virus excreted in aerosols is found during the first 36 hours after exposure and usually before lesions are seen (Donaldson *et al.*, 1970; Forman *et al.*, 1974). FMDV was demonstrated in aerosols generated by infected sheep for periods of 2-7 days after exposure (Sellers *et al.*, 1977; Sellers and Parker, 1969). Virus was found in the pharynx of sheep for periods of up to 5 days before lesions appeared following contact with FMDV $O_{1\ BFS\ 1860}$ infected donor steers inoculated with FMDV 4 to 7 days prior to the contact period (Table 1). Virus titres in oesophago-pharyngeal fluids ranged from $10^{1.2}$ to $>10^{3.5}$ pfu/sample with a mean of $10^{2.9}$ pfu/sample (Burrows, 1968a). FMDV could be recovered from the pharynx of sheep for at least 28 days after they were exposed for 1 hour to pigs infected with the $O_{1\ MALTA}$ strain, (Donaldson *et al.*, 1981). Maximum virus titres were $10^{6.0}$, $10^{3.25}$, $10^{3.75}$ and $10^{3.25}$ $TCID_{50}$ per ml on days 7, 14, 21 and 28 post-contact. When sheep were infected by coronary band inoculation of FMDV types O_1 and O_2, they were found to excrete FMDV in

aerosols up to a maximum of $10^{3.2}$ $TCID_{50}$ per hour with the total excretion being estimated at $3x10^4$ $TCID_{50}$ per animal over 4 days (Sellers and Parker, 1969). Maximum virus recovery occurred 17 hours after infection and before lesions had been observed. If the aerosol excretion is expressed as the infectivity of the aerosols produced then the air contained 26, 4 and 1 $TCID_{50}$ per cubic metre at 17, 41 and 65 hours post-inoculation.

Table 2. Aerosol production by cattle after intradermal or intramuscular inoculation with FMDV

Days post-infection or contact	Virus Type $C_{LEBANON}$ i/t	$C_{NOVILLE}$ i/t	A_5 i/t	A_{22} i/m	A_{22} contact
1	0.7*	21	2	-	-
2	7	3	7	-	-
3	7	33	5	6	0.7
4	8	0.7	2	9	0.7
5				8	0.7
6				7	0.7
7					11
8					5

* - mean infectivity of aerosol cloud produced by a single animal expressed as $TCID_{50}$ per cubic metre of air

i/t = inoculation into tongue epithelium
i/m = intramuscular inoculation

Gibbs *et al.* (1975a) reported on airborne excretion by sheep after contact exposure, for 2 hours, to cattle infected with type $C_{NOVILLE}$ virus. No clinical disease developed in any of the 4 sheep so exposed. Two of the sheep did not develop detectable serum antibody over 28 days even though they had virus titres of at least 10^2 $TCID_{50}$ per ml in their oesophago-pharyngeal fluids. One animal had $10^{2.25}$ $TCID_{50}$ per ml of oesophago-pharyngeal fluid at day 7 post-exposure, the other had $10^{2.0}$ $TCID_{50}$ per ml of oesophago-pharyngeal fluid at day 10 and $10^{2.75}$ $TCID_{50}$ per ml of oesophago-pharyngeal fluid at day 21 post-exposure. Aerosol clouds containing a mean of 33 and 66 $TCID_{50}$ per cubic metre were produced by each sheep on day 0 and day 2 post-exposure. Oesophago-pharyngeal fluids were collected from these sheep on days 3, 7, 10, 14, 21 and 28 post-contact and virus was found in concentrations of $10^{3.6}$, $10^{4.1}$, $10^{3.6}$, $10^{3.4}$, $10^{2.4}$

and $10^{3.9}$ $TCID_{50}$ per ml respectively. In a further experiment, Gibbs *et al.* (1975b) measured the production of aerosols by sheep exposed for 2 hours to steers with generalised lesions of FMD due to type $C_{NOVILLE}$ virus. No clinical disease developed in any of the 4 sheep exposed, however a viraemia was detected and at 28 days post-exposure each sheep had $>10^4$ $TCID_{50}$ of virus in samples of their oesophago-pharyngeal fluid. The mean concentration of virus in air produced by each sheep was 143, 143 and 15 $TCID_{50}$ per cubic metre on days 1, 4 and 5 post-exposure. The effect of the strain of infecting FMDV on aerosol production was demonstrated by work conducted by Donaldson *et al.* (1970) who examined the aerosols produced by sheep after intradermal inoculation of the coronary band using various strains of FMDV (Table 3).

Table 3. Effect of virus strain on excretion of FMDV in aerosols by sheep infected by intradermal inoculation of the coronary band

Days post-infection	Virus Type			
	$C_{LEBANON}$	$C_{NOVILLE}$	A_5	A_{22}
1	0.02* 84	0.2	0.02	
2	0.02	0.05	0.3	0.02
3	-	0.03	0.2	0.02
4	-	-	-	-

* - mean infectivity of aerosol cloud produced by a single animal expressed as $TCID_{50}$ per cubic metre of air

The aerosol excretion of virus from goats infected after one hour contact exposure to pigs affected with the $O_{1\ MALTA}$ strain isolated from an FMD outbreak was examined by (Donaldson *et al.*, 1981). Mean virus recoveries per animal over a 30 minute sampling period were $10^{2.2}$, $10^{2.3}$, $10^{2.2}$ and $<10^{1.6}$ $TCID_{50}$ per sampling period on days 2, 3, 4 and 5 post exposure respectively. These translate into mean virus concentrations in the air of 5, 7 and 5 $TCID_{50}$ per cubic metre at the time of sampling. Peak levels of virus excretion were about 4 to 12-fold lower than those obtained in studies with sheep and goats using O_1, O_2 and $C_{NOVILLE}$ strains (Donaldson *et al.*, 1970; Sellers and Parker, 1969). Virus was detected in the oesophago-pharyngeal fluids of goats infected by contact with the $O_{1\ MALTA}$ strain at titres of $10^{6.5}$ and $10^{3.5}$ $TCID_{50}$ per ml on day 7 and day 14 post-contact (Donaldson *et al.*, 1981). Trace amounts of virus were detected in the pharynx of these goats for up to 28 days post-contact.

In pigs the maximum concentration of virus in air occurs when there is generalisation of lesions (Donaldson *et al.*, 1970). Sellers *et al.* (1977) examined aerosols from pigs which had been infected by contact with donor pigs infected with FMDV types $C_{NOVILLE}$ and $O_{1\ BFS\ 1860}$ or by intradermal inoculation of the feet with the $C_{NOVILLE}$ strain. Exposure times for contact with infected donor pigs were 48 hours for the $C_{NOVILLE}$ strain and 2 hours for the $O_{1\ BFS\ 1860}$ strain experiment (Table 4). Donaldson and Ferris (1980) examined FMDV in nasal air of infected pigs by using a face mask to sample air from anaesthetised animals. The mean amount of virus in expired air when pigs were exposed to infected donor pigs by direct contact was 95 $TCID_{50}$ per cubic metre during the incubation period and the early stages of disease. When pigs were exposed by indirect contact, i.e. susceptible pigs were separated from the infected donor pigs by a low partition, mean virus titres in expired air were 416 $TCID_{50}$ per cubic metre during the incubation period and the early stages of disease and 1163 $TCID_{50}$ per cubic metre during the advanced stages of disease. When pigs were infected with the same virus by intradermal inoculation the mean levels were 262 $TCID_{50}$ per cubic metre during the incubation period and the early stages of disease and 2160 $TCID_{50}$ per cubic metre during the advanced stages of disease. These measurements were taken using a face mask to collect expired air, however the air sampling device sampled at a rate of 55 litres per minute but the tidal volume of a pig is approximately 10 litre/minute. This suggests that air from the environment also passed through the apparatus probably resulting in a dilution of the expired air. The authors reported that airborne virus was detected in recipient pigs immediately after their removal from contact with donor pigs. This probably represented virus trapped on the bristles around the mouth and on the snout, and perhaps along the lumen of the upper respiratory tract. No virus was recovered from pigs sampled 4 hours after removal from contact exposure indicating that any trapped virus had been dispersed, inactivated or removed by respiratory clearance mechanisms or had been adsorbed and was in the early stages of replication. Pigs infected by intradermal inoculation into the heel with FMDV types O_1 and O_2 were found to excrete virus in aerosols to a maximum of $10^{4.7}$ $TCID_{50}$ per hour. Excretion per animal averaged a total of 10^6 $TCID_{50}$ over 5 days (Sellers and Parker, 1969). Following contact with steers infected with FMDV type $O_{1\ BFS\ 1860}$ pigs were found to be possible sources of FMDV for periods up to 10 days before lesions appeared (Table 1). Virus titres in oesophago-pharyngeal fluids ranged from $10^{0.7}$ to $>10^{3.3}$ pfu sample with a mean of $10^{2.2}$ pfu/sample (Burrows, 1968a). Donaldson *et al.* (1970) examined the aerosols produced by pigs after intradermal inoculation of the coronary band using various strains of FMDV (Table 5).

Table 4. Effect of virus strain on the excretion of FMDV aerosols by pigs

Days after initial exposure/ inoculation	$C_{NOVILLE}$ contact		$O_{1\ BFS\ 1860}$ contact		$C_{NOVILLE}$ intradermal	
	No. with lesions	Aerosol virus	No. with lesions	Aerosol virus	No. with lesions	Aerosol virus
0			0/8	11	0/5	-
1			1/8	590	0/5	38
2	3/7	1026*	3/8	590	5/5	2580
3	6/7	31731	5/8	20	5/5	1665
4	7/7	365	6/8	7	5/5	2700
5					5/5	132

* - mean infectivity of aerosol cloud produced by a single animal expressed as $TCID_{50}$ per cubic metre of air

Table 5. Effect of virus strain on the aerosol excretion of FMDV by pigs after intradermal inoculation

Days post-infection	Virus Type			
	$C_{LEBANON}$	$C_{NOVILLE}$	A_5	A_{22}
1	4*	0	0.4	0.4
2	375	3325	21	296
3	375	1325	12	53
4	47	0	2	3

* - mean infectivity of aerosol cloud produced by a single animal expressed as $TCID_{50}$ per cubic metre of air

Note: Maximum virus titres after type A_5 was inoculated intradermally were 1, 830, 65 and 52 $TCID_{50}$ per cubic metre of air on days 1, 2, 3 and 4 post-inoculation.

Airborne concentrations of FMDV when pigs with generalised lesions were held in a loose-box for 24 hours varied with the strain of infecting virus (Sellers *et al.*, 1971). Mean aerosol concentrations expressed as $TCID_{50}$ per cubic metre production per pig were 1200 for $O_{1\ BFS\ 1860}$, 15080 and 66000 for $C_{NOVILLE}$ and 6980 for $O_{1\ SWISS}$. When pigs were placed in a loose-box for 45 minutes before air sampling the mean aerosol production per pig was 700 and 1320 $TCID_{50}$ per m^3 for the $O_{1\ BFS\ 1860}$ strain and 6600 $TCID_{50}$ per m^3 for the $C_{NOVILLE}$ strain.

The experiments of Donaldson *et al.* (1970) and Sellers *et al.* (1971) would seem to indicate that the infecting strain has some effect on the amount of virus excreted in respiratory aerosols. Infection with the $C_{NOVILLE}$ strain produced more infective aerosols than other strains. Donaldson *et al.* (1970) noted that it was not certain whether the variation between strains in the amounts of virus recovered is due to better survival in air or to greater excretion from the animal. They noted that types O_1 and $C_{NOVILLE}$ are representative of the strains involved in most of the recent epidemics in Europe since 1964. The other strains used in their experiments came from the Near East. It is possible that the difference observed reflects a selection for ability to spread by the airborne route or by animal movement depending on the prevalent climatic conditions. The authors noted that the amount of virus released by a species of animal is only one of the factors influencing the degree of airborne spread after an initial outbreak. The numbers of animals involved, the period before the disease is reported as well as the topography of the area, the livestock density and the climatic conditions are all important.

A summary of the foregoing reports of virus excretion by pigs, sheep and cattle is presented in Table 6 and shows that pigs produce at least thirty times as much aerosol virus as do sheep and cattle.

(ii). Saliva

Most of the reported FMDV titres in cattle saliva are from animals infected by inoculation of virus into tongue epithelium. This technique produces early mouth lesions and the results obtained for the presence of FMDV in saliva may be overestimates because of this method of infection. When bulls were exposed to airborne infection with FMDV strain $O_{1\ BFS\ 1860}$ the titre of virus in their saliva ranged from $10^{1.0}$ to $10^{6.7}$ pfu per sample (Sellers and Parker, 1969). Cattle were found to have FMDV in their saliva for 168 hours after experimental infection with an Asia 1 FMDV strain isolated from Indian outbreaks (Prasad and Kumar, 1981).

Table 6. Aerosol excretion resulting from infection with various FMD virus strains

Virus		Route of infection	Aerosol ($TCID_{50}$ per m^3)
$C_{LEBANON}$	Cattle	i/t	8
$C_{NOVILLE}$	Cattle	i/t	33
A_5	Cattle	i/t	7
A_{22}	Cattle	i/m	9
A_{22}	Cattle	con	11
$O_{1\ BFS\ 1860}$	Cattle	i/t	26
$O_{1\ BFS\ 1860}$	Cattle	con	7
$C_{LEBANON}$	Sheep	i/d	0.02
$C_{NOVILLE}$	Sheep	i/d	84
$C_{NOVILLE}$	Sheep	con	66
$C_{NOVILLE}$	Sheep	con	143
A_5	Sheep	i/d	0.2
A_{22}	Sheep	i/d	0.02
$O_{1\ BFS\ 1860}$	Sheep	i/t	26
$C_{LEBANON}$	Pigs	i/d	375
$C_{NOVILLE}$	Pigs	i/d	2700
$C_{NOVILLE}$	Pigs	i/d	3325
$C_{NOVILLE}$	Pigs	con	31731
$C_{NOVILLE}$	Pigs	con	15080
$C_{NOVILLE}$	Pigs	con	66000
A_5	Pigs	i/d	21
A_{22}	Pigs	i/d	296
$O_{1\ BFS\ 1860}$	Pigs	con	590
$O_{1\ BFS\ 1860}$	Pigs	con	1200
$O_{1\ SWISS}$	Pigs	con	6980

i/t - inoculation of tongue epithelium i/m - intramuscular inoculation
i/d - intradermal inoculation of coronary band
con - infection by contact with infected animals

When lesions had developed in cattle after tongue epithelium inoculation, saliva contained FMDV in the range $10^{5.25}$ to $10^{8.5}$ MID_{50} per ml (Hyslop, 1965). Following intradermal inoculation of FMDV into the tongue, Scott *et al.* (1966) recorded the occurrence of virus in saliva following the appearance of clinical signs (Table 7). Generally, clinical signs occurred one day after inoculation.

Scott *et al.* (1966) also reports that FMDV had been found in saliva of pigs during the first 3 days of disease. No information was provided on the virus titres in the saliva. Information on the excretion of FMDV in saliva from sheep was not found in the literature search conducted as part of this review.

Table 7. Excretion of FMDV in saliva and nasal discharge of cattle after tongue inoculation

Days post-signs	Range of virus titre in saliva	Range of virus titre in nasal discharge
0	$10^{3.2}$ to $10^{7.8}$ pfu/ml	0 to $10^{6.2}$ pfu/ml
1	$10^{4.9}$ to $10^{8.0}$ pfu/ml	$10^{4.7}$ to $10^{7.7}$ pfu/ml
2	$10^{4.0}$ to $10^{6.5}$ pfu/ml	$10^{4.3}$ to $10^{6.2}$ pfu/ml
3	$10^{4.8}$ to $10^{6.1}$ pfu/ml	$10^{3.2}$ to $10^{7.4}$ pfu/ml
4	$10^{3.4}$ to $10^{4.3}$ pfu/ml	$10^{3.7}$ to $10^{6.6}$ pfu/ml
5	$10^{1.8}$ to $10^{4.7}$ pfu/ml	$10^{1.5}$ to $10^{3.7}$ pfu/ml
6	0 to $10^{4.0}$ pfu/ml	0 to $10^{2.8}$ pfu/ml
7	0 to $10^{3.1}$ pfu/ml	no virus isolated
8	no virus isolated	no virus isolated
9	0 to $10^{1.1}$ pfu/ml	no virus isolated

(iii). Milk

The excretion of FMDV in milk by cattle has been studied by many authors primarily to assess the dangers of the spread of FMD by milk and milk products. In many of these studies cattle have been infected by inoculation of virus into the teat canal. However, the results obtained may not reflect the situation which occurs in natural infections and may overestimate the dangers of infection from this source. For example, FMDV excretion in the milk of cows infected by contact was compared with excretion by cows infected by combined inoculation through the teat canal and intravenously with a virulent suspension of type $O_{1\ Lausanne}$ virus by Dhennin and Labie (1977). The viral titres they obtained in milk produced by the inoculated cows were

higher than those of the contact infected cows. In both groups the milk contained FMDV before a rise in body temperature or other clinical signs appeared, and in all cases the levels of virus in milk were higher than blood titres. As noted previously, Hyde *et al.* (1975) found that virus titres in milk from cows infected by intramammary and intravenous inoculation were about 100 fold greater than that reported by Hedger and Dawson (1970), who found FMDV titres of $10^{5.5}$ $TCID_{50}$ per ml, in infected milk from cows in a disease outbreak.

Antibodies have been found in milk, early in the incubation period, together with the virus. FMDV has also been detected in the milk from some cows, infected by intramammary inoculation, for periods of 3-7 weeks in spite of the presence of virus neutralising antibody in the blood (Burrows *et al.*, 1971). It has also been found that interferons are present in milk from cows infected by intramammary inoculation but not in milk from cows infected by the intranasal route (de Leeuw *et al.*, 1980).

In cattle, exposed to infection by direct contact with pigs infected with type $O_{1\ BFS\ 1860}$ FMDV, the concentrations of virus in milk 1 day before the appearance of clinical lesions ranged from $10^{2.4}$ to $10^{5.2}$ pfu/ml (Burrows *et al.*, 1971). When animals were exposed by indirect contact to FMDV type A_{22} virus titres ranged from $10^{1.2}$ to $10^{2.8}$ pfu/ml 1 day before the appearance of lesions. Cows infected by contact with FMDV-infected donor steers excreted virus in their milk for periods up to 4 days before lesions appeared in the animals concerned (Burrows, 1968a). Virus titres observed in the milk from the cows ranged from $10^{1.0}$ to $10^{5.2}$ pfu/ml with a mean excretion of $10^{3.0}$ pfu/ml from each animal.

Burrows *et al.* (1971) reported that FMDV could persist in the udder of convalescent cows and suggested that the udder may be the site for the persistence of some strains of FMDV.

During the FMD outbreak in England in 1967/68, Hedger and Dawson (1970) studied virus titres in milk samples obtained from farms before disease was suspected. They showed that infective virus was being excreted in milk at least 33 hours and probably longer before clinical disease would have been apparent. The virus titre in retail bottled milk prepared from the milk of 59 cows, 8 of which were found to be infected at slaughter was $10^{4.0}$ MID_{50} per ml. Samples of milk from a tanker milk of 1220 gallons capacity which contained 136 gallons of milk from an infected farm of 75 cows of which 8 were found to be infected at slaughter also had virus titres of $10^{4.0}$ MID_{50} per ml. Milk from a tanker containing 547 gallons, 219 gallons of which came from one infected herd of 107 cows in which 1 animal was found to be infected at slaughter contained virus at a concentration of $10^{3.75}$ MID_{50} per ml.

Table 8. Maximum titres of FMDV in various components of milk obtained from cows infected by intramammary and intravenous inoculation

Component	Day 1	Day 2	Day 3	Day 4
		post-inoculation		
Whole	6.38*	4.5	4.66	5.5
Skim	6.29	4.2	4.36	5.76
Cream	6.9	4.19	4.34	5.65
Pellet	4.2	NT	NT	NT

* $\log_{10}$ pfu/ml NT = Not tested

de Leeuw *et al.* (1980) found that FMDV excretion in milk was biphasic in dairy cows infected by intramammary and intravenous inoculation with type O_1 FMDV: a first peak occurring 16 to 24 hours post-inoculation and a second peak on day 3 or 4. Virus was not detected in samples obtained on day 7 post-inoculation. Two cows infected by intranasal instillation developed lesions on day 4, coinciding with the first time virus was detected in their milk. They excreted virus in milk during the next 3-4 days. Virus titres remained between $10^{1.2}$ and $10^{2.7}$ $TCID_{50}$ per ml.

FMDV may be found in all components of milk. The maximum titres of FMDV found by Blackwell and Hyde (1976) in the various fractions of milk obtained from cows infected by intramammary and intravenous inoculation are shown in Table 8.

McVicar and Sutmoller (1972b) have reported on the excretion of virus in the milk of goats infected with 3 strains of FMDV (Table 9). The goats were infected by contact with infected steers.

(iv). Faeces

Excretion of FMDV in faeces has been reported by a number of authors, however in estimating faecal virus output it is important to know the origin of the samples examined. Parker (1971) noted that the virus titres in faecal samples collected from enclosures in which animals are held represents the total virus in the environment rather than the small amount which would have come solely from the intestinal tract. Caution is therefore needed when interpreting faecal excretion data. An examination of the literature shows that samples taken by rectal swab have lower levels of virus than samples collected from the floor of holding pens.

Table 9. Excretion of FMDV in milk from goats following infection resulting from contact with FMDV-infected steers

	FMDV O_1		FMDV A_{10}				FMDV C_4	
Days post-contact	1	2	1	2	3	4	1	2
1								
2								
3								
4						<2.0	2.0	
5	2.7*	6.9				<2.0	3.4	
6	4.4	died	>6.7					3.7
7	3.8		6.7	4.3	2.2			
8	died		6.6	6.7	6.6			
9			5.2	6.0	7.5			
10			3.1	5.2	5.9	<1.4		
11			2.9	3.5	NT	4.5		
12			1.7	3.1		7.0		
13				2.5		6.2		

*$TCID_{50}$ per ml in 2 animals. NT = Not tested

Parker (1971) showed that faecal samples, collected daily from cattle which had been infected by intradermolingual inoculation with 1 of 4 types of O_1 FMDV, usually contained virus between the 2nd and 11th day post-inoculation. Virus titres ranged from $<10^{1.8}$ to $10^{5.5}$ MID_{50} per gram of faeces. Peak yields occurred during the first six days of infection. Sellers and Parker (1969) also infected cattle by intra-dermolingual inoculation with FMDV type O_1 and O_2 and found that maximum titres of FMDV in faecal samples were $10^{4.9}$ $TCID_{50}$ per gram or greater from the 2nd to 5th day of infection.

The amount of virus excreted in faeces appears to be somewhat lower following natural infection. For example, Burrows (1968a) found that the virus titres in rectal samples collected from 2 of 4 cows infected by contact with $O_{1\ BFS\ 1860}$ FMDV-infected donor steers one day prior to the development of lesions were $10^{1.0}$ and $10^{1.6}$ pfu/gram. Similar levels ($10^{1.0}$ to $10^{2.0}$ pfu/gram) were found by Sellers *et al.* (1969) in bulls exposed to airborne infection with the same FMDV strain. Indian researchers have reported that following the inoculation of cattle with Asia 1 FMDV, the virus could be isolated from faeces for up to 216 hours post-inoculation (Prasad and Kumar, 1981).

Parker (1971) also reported that faecal excretion in pigs, infected with 4 types of O_1 FMDV by intradermal inoculation into the heel, was intermittent until 7 to 10 days post-inoculation. Titres of virus in their faeces ranged from $10^{<1.8}$ to $10^{2.9}$ MID_{50} per gram. In a similar experiment, Sellers and Parker (1969) found $10^{2.9}$ $TCID_{50}$ of FMDV per gram of faeces in pigs inoculated in the heel with FMDV types O_1 and O_2. Maximum titres of $10^{2.9}$ $TCID_{50}$ per gram were found on the second or later days after infection. Burrows (1968a) also reported that faecal excretion of FMDV in pigs occurred during the incubation period and that FMDV was present in faeces for periods up to 10 days before lesions appeared in the animals concerned. All infections in this experiment were produced by allowing susceptible pigs to be in contact with infected donor steers inoculated with FMDV 4 to 7 days prior to the contact period. Virus titres ranged from $10^{0.6}$ to $10^{2.6}$ with a mean, for 10 pigs, of $10^{1.3}$ pfu/sample.

Information on faecal excretion of FMDV by sheep is somewhat sparse. After coronary band inoculation with FMDV types O_1 and O_2, maximum titres of virus of $10^{2.7}$ $TCID_{50}$ per gram of faeces were found on the second or later days after infection (Sellers and Parker, 1969). In contrast, Parker (1971) found that virus was only recovered from the faeces of sheep infected with $O_{1\ BFS\ 1860}$ by coronary band inoculation on the second day post-inoculation. A ram infected intranasally with FMDV strain $O_{1/2/72}$ excreted virus in its faeces for 6 days after infection (Nasser *et al.*, 1980). Virus excretion was highest 2 days after infection.

(v). Urine

Early experimental studies in the England showed that FMDV was intermittently present in the urine of infected cattle (anon, 1931). Virus could be detected as early as 48 hours prior to the development of lesions. In an extensive study of infected bulls by Cottral *et al.* (1968), urine taken at 12, 17 and 20 hours following intradermal inoculation of FMDV into their tongue epithelium contained FMDV with titres ranging up to $10^{1.1}$ MID_{50} per ml. During the onset of clinical signs FMDV was isolated from 13 of 15 urine samples. The highest titre recorded was $10^{4.9}$ MID_{50} per ml from a bull at 78 hours post-inoculation. Urine samples of 2 of 6 bulls contained FMDV at 7 days post-inoculation but virus was not isolated during the remainder of the sampling period which extended to 21 days post-inoculation. After the third day of infection, high titres of FMDV were not present in urine samples. Some of the virus titres in the urine from these bulls are shown in Table 10. Prasad and Kumar (1981) reported that FMDV type Asia 1 isolates of Indian origin in experimentally infected cattle persisted in urine for 192 hours post-infection. It has also been reported that FMDV can be isolated from the kidneys of cattle for up to 6 days after intradermal inoculation into the tongue (Hess *et al.*, 1960). The highest virus titres were on the second day after inoculation.

After contact infection, FMDV was detected in urine of 2 of 3 cows (anon, 1937). In one animal, FMDV was detected once at 165 hours after contact with infected animals and 111 hours after lesions first appeared. In the second animal, FMDV was detected at 63 hours and 89 hours after contact with infected animals. The first lesions appeared at 54 hours post-contact. Salt (1993), citing early German work, reported that FMDV was excreted in the urine of cattle for several months after they had recovered from clinical FMD.

No information on excretion of FMDV in urine by pigs and sheep was located in the literature search.

Table 10. Excretion of FMDV in urine of bulls infected by intradermal inoculation of FMDV into the tongue.

	Days post-inoculation		
Virus	1	2	3
A_{119}	0*	1.7	2.1
O_{M11}	0	2.1	0
C_{149}	0	0	0
SAT-1	0	3.8	0.9
SAT-3	0	1.9	2.1
Asia-1	1.1	2.7	2.8

* $\log_{10}$ MID_{50} per ml.

(vi). Semen

Cottral *et al.* (1968) found FMDV in the semen of bulls infected by intradermal inoculation of the tongue as early as 12 hours after inoculation . Virus titres in 6 bulls before the development of clinical signs ranged from $10^{1.7}$ to $10^{4.7}$ MID_{50} per ml. Fourteen of the 16 bulls infected had FMDV in their semen at the time of the onset of clinical signs. The highest titres of virus in semen were $10^{5.7}$ MID_{50} per ml. FMDV was isolated from semen in some bulls for as long as 8 days after inoculation. Further

information on the virus titres in semen observed in this study are shown in Table 11. Kahrs *et al.* (1980) found FMDV titres of up to $10^{3.9}$ pfu/ml in semen of infected bulls prior to the onset of clinical signs. These authors also made the point that even if bulls are serologically negative for FMDV antibody there is no guarantee that semen will be free of FMDV because bulls undergoing primary viral infection can excrete virus in semen. In another study, virus titres of $10^{5.2}$, $10^{2.9}$ and $10^{5.6}$ MID_{50} per ml were found in semen of 3 bulls infected with $O_{1\ BFS\ 1860}$ FMDV on the day lesions were first observed by Sellers *et al.* (1968). These bulls had been infected after being placed in contact with infected steers. Spermatozoa from semen collected on the 18th day after exposure, in this experiment, had a viability of less than 3 per cent which would have precluded its use for artificial insemination. Sellers *et al.* (1969) also found that following exposure of bulls to airborne infection with FMDV strain $O_{1\ BFS\ 1860}$ virus levels in preputial samples ranged from $10^{1.0}$ to $>10^{3.2}$ pfu per sample.

Table 11. Excretion of FMDV in bull semen following infection by intradermal inoculation into the tongue with various FMDV strains

Virus	Days post-inoculation 1	2	3
A_{119}	0*	3.1	2.5
O_{Kaz}	5.0	2.9	0
C_{140}	1.8	2.1	1.9
SAT-1	4.1	4.7	3.5
SAT-3	3.9	1.9	2.2
Asia-1	3.8	4.7	5.0

* $\log_{10}$ MID_{50} per ml.

There are difficulties in detecting FMDV in boar semen. Richmond (1978) found substances in boar semen which either inhibited replication of FMD virus or masked its infectivity. There are also non-specific antiviral and cytotoxic substances in boar semen which provide additional complications for virus detection. The cytotoxic factor, but not the antiviral activity, can be removed by adsorption with kaolin. Because the cytotoxicity induced by boar semen apparently alters the ability of tissue culture cells to support virus replication, pre-adsorption with kaolin increases the probability of detecting this virus in boar semen samples. No other studies were located which used this technique for detecting FMDV in boar semen. McVicar and Eisner (1977) found that, when boars were exposed to infected pen mates and their semen was collected by the gloved hand technique, an insemination dose contained about 250 pfu of virus. A boar killed after natural exposure to FMDV was viraemic and had lesions on the snout, tongue and feet. FMDV was found in testicular tissue but not from epididymis, seminal vesicles, bulbourethral glands and prostate.

No information of excretion of FMDV in sheep semen was found in the literature search.

(vii). Female Reproductive Tract

FMDV has been found in the uterine fluids of 8 of 30 FMD viraemic cattle used for the collection of embryos (Mebus and Singh, 1991). The cows were infected by intravenous inoculation of FMDV type A_5. Virus titres in their uterine fluids ranged from $10^{1.7}$ to $10^{2.65}$ pfu/ml. Following intranasal instillation of the same virus 8 of 11 cows were found to have FMDV in uterine sediments collected during embryo collection (McVicar *et al.*, 1986). Virus titres ranged from $<10^{0.7}$ to $10^{2.05}$ pfu/ml. Vaginal swabs had FMDV titres between $10^{0.7}$ and $10^{5.6}$ pfu/swab with a mean titre of $10^{4.7}$ pfu/animal/swab. Virus was also found in the ovaries and uterine tissues of these animals in high titres. Vaginal samples taken from 4 cows exposed to $O_{1\ BFS\ 1860}$ FMDV-infected donor steers contained virus at least one day before lesions appeared in the animals concerned (Burrows, 1968a). Virus titres ranged from $10^{2.9}$ to $>10^{3.3}$ pfu/swab with a mean of $>10^{3.2}$ pfu/swab.

FMDV was detected in the vaginal fluids of sows exposed to donor steers which were infected with FMDV type $O_{1\ BFS\ 1860}$ for periods of up to 7 days before lesions occurred (Burrows, 1968a). Virus titres in the vaginal swabs ranged from $10^{0.6}$ to $10^{3.0}$ pfu/swab with a mean of $10^{1.6}$ pfu/swab.

(viii). Other Excretions/Secretions:

FMDV may be excreted in a variety of other excretions or secretions such as conjunctival and nasal secretions. Scott *et al.* (1966) reported on the excretion of FMDV in nasal secretions in cattle following tongue inoculation, details of which are given in Table 8. The source of the virus in nasal discharges are likely to be from 2 sources, firstly from virus particles trapped on hairs and surfaces in the nasal passages if animals are in areas where there are infected aerosols, and secondly from oesophago-pharyngeal fluids. High concentrations of virus may be found in blood and vesicle fluids. These sources of virus are most likely to be important during slaughter and disposal of animals. Scott *et al.* (1966) reported that virus titres in vesicles could be as high as $10^{9.8}$ pfu/gram during the clinical stages of FMD.

(b). Virus Presence in Animal Tissues

(i). Muscle

FMDV is rapidly inactivated in muscle tissues by the lowering of the pH, after death, to a level which inactivates the virus. If muscle samples are tested immediately after collection, the presence of FMDV can be demonstrated in animals before and during the occurrence of clinical signs. For example, Dhennin *et al.* (1979) reported that FMDV was detected in the musculature of pigs 20 hours before the development of clinical signs. Prolonged survival of FMDV might be expected in meat if the pH value does not fall below 6.2 (Cottral *et al.*, 1960). This may occur when lactic acid formation is suspended by quick freezing. In such situations FMDV may survive in carcases for months.

(ii). Lymph Nodes

The amounts of FMDV found in the popliteal lymph nodes of cattle following inoculation of tongue epithelium with various virus strains were reported during a study on the possibility of the spread of FMD by meat (anon, 1966). Some examples of the virus titres found in this study which were measured by tissue culture and mouse inoculation are shown in Table 12.

Table 12. Virus titres of FMDV in lymph nodes of cattle after intradermal inoculation of the tongue

Virus strain	sampling time (days p.i.)	pfu/gram	MID_{50}/gram
C_3	6	$10^{4.0}$ to $10^{5.8}$	$10^{3.8}$ to $10^{4.7}$
A_1	3	$10^{1.5}$ to $10^{4.6}$	$10^{0.9}$ to $10^{4.7}$
O_2	5	$10^{4.4}$ to $10^{5.5}$	$10^{3.7}$ to $10^{5.0}$
	13	Not examined	$10^{1.4}$

(iii). Bone Marrow

Cottral (1969) reported that $O_{CANEFA.9}$ FMDV inoculated into the tongue of cattle was found in bone marrow at levels of $10^{4.3}$, $10^{5.9}$, $10^{4.6}$ and $10^{1.0}$ pfu/gram on days 1, 2, 3 and 4 post-inoculation. FMDV was not detected after the 6th day post-inoculation.

(iv). Blood

FMDV was detected in the blood of 4 cows, exposed to $O_{1\ BFS\ 1860}$ FMDV-infected donor steers, for periods up to 2 days before lesions appeared by Burrows (1968a). Virus titres ranged from $10^{1.0}$ to $10^{4.1}$ pfu/ml with a mean titre of $10^{2.2}$ pfu/ml. Titres in the blood of cows 4 to 5 days after inoculation with A_5 virus ranged from $<10^{0.4}$ to $10^{6.0}$ pfu/ml (McVicar *et al.*, 1986). In an experiment where 3 susceptible cows were exposed to 3 other infected animals FMDV could be detected in the blood 51 hours after the initial contact exposure (anon, 1937). The viraemia coincided with the first appearance of lesions and persisted for approximately 24 to 48 hours.

FMDV was detected in the blood of pigs 32 hours before the development of clinical signs by Dhennin *et al.* (1979). Burrows (1968b) also found varying titres of FMDV in the blood of sheep after infection with 4 viruses by coronary band and contact infection (Table 13).

Table 13. Virus titres in blood of sheep following inoculation into the coronary band and after contact infection.

Virus strain	A_{119}	A_{IRAQ}	A_{IRAQ}	O_{ISRAEL}	O_{ISRAEL}	SAT 1
Days post-exposure	i/d	i/d	contact	i/d	contact	i/d
1	2.90*	3.06	-	2.99	-	4.03
2	4.31	2.7	-	3.59	-	5.04
3	2.98	1.75	-	2.31	-	2.20
4	0.85		2.00		1.80	
5	-	-	1.63	-	-	-
6	-	-	-	-	2.35	-
7	-	-	-	-	2.50	-

* mean virus titre $\log_{10}$ pfu/ml.
\- not tested
i/d coronary band inoculation

(v). Intestines

In a study designed to demonstrate whether FMD virus would persist in sheep intestines used as casings for sausages, 5 sheep were infected intranasally and on the buccal mucosa with an FMD virus suspension, and killed 2, 3 or 4 days later (Bohm and Krebs, 1974). FMD virus was present intracellularly in intestines which had been cleaned, salted, and stored at 4°C for 14 days.

(vi). Liver –

Cottral (1969) reported that $O_{CANEFA-9}$ FMDV inoculated into the tongue of cattle was found in liver at titres of $10^{2.6}$, $10^{2.6}$ and $10^{1.5}$ pfu/gram on days 1, 2 and 3 post-inoculation. FMDV was not detected after the 4th day post-inoculation.

(vii). Skin/Hair/Wool

All 7 principal antigenic types of FMDV have been found to have an affinity for skin. For example, it has been shown that FMDV may persist in the skin of infected cattle for 4 to 5 days after the cessation of viraemia by Gailiunas and Cottral (1967). The highest titres of virus, approximately $10^{5.0}$ pfu/gram, were found in the skin of the extremities. Most of the virus appeared to be localised in the epidermal and the superficial layers of the dermis. Steers killed between 10 and 34 days post-inoculation had no detectable virus in skin. The same authors found FMDV in pig and sheep skins from animals which did not exhibit gross lesions or clinical signs of FMD.

FMDV has been isolated from the wool of sheep exposed by intranasal instillation and by contact with FMDV-infected sheep by Eisner and McVicar (1980). The titres of virus were low, $10^{1.9}$ pfu/gram on day 7 post-exposure in wool from sheep infected by contact with infected donor animals and $10^{2.2}$ to $10^{3.0}$ pfu/gram in wool from the intranasally-infected sheep on day 3-4 post-exposure. No virus was isolated from blood or oesophago-pharyngeal fluids collected from the contact infected animals. These animals also did not develop any detectable serum antibodies to FMDV suggesting that the wool might have been contaminated from virus excreted by the infected donor animals.

(c). Summary

A consideration of the possible sources of FMDV from the live animal suggests that aerosols, milk, faeces, urine, semen, bone marrow, lymph nodes and visceral organs are most likely agents for the spread of FMD. Muscle tissue is unlikely to be a source of infection because FMDV is normally inactivated by the biochemical changes occurring after the death of an animal. Skin, hair and wool would seem to be sources of FMDV, but it is difficult to see how they might transmit infection to other animals.

A summary of the daily output of FMDV in aerosols, faeces and milk from contact or naturally infected animals using maximum virus titres reported and data provided by Sellers (1971) is given in Table 14. Examination of the data shows that pigs produce the greatest daily aerosol output of FMDV.

No data was available for FMDV excretion in urine following contact or natural infection. However, virus excretion in urine together with faecal excretion is likely to lead to large amounts of virus in the immediate environment of animals, especially if they are housed.

In relation to milk or semen, it can be calculated that a litre of milk could contain $10^{8.5}$ $TCID_{50}$ of FMDV and that a bovine semen ejaculate, average volume of 2 to 12 ml, could contain between $10^{5.9}$ and $10^{6.8}$ $TCID_{50}$ of FMDV.

Information on FMDV concentrations in bone marrow after natural infection was not located. Using data from animals infected by intradermolingual inoculation, Sellers (1971) calculated that a bovine femur may contain $10^{8.2}$ ID_{50} of FMDV, whilst $10^{6.4}$ ID_{50} may be in a sheep femur and $10^{7.2}$ $TCID_{50}$ in a pig femur. Bone marrow weights used in these calculations were 195 g, 25 g and 11 g for cattle, sheep and pigs respectively.

The amount of FMDV contained in whole bovine and porcine livers was calculated to be $10^{6.3}$ and $10^{8.9}$ $TCID_{50}$ by Sellers (1971), using data obtained from animals infected by intradermolingual inoculation. Bovine popliteal lymph nodes from animals infected by intradermolingual inoculation (anon, 1966) contained $10^{5.6}$ $TCID_{50}$ per gram.

Data such as that calculated above can be used, in conjunction with virus survival data, to calculate the possibility that these sources of FMDV can spread FMDV.

TABLE 14. Potential virus output by animals following natural infection with FMDV

Aerosol Production

	air respired per day	Maximum virus output reported	Daily output
Cattle	90 to 170 m^3	11 ID_{50}/m^3	$10^{3.0}$ to $10^{3.3}$ ID_{50}
Sheep	7 to 10 m^3	143 ID_{50}/m^3	$10^{3.0}$ to $10^{3.2}$ ID_{50}
Pigs	4 to 32 m^3	$10^{4.8}$ ID_{50}/m^3	$10^{5.4}$ to $10^{6.3}$ ID_{50}

Faecal Output

	Volume produced (daily)	Maximum virus output reported	Daily output
Cattle	15 to 45 kg	$10^{2.0}$ ID_{50} per g	$10^{6.2}$ to $10^{6.6}$ ID_{50}
Sheep	0.5 to 3 kg	$10^{2.7}$ ID_{50} per g	$10^{5.4}$ to $10^{6.2}$ ID_{50}
Pigs	0.5 to 3 kg	$10^{2.6}$ ID_{50} per g	$10^{5.3}$ to $10^{6.1}$ ID_{50}

Milk Production

	Volume produced (daily)	Maximum virus output reported	Daily output
Cattle	15 l	$10^{5.5}$ ID_{50} per ml	$10^{9.7}$ ID_{50}

VIRUS SURVIVAL

The longest recorded survival of FMDV in the field occurred during the 1924 California outbreak when virus apparently persisted on a farm for 347 days. During the 1967/68 FMD outbreak in the United Kingdom 11 farms became affected a second time soon after they were restocked (Hugh-Jones and Tinline, 1976). Some of the outbreaks occurred between 41 to 145 days after disinfection had been completed and the reappearance of the disease was attributed to contaminated hay, straw, silage, manure, sacks, animal transport or in one case to a knife used for paring sheep's feet. FMDV is able to survive for some weeks away from animals provided it is protected from direct sunlight and the effects of drying (Murray and Snowdon, 1976). In animal carcases, the amount of virus rapidly decreases because of the low pH reached in the tissues after death. It has been suggested that FMDV is effectively stable in field conditions and that loss of virus occurs more through erosion than loss of viability. An observation that may influence the effect of virus survival on the epidemiology of FMD is that many strains of FMDV are characterised by their resistance to thermal inactivation, but often it is only a small number of virus particles in the inoculum which exhibit this resistance (Bachrach, 1968).

(a). Survival in Aerosols

The factors having direct influence on FMDV survival in aerosols are relative humidity (RH), the pH of its suspending fluid, temperature and ultra violet light (Rumney, 1986). The critical RH is between 55% and 60% where good virus survival is differentiated from poor virus survival. It has been noted by Rumney (1986) that virus strains originating from drier climates survived better in aerosols than those from more temperate climates, with more than 10% of virus surviving at 35% RH. Gainaru *et al.* (1986) noted that conditions favourable to virus survival in aerosols are a relative humidity greater than 60% and environmental temperatures of less than 21°C and little or no wind. The effect of temperature on aerosol virus survival was thought to be minor by (Rumney, 1986) as the virus can survive both sub-freezing temperatures and exposures of 30-60 minutes at 27°C.

Viruses containing RNA, like FMDV, are inactivated by ultraviolet light through changes in their uracil residues (Brown *et al.*, 1963). The RNA core of FMDV is destroyed by ultraviolet light but the protein coat is unaffected. As a consequence, FMDV treated with ultraviolet light retains its antigenicity and ability to attach to susceptible cells but the amount of extractable infectious RNA is diminished. Donaldson and Ferris (1975) attempted to measure the effect of daylight on survival of FMDV by depositing virus droplets from aerosols onto spider microthreads and

exposing the threads to daylight of varying intensities for 30 minutes. Environmental temperatures during the experiment ranged from 21 to 27°C with relative humidities ranging from 76% to 90%, equating to English autumn and winter sunlight conditions. A general photoresistance was demonstrated but the authors noted that it was difficult to predict the extent to which daylight influenced virus survival in aerosols generated by exhaled air and faecal slurries and that relative humidity and temperature will be further confounding variables. They failed to demonstrate any effect of temperature and strong sunlight on the survival of FMDV deposited from aerosols onto spider microthreads. But Rumney (1986) states that survival of FMDV in the presence of ultraviolet light depends to some extent on the nature of the aerosol.

Donaldson (1972) examined the effect of relative humidity on the survival of FMDV in aerosols made from saliva. Maximum survival of FMDV in an aerosol cloud of 1 minute duration occurred when there was a 60% relative humidity. Little infectivity was detected in 1 minute aerosols kept at a relative humidity of 20%. At low relative humidities the survival of A strains of FMDV in aerosols was about 10-fold higher than that for O and C strains. He also noted that the less stable strains appeared to be those which were excreted in greater amounts by infected animals, and conversely the more stable strains were those excreted in the small amounts. The effect of various suspending fluids on the survival of FMDV type $O_{1\ BFS\ 1860}$ was studied by Barlow and Donaldson (1973). Aerosols of the FMDV suspended in saliva were more unstable at high relative humidities than aerosols of virus in nasal fluid, milk, faeces or cell culture fluid. The instability in saliva aerosols appeared to be due to undefined organic molecules in the saliva. If airborne virus is suspended in salivary fluid it is less likely to survive long enough to infect a susceptible animal than if it is suspended in nasal fluid, milk or faecal slurry. Donaldson (1973) used 2 strains of type O_1 FMDV to examine the survival of aerosols of FMDV in milk and faecal slurry. Aerosols generated from pig faecal slurry showed no significant loss of infectivity between 60 and 100% relative humidity but at relative humidities of less than 60% there was increasing loss of infectivity until at 25% relative humidity there was a 90% loss of infectivity. A similar pattern was observed with cattle faecal slurry but infectivity was not lost until 50% relative humidity and at 20% relative humidity only 1% of infectivity remained. Aerosols from milk were also most stable at 50% relative humidity. When aerosols were held at 55% relative humidity for 60 minutes only 0.15 to 5% of infectivity remained in both faecal and milk aerosols. These results were obtained in laboratory experiments and it is possible that under field conditions FMDV aerosols may have decreased stability.

(b). Saliva

Barlow and Donaldson (1973) found an undefined molecule in saliva which reduced the survival of FMDV. However, Scott *et al.* (1966) reported that FMDV in saliva survived for 1 day when held at 37°C, 24 days at 23°C and for at least 35 days at 5°C.

(c). Milk

The reports of survival of FMDV in milk and dairy products are confounded by the method used to infect lactating animals and whether the milk used for virus survival studies has been artificially contaminated with FMDV. For example, Blackwell (1978a) noted that FMDV survived better in milk from infected cows than in milk artificially contaminated with FMDV. Interferons have been detected in the milk of cows inoculated by the intramammary route but not in infected milk obtained from intranasally-infected cows (de Leeuw *et al.*, 1980). The virus titres in the milk from the intramammary-infected cows were also higher, suggesting that the death curves for heat treatment of milk from naturally infected cows may be different. It has also been found that FMDV in milk from the intramammary-infected cows is inactivated more slowly than virus which has been added to milk.

Callis (1979) noted that when FMDV is present in milk or milk products, the virus has an unusual ability to survive otherwise adverse temperatures and pH changes. For example, some viral particles in milk will survive heating to 72°C for 15 seconds and acidulation to pH 4.6. Virus was recovered from milk, obtained from cows infected by intramammary and intravenous inoculation, 12 hours after inoculation and heat treated at 60°C for 0.2, 0.5, 1 and 2 minutes; at 63°C for 0.2, 0.5 and 1 minute; but not at 72°C for 0.2 and 0.5 minutes (de Leeuw *et al.*, 1980). Blackwell (1978a) used milk from cows infected by intramammary inoculation to make butter after milk was collected on days 1 to 4 post-inoculation. FMDV was detected in butter and butter-oil samples prepared from the cream used immediately after collection and also after storage for 18 hours at 4°C. Virus also survived in cream heated at 93°C for 16 seconds. Virus could be detected in butter, made from fresh cream, for at least 45 days at 4°C. Blackwell and Hyde (1976) recovered FMDV from whole milk obtained from cows infected by intramammary and intravenous inoculation even after the milk was heated at 72°C for 5 minutes. It was also found in the skim milk component after heating to the same temperature for 2 minutes, and in the cream component after heating at 93°C for 0.25 minutes. At least $10^{2.0}$ pfu/ml of FMDV was recovered from milk, obtained from cows infected by intramammary and intravenous inoculation, which was pasteurised at 72°C for 15-17 seconds and $10^{1.0}$ pfu/ml in samples pasteurised at 80°C for 15-17 seconds (Hyde *et al.*, 1975). The titres in the raw milk

obtained on day 1 post-inoculation were 100 fold greater than that reported by Hedger and Dawson (1970) in infected milk from cows in a disease outbreak. The authors noted that the experimental infective dose they used was probably too large. However, they reported that skim milk containing $10^{5.0}$ pfu/ml of FMDV was not completely cleared of virus after pasteurisation.

FMDV added to fresh untreated milk survived for 7 days but not for 11 days at 18°C (Sellers, 1969). When FMDV was added to sterilised milk it survived for 35 days at 18°C but not for 45 days. Sterilisation appeared to remove some factor which was unfavourable to virus survival in milk.

TABLE 15. Survival of FMDV in dairy products prepared from milk of infected cows

Whole milk	110°C / 30 sec	survived	de Leeuw et al., (1980)
	60°C / 120 sec	survived	de Leeuw et al., (1980)
	63°C / 60 sec	survived	de Leeuw et al., (1980)
	72°C / 30 sec	FMDV killed	de Leeuw et al., (1980)
	148°C / 2 sec	FMDV killed	Cunliffe et al., (1979)
	72°C / 300 sec	survived	Blackwell and Hyde (1976)
	72°C / 15 sec	survived	Hyde et al., (1975)
	80°C / 15 sec	survived	Hyde et al., (1975)
Cream	93°C / 15 sec	survived	Blackwell and Hyde (1976)
Cultured butter	93°C / 15 sec	survived > 4 months	Blackwell (1978a)
Casein	72°C / 15 sec isoelctric precipitation, pH 4.6	survived 45 days	Cunliffe and Blackwell (1977)
Cheese			
Cheddar	No Heat	survived 90 days curing but not 120 days curing	Blackwell (1976)
Cheddar	63°C / 6 sec	survived <30 days curing	Blackwell (1976)
Camembert	72°C / 15 sec	survived 21 days but not 35 days	Blackwell (1976)
Mozzarella	72°C / 15 sec	FMDV inactivated during cheese making	Blackwell (1976)

Cottral (1966) reported that FMDV can survive in dried milk for at least 2 years.

FMDV survival times in milk and milk products are summarised in Table 15 which is a modification of that published by Blackwell (1984). There is little information available on the survival of FMDV in milk from cows with natural infections.

The significance of the survival times of FMDV in milk and milk products needs to be considered in relation to the pasteurisation recommendations in the Australian Food Standards Code. These recommend that milk for human consumption should be pasteurised by heating at a temperature between 61°C and 65°C for 30 minutes; or between 72°C and 73.5°C for 15 seconds. Most milk is pasteurised at the latter temperatures. The recommendations for clotted or scalded cream are heating at 83°C for at least 30 minutes. The research work discussed above would suggest that the present recommendations for pasteurisation would not be completely effective in eliminating FMDV from milk and that it could be a means of disseminating virus from infected areas, especially if fed to susceptible animals.

(d). Faeces

Callis *et al.* (1980) reported that FMDV could survive under natural conditions for 14 days in dry manure from animal stalls, for 8 days in moist manure from the same stalls and for less than 6 days in manure mounded to a depth of 30 cm. Faeces from infected cattle which contained $10^{5.3}$ to $10^{5.7}$ pfu per gram of type O_1 FMDV and held at 4°C lost 1 log of virus in the first 2 weeks of storage and 2 log units after 3 weeks (Parker, 1971). After a holding period of 9 weeks only 0.1% of the original virus remained. During the same time the pH fell from 7.25 to 6.70. In liquid manure at pH 8-8.4 and 4°C about 10% of the original virus remained after a month (Bauer and Eissner, 1972). The survival of FMD virus in liquid manure with an 87-95% moisture content and pH 6.8-7.8 was studied by Rozov and Andryunin (1972) by adding FMD virus with a titre of 10^6 $TCID_{50}$ to the liquid faecal suspension. It was found that virus survived for 34-42 days at 12-22°C and up to 180 days in frozen manure during the winter. Virological examinations of a compost prepared by mixing infected manure with sawdust, peat or straw in the proportion of 1:4, with samples taken at various depths, showed that it was not always free from FMD virus. It has also been reported that FMDV survived in water used to wash down animal pens when it was held at a temperature of 17-21°C for 21 days, at storage temperatures of 13-18°C virus survived for 49 days and at 4-13°C for 103 days.

These observations reinforce the need for safe removal and disposal of manure from buildings, vehicles and other areas to prevent the spread of FMD.

(e). Urine

Callis *et al.* (1980) found that FMDV survived in urine for 39 days under natural conditions . This would appear to be unusual as the pH of normal urine should inactivate the virus.

(f). Skin/Hair

Titres of FMDV in wool taken from infected sheep were reduced after storage for 2 weeks at -80°C (Eisner and McVicar, 1980). After the wool was stored for an additional 7 days at room temperature no virus could be recovered from samples that were originally virus positive. Artificially contaminated wool, contaminated with approx 10^5 pfu, was also examined and virus titres of $10^{1.5}$ pfu/gram was recovered after 7 days storage at room temperature but not after 14 days storage. The authors noted that the survival of FMDV on wool may depend on how virus is deposited on wool. Lesion material and salivary and nasal discharge may contain large amounts of virus and heavily contaminate wool, while faeces usually contain lower amounts of virus and would be less likely to be a contamination risk. Other factors influencing FMDV survival on wool include temperature, relative humidity and sunlight in storage. Dardiri and Hamdy (1978) reported that FMDV may survive for up to 30 days in wool bales stored at 13-15°C.

Gailiunas and Cottral (1967) examined the survival of FMDV in cattle hides treated by 4 conventional methods. The hides used were from steers killed at an acute stage of infection with FMDV. In green salted hides, stored at 15°C, FMDV persisted for up to 90 days and for 352 days at 4°C. Hides cured for 20 hours in saturated brine with the addition of up to 500 ppm of available chlorine still had infectious FMDV after 4 weeks storage at 15°C. FMDV was also detected in a hide sample dried for 42 days at 20°C and 40% relative humidity. Hides cured in salt for 7 days and then dried at 20°C were found to be infectious for 21 days.

(g). Semen

Cottral *et al.* (1968) found that semen from bulls infected by inoculation of FMDV into the tongue epithelium did not lose virus titre during storage at -50°C for as long as 320 days. The semen samples, were in some cases, diluted with egg yolk extender before storage. Gierloff and Jakobsen (1961) found that FMDV remained viable for 1 month in artificially contaminated bovine semen stored at -79°C.

(h). Animal Tissues

Under normal conditions the biochemical reactions which occur in muscle after death cause a rapid fall in tissue pH which is detrimental to the survival of FMDV. Cottral *et al.* (1960) noted that inactivation of FMDV in carcases is brought about by changes in pH and other components of meat tissue which occur after death. Prolonged survival of FMDV might be expected in meat if the pH value does not fall below 6.2. Cottral *et al.* (1960) found that when lactic acid formation is suspended by quick freezing FMDV may survive in carcases for months. They also observed that conditions were favourable for virus survival near the superficial surface of carcase muscles when a carcass has been chilled rapidly. These authors also reported that FMDV was not found in infected cattle carcases stored at -1°C for 13, 14, 22, 24 and 33 days. Blackwell (1984) reported that FMDV was inactivated within 48 hours in skeletal muscle kept at 4°C but that it survived for a minimum of 120 days at the same temperature in lymph nodes, coagulated blood and bone marrow from the same carcases. FMDV has also been found in cheek and tongue tissue stored at -1°C for 33 days, and in meat-wrapping cloths stored for 40 days. In infected beef stored at 4°C, FMDV survived in muscle tissue for 24 hours, but was inactivated by the 3rd day when the pH fell to 5.3. Similar studies with infected pork failed to demonstrate FMDV after 1 day at usual holding temperatures and in frozen pork held for 12 days, but FMDV was found in the bone marrow of these animals. Viable FMDV was not found in lymph nodes of beef stored at 4°C for 5 to 6 months (Cottral *et al.*, 1960). However, the same authors also demonstrated that FMDV survived in unsalted lymph nodes stored 16, 30 and 50 days, and in cured lymph nodes held for 30 and 50 days. The reason for the prolonged survival of FMDV in bone marrow is because it does not develop sufficient acidity to inactivate FMDV. The virus has been observed to survive in the bone marrow of beef held for 76 days, in chilled pork for 42 days and in frozen pork for 76 days (Cottral *et al.*, 1960). FMDV survived in bone marrow for at least 80 days in beef carcases held at -1°C. Cox *et al.* (1961) also found that FMDV survived in the bone marrow from the ribs of an infected beef carcase stored at 1°C for 194 days. In other studies using samples from the forequarters cut from carcases of infected donor cattle stored at 4°C, FMDV was detected in the rib bone marrow at 14, 60 and 73 days and was present in lymph nodes and large blood clots when tested at 60 days (Cottral *et al.*, 1960). It has been reported that FMDV in bone marrow from an infected carcase, which had been frozen and thawed 6 times in the course of 30 days, was still infective (anon, 1931). FMDV has been found to survive for at least 4 days in infected blood which was deliberately splashed on the surface of a swine carcass held at 15°C (Cottral *et al.*, 1960). Henderson and Brooksby (1948) found that blood clots in carcases stored at 4°C remained infective for 6 weeks. FMDV was found in rumen tissue from infected cattle stored at -20°C for nearly 6 months (Cottral *et al.*, 1960). At the same storage temperature, FMDV was found to survive in liver for nearly 6 months and in

kidneys for as long as 7 weeks. Other studies with bovine liver and spleen held at 4°C showed that FMDV survived for more than 24 hours but was inactivated after 48 hours storage. Frozen liver, lung, spleen, kidneys, brain and stomach from infected pigs carried virus for long periods. Storage of these organs at approximately 21°C showed that FMDV survived for as long as 4 to 6 days but by the 10th day it was present only in kidney and blood. Henderson and Brooksby (1948) found that livers from FMDV-infected cattle remained infective for 4 months when stored at -20°C.

(i). Animal Products

Meat

As noted above, the fall in muscle pH induced by biochemical changes following death is detrimental to the survival of FMDV in muscle tissue. However, FMDV will survive in bone marrow, lymph nodes and visceral organs, such as the liver. Blackwell (1984) found that FMDV, in infected lymph nodes packed in minced beef, was inactivated when processed by retort cooking to an internal temperature of 68.3°C. It also survived in hams prepared from infected pigs and cooked to an internal temperature of 69°C. FMDV survived in minced beef cooked in flexible pouches to an internal temperature of 72°C but not at a temperature of 79.4°C. Garcia-Vidal *et al.* (1988) also examined the effect of cooking meat in flexible pouches on the survival of FMDV. Cooking the meat products at 75°C for 20 minutes or 80°C for 15 minutes was virucidal, but FMDV survived cooking at lower temperatures and for shorter periods.

Cottral *et al.* (1960) found that curing mixtures, containing various salts, had little effect on FMDV survival in cured products such as corned beef or hams. Survival of FMDV in pickling solutions containing 24% sodium chloride has been reported and uncured bacon frozen at -15°C contained FMDV for at least 55 days of the storage time. In studies of hams, shoulders of pork, salted bacon and sausages, the longest survival of FMD virus was 190 days in salted bacon, while the virus survived for up to 183 days in ham fat (Dhennin *et al.*, 1980). Virus survived longest in adipose tissue. FMDV could not be recovered from Parma hams made in the U.S. from experimentally infected pigs after 108 days, and in hams made in Italy from naturally infected pigs after 170 days (McKercher *et al.*, 1987). FMDV was found by Savi *et al.* (1962) to survive in the bone marrow of hams for up to 89 days post-processing. Savi and Baldelli (1962) also reported that sausage, salami and mortadella made from infected meat and fat were not infected because the lowering of the pH during processing killed FMDV. A similar situation appeared to occur in other Italian sausages, even those with a high fat content such as capacola. FMDV was also not detected 56 days after processing of sausages prepared from five pigs infected with FMD (Dhennin *et al.*,

1980). It was noted that FMDV was more readily isolated from fat than muscle tissue. FMDV can occur in sausage casings and Bohm (1975) and Bohm and Krebs (1974) found that contaminated salt-cured sheep intestines stored for 14 days at +4°C had the same virus titre ($10^{2.5}$ $TCID_{50}$ per ml) as before salting.

Cheese

The comments made in regard to survival of FMDV in milk and the use of data from experimentally infected cows also applies to dairy products. It is possible that survival times of FMDV in dairy products might be different if they were made from milk obtained from naturally infected cows. The data given in this review serve as a guide in the assessment of the dangers of dairy products as a means of spreading FMDV.

Blackwell (1976) used milk from cows infected via the intramammary and intravenous route for making Cheddar, Mozzarella and Camembert cheese. FMDV survived processing but not curing for 30 days in Cheddar cheese prepared from milk heated at 63°C for 6 seconds. FMDV survived curing for 60 days but not 120 days in Cheddar cheese made from unheated milk. FMDV survived in Camembert cheese for 21 days at 2°C but not for 35 days. This cheese was prepared from milk pasteurised at 72°C for 16 seconds. FMDV failed to survive processing for Mozzarella cheese when made with milk pasteurised at 72°C for 16 seconds. FMDV was detected by in Cheddar cheese made from heat treated milk from infected cows, 24 hours after pressing but not at 30 days (Blackwell, 1975). The milk was treated at 67°C for 1 minute.

The Australian Food Standards Code recommends that milk for cheese making is heated for 15 seconds at either 66°C or 73°C. This would suggest that cheese prepared from milk produced by FMDV infected cows, following any outbreak in Australia, may be infective, at least for periods of up to a month.

Whey

FMDV was detected in the sweet whey by-product of Cheddar and Camembert cheese made from milk of FMDV-infected cows, but not in the acid whey by-product of casein manufacture (Blackwell, 1978b). The milk used for the production of the whey was heated at 72°C for 15 seconds and had a pH of 4.6 for the production of acid whey and a pH of 5.2 for the sweet whey. The whey constituents, alpha-lactalbumin, beta -lactoglobulin and lactose, produced from sweet whey, were not infective when inoculated into cattle.

Casein

FMDV survived in casein obtained by coagulating skim-milk with hydrochloric acid and leaving the mixture for one hour (Gaggino *et al.*, 1977). Dried casein produced from pasteurised milk of dairy cows infected with FMD virus retained infectivity for cattle in one of seven tests for 42 days' storage at 25°C (Cunliffe *et al.*, 1978). Infective FMDV survived pasteurisation of milk at 72°C for 15 seconds, acid precipitation and washing of casein, followed by drying of the casein in a hot air flow and conversion to sodium caseinate.

Cattle inoculated with casein made by isoelectric precipitation from milk of FMDV-infected cows became infected in one of two trials where raw skimmed milk was used and in three of six where the skim milk was pasteurised, at 72°C for 15 seconds, and used to make the casein (Cunliffe and Blackwell, 1977). Samples from one of two dried casein batches infected test cattle, and samples from four of six batches of casein from uninfected cow's milk to which FMDV was added before pasteurisation also infected cattle.

These are the processes that are used for casein manufacture in Australia, including the pasteurisation temperature regime, suggesting that processing of milk from FMDV-infected cows into casein would not be a safe method of disposing of such milk during an FMD outbreak.

(j) Fomites and Feeds

Early British studies found that FMDV survived for 2-3 days in blood dried on glass, iron, zinc, ceramic tiles, brick or wood. But when infected blood was dried on leather from a boot or rubber from a gum boot FMDV survived for 80 to 102 days (anon, 1931).

Callis *et al.* (1980) reported that FMDV survived for up to 28 days on the surface of soil in autumn and for 3 days in summer. However, the location of this experiment was not given. These authors also reported that FMDV survived on sacks for 20 weeks and on hay dried at 22°C. Early studies in America found that FMDV remained infective for at least 25 days in dried garden soil saturated with a suspension of FMDV and kept at 9°C for 10 days and then at 20°C (Olitsky *et al.*, 1928). The soil was kept moist for 15 days and then allowed to dry out. Gailiunas *et al.* (1969) citing Camion and Gatto (1961) noted that FMDV survived for 11 days on soil under natural conditions in Argentina. They also cite reports from Russia which stated that in the Kazakhstan province, a drought area, FMDV survived on contaminated plants and soil

for up to 15 days in summer and up to 165 days in autumn and winter. FMDV also survived in hay stacks in the same area for 185-200 days.

FMDV has been found to survive on straw, flour, cow hair and cane sugar for periods of 5 days to 7 weeks, although on straw and flour its survival was irregular (anon, 1928). FMDV on hay and bran survived for 15 and 20 weeks respectively provided these contaminated materials were stored under conditions of relative dryness and in the absence of light. It was also found that FMDV in watery filtrates survived for 20 weeks on bran (anon, 1931). Bran, hay and straw soiled with FMDV-infected milk remained infective for 17 days, with the contaminated bran still infective 32 days after contamination (anon, 1931). However, FMDV did not survive on wood soiled with infected milk.

Gailiunas *et al.* (1969) found that FMDV survived for 33 to 35 days in serum which had been smeared on cardboard, wood and metal. When infected blood was smeared on the same materials, FMDV survived for a period of 55 days. FMDV in ground lymph nodes smeared on cardboard, wood and metal survived for 57 days, but when fat was the suspending medium, virus survival was increased to 398 days. In all these experiments, the contaminated cardboard, wood and metal were stored at 4°C and at a relative humidity of 85%.

On fabrics such as paper, silk and wool, and in sand and butter FMDV was found to survive in a dry state for up to 14 days (anon, 1928). FMDV in blood on hessian and stockinette used to wrap chilled and frozen carcases retained its infectivity for 45 days at 13.4 to 30.4°C storage and at -2 to +2°C (anon, 1931). Gailiunas *et al.* (1969), citing German reports, noted that FMDV had survived for up to 100 days when dried on clothing. In addition, a case was reported where FMDV was apparently transported in the grooves of tyre treads.

It is difficult to assess the information available on the survival of FMDV on fomites because often there is no indication of the amount of virus recovered at the time of sampling. The methods used to detect FMDV in some studies are usually animal or tissue culture inoculation which may detect extremely small amounts of virus that in nature may not be sufficient to transmit FMDV. The data are presented here as an indication of the survival times of FMDV on various surfaces. However, the survival of FMDV on such surfaces does not mean that they will be a source of infection for animals. Survival of FMDV on fomites is affected by the medium in which the virus is suspended. For example, survival times for free virus are shorter than when FMDV is within cells and is protected by colloids and tissue debris (Cottral, 1969).

(k). Summary

The survival of FMDV in various secretions and animal products can be grouped according to the length of time it survives in unprocessed and processed products. FMDV appears to have a relatively short survival time, usually less than 24 hours, in aerosols, urine, muscle, liver and spleen held at normal temperatures. FMDV may survive for extended periods in faeces, saliva and skin and on wool. Prolonged periods of virus survival occur when FMDV is contained in semen, bone marrow and lymph nodes held at low temperatures. FMDV can also survive in milk subjected to normal pasteurisation procedures and may subsequently survive for prolonged periods in cheese made from pasteurised milk, and for greater periods in dried milk.

FMDV appears to survive for long periods when body fluids and tissues are dried on fomites.

The ability of FMDV to survive in aerosols is believed to be one of the most important factors in the spread of FMD in Great Britain and Europe. Wind-borne spread of FMDV is most likely when climatic conditions are favourable for virus survival in aerosols.

The survival of FMDV in aerosols formed from saliva is poor, but may be prolonged when held in a moist, dark conditions. It is likely that FMDV in saliva will only be important in close contact spread between animals, and as a contributor to the contamination of the environment in areas where animals are held. FMDV in faeces and urine will also contribute to the levels of virus in the environment. FMDV may survive in liquid faecal wastes for prolonged periods and could be a source of infection, especially if such wastes are disposed of in such a way as to create aerosols.

FMDV concentrations in semen and its prolonged survival after processing and storage for artificial insemination use suggest that semen may be a vehicle for the spread of FMD.

ROUTES OF INFECTION

The diversity of portals of exit and entry, the numerous tissues in which it will multiply, and the extreme range of clinical manifestations all contribute to making FMDV a well-adapted parasite (McVicar, 1977). FMDV can readily infect abraded epithelium and can gain entrance to the bloodstream from lung alveoli and thereby reach numerous multiplication sites (McVicar, 1977). In cattle, inoculation of virus into the epithelial layers of the tongue is the most reliable method of producing experimental infections with minimal doses. In both sheep and pigs, inoculation of virus into the coronary band of the feet or the bulbs of the heels is the most reliable method of producing FMD infection. However, the main portals for entry of infection are the respiratory tract, digestive tract and the female genital tract. The skin or conjunctiva are probably unimportant portals for entry of virus. This review has concentrated on the amounts of virus needed for successful infection through these routes. Data on the amounts of virus needed to cause infection by the intradermal, intramuscular, intravenous, intramammary, intratracheal, intraperitoneal and sub-cutaneous routes may be found in the review by (Sellers, 1971). Experimentally, the minimum infective dose for cattle by intradermal tongue inoculation has been found to be 0.01 to 2.0 $TCID_{50}$ by Cottral *et al.* (1965). Henderson (1952) compared the doses of FMDV needed to produce infection by subcutaneous injection and by intradermal inoculation of the tongue. Intradermal inoculation into the tongue required an average of several hundred to several thousand times smaller doses to produce infection when compared to sub-cutaneous inoculation. There was a strain effect with one strain requiring 250,000 times as much virus to produce infection by the subcutaneous route as by the intradermal route while another strain only required 4 times the dose.

There also appears to be marked strain differences in the amounts of virus needed to produce infection both by natural and experimental routes.

(a). Aerosol infection

Donaldson (1981) and Donaldson *et al.* (1987) conducted studies to determine the minimum infective aerosol doses of FMDV for sheep and cattle. Animals were exposed for 1-15 minutes to either artificial aerosols, produced by a spinning-top (May) generator, which were homogenous and less than 3 microns diameter or to natural aerosols from infected pigs which were heterogenous in particle size. Exposure for 10 minutes to 1-50 $TCID_{50}$ of strain $O_{1\ BFS\ 1860}$ virus infected 58% of sheep. The lowest dose which infected sheep was 10.6 $TCID_{50}$. Around 27% of sheep which were infected did not develop overt disease. The lowest doses which infected cattle were 12.5 $TCID_{50}$ of

strain $O_{1\ BFS\ 1860}$ and 25 $TCID_{50}$ of strain SAT 2. Following exposure to low doses of FMDV, 12 to 316 $TCID_{50}$, 33% of cattle exposed to $O_{1\ BFS\ 1860}$ virus and 27% exposed to SAT 2 virus were infected but none developed vesicular lesions. Seventy three (73) percent of cattle exposed to natural aerosols and receiving doses of between $10^{1.4}$ and $10^{2.4}$ $TCID_{50}$ became infected but 26.7% did not develop clinical lesions. This finding agrees with that of Sutmoller *et al.* (1968) who reported inapparent infection in cattle given minimal amounts of FMDV by intranasal or intrapharyngeal inoculation. It was noted that in cattle exposed to artificial aerosols of FMD virus the incubation period was extended to 6-10 days, compared to the 3-7 days seen after exposure to natural aerosols. This was thought to be due to the nature of the artificially generated aerosols which had particles of relatively uniform diameters of 3 microns. In contrast, natural aerosols tend to be more variable with particles which range in size from less than 3 microns to greater than 10 microns. The authors noted that smaller particles were probably inspired and caused primary infection in the lungs whilst larger particles were trapped higher in the respiratory tract and initiated infection in the pharyngeal region. There is also evidence to suggest that alveolar route is less efficient than the pharyngeal route in initiating infection with FMDV.

In an experiment designed to determine the minimum aerosol dose of FMDV required to infect sheep, Gibson and Donaldson (1986) reported that 7 of 12 sheep exposed for 10 or 15 minutes to air extracted from a cabinet containing infected pigs and containing 1 to 50 $TCID_{50}$ became infected, while 83% those who received a dose of 51 to 100 $TCID_{50}$ became infected, and all sheep exposed to more than 100 $TCID_{50}$ became infected. The lowest dose which infected sheep was 10.6 $TCID_{50}$.

Terpstra (1972) reported that infection in pigs could be established by the aerogenic route with a total inhaled dose of approximately 600 MID_{50}.

(b). Conjunctival infection

Sutmoller and McVicar (1973) studied the possibility that aerosols of FMDV could result in conjunctival infection. Steers were exposed to FMDV type O_1 by instillation of one drop of virus suspension containing 10, 100, 1000 or 10000 pfu into the conjunctival sac. The steer inoculated with 10 pfu of virus remained clinically normal and virus could not be recovered from lacrimal fluid, oesophago-pharyngeal fluid, blood or faeces. The other doses produced lesions of FMD and virus in all the above fluids. Highest titres in oesophago-pharyngeal fluids were $>10^{6.0}$ pfu/ml on day 2 post-inoculation. In spite of these findings the authors conclude that aerosolized virus has more chance of being trapped in the respiratory tract than on the conjunctiva.

(c). Oral infection

The amount of FMDV needed to produce infection by the oral route is many times that required for infection by aerosol in cattle, sheep and pigs.

Burrows *et al.* (1981) found that steers fed $10^{6.7}$ pfu of FMDV type O_1 in food or water on 2 occasions with a 7 day interval did not become infected. While Sellers (1971) found that less than half a group of cattle given $10^{5.8}$ to $10^{6.8}$ $TCID_{50}$ of FMDV by mouth became infected. Successful infection of pigs by the oral route required doses of $10^{5.0}$ to $10^{6.5}$ $TCID_{50}$ (Terpstra, 1972; anon, 1925; anon, 1931; Henderson, 1952). The course of the FMD in pigs following oral infection was observed by Terpstra (1972) to be more rapid than when infection was by aerosol with virus present in a number of tissues within 24 hours of infection. In contrast, following inhalation virus was only observed in lung tissues for up to 48 hours after infection. There appears to be no information on the amounts of virus needed to infect sheep by ingestion.

(d). Infection via the reproductive tract

Research by Cottral *et al.* (1968), using semen from bulls infected with a variety of FMDV strains by intradermal inoculation of the tongue, demonstrated that doses of FMDV in semen of between 2300 and 2,500,000 MID_{50} would produce infection in heifers. In general, only heifers which were in oestrus when inseminated became infected. There does not appear to be any comparable data for sheep or pigs.

(e). Summary

The reported lowest doses of FMDV shown to initiate infection by the respiratory and oral routes are shown in Table 16.

Inhalation is the most effective route of infection with FMDV with only 10^1 to 10^2 $TCID_{50}$ needed to initiate infection, compared to a dose of 10^5 to 10^7 $TCID_{50}$ necessary to initiate infection by the oral route. Other portals of entry of infection such as sub-cutaneous, conjunctival or reproductive tract appear to be only of minor importance.

Table 16. Lowest doses of FMDV reported to infect animals by the oral and respiratory routes

Route	Species	Virus	Dose*	Reference
AEROSOL	Cattle	$O_{1\ BFS\ 1860}$	12.5	[Donaldson et al, 1987]
		SAT 2	25	[Donaldson et al, 1987]
	Sheep	$O_{1\ BFS\ 1860}$	10.6	[Gibson and Donaldson, 1986]
	Pigs	$O_{1\ WEERSELO}$	600	[Terpestra, 1972]
ORAL	Cattle	$O_{1\ BFS\ 1860}$	$10^{5.5-6.5}$	[Burrows et al, 1981]
			$10^{6.5}$	[Henderson and Brooksby, 1948]
	Pigs	$O_{1\ WEERSELO}$	$10^{5.4}$	[Terpestra, 1972]
		O_{39}	$10^{5.2}$	[Anon, 1931]
		O_{ASJ}	$10^{5.0}$	[Henderson and Brooksby, 1948]
		O	$10^{6.5}$	[Henderson and Brooksby, 1948]

* total dose in $TCID_{50}$

ORAL AND RESPIRATORY INTAKE

Information on oral and respiratory intakes can be used, in conjunction with virus excretion data, virus survival and infectious dose information to estimate the likelihood of various means of disease transmission.

Data in (anon, 1974) show resting respiration intakes for adult cattle ranging from 92 l/min for Jerseys to 114 l/min for Holsteins. Similar information for Hereford heifers (body weight 144 kg) was 46 l/min and for Holstein heifers (body weight 164 kg) was 61 l/min. Figures for sheep are given as 7.2 l/min and for pigs (body weight 225 kg), 37 l/minute.

Chamberlain (1970) citing Scott-Russell (1966) gave the following data for herbage grazed and air inspired per day for sheep and cattle.

	Herbage kg/day dry wt.	Air m^3/day
Cow	15	130
Sheep	1.2	9

Sellers and Parker (1969) noted that the volume of tidal air breathed varies with species. He reported that at rest a 400 kg Holstein cow would sample 85 l/min, a 25 kg pig 9.27 l/min and a sheep about 5 l/min. These are slightly lower than the data provided above with daily air inspiration equivalents of 123 m^3 for cattle and 7.2 m^3 for sheep. Within species the tidal air volume in adults is greater than in young animals.

MODES OF TRANSMISSION

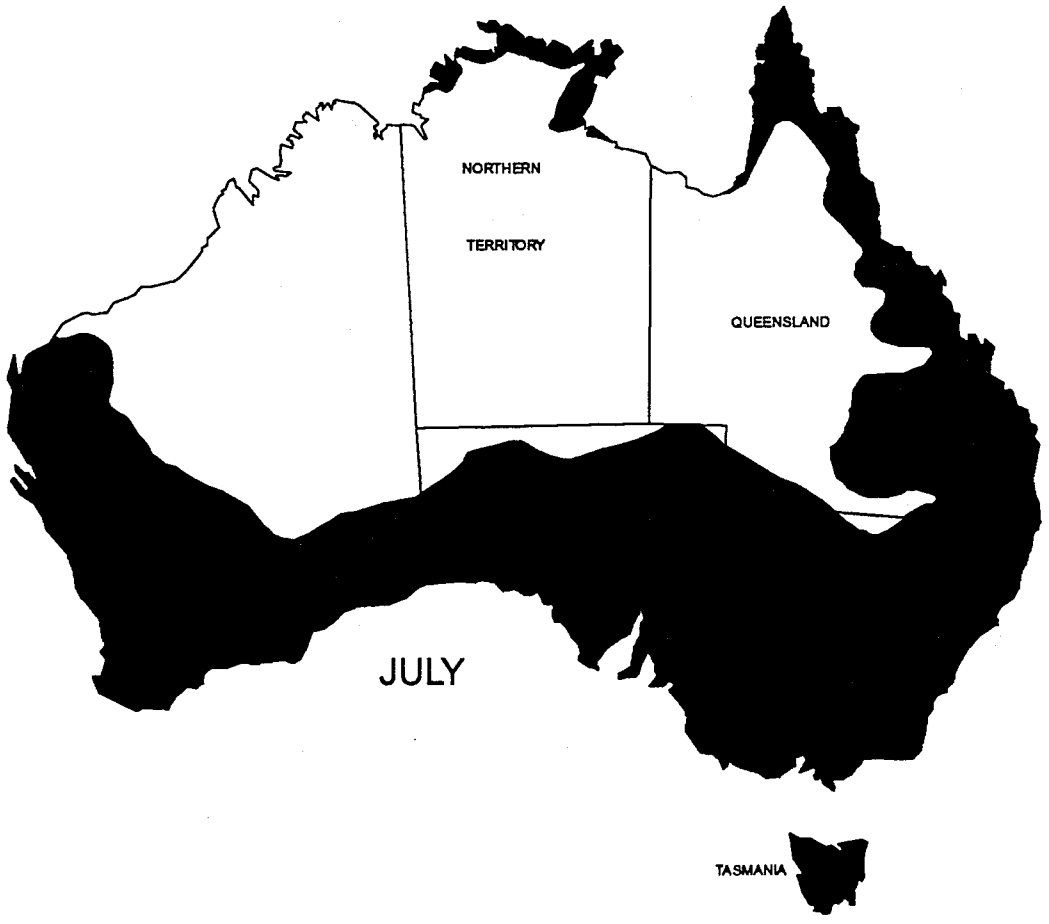
(a). Airborne Spread

There are 2 components to be considered in airborne spread of FMDV, close contact and distant spread. The most common method of contact spread of FMDV between animals is likely to be by inhalation of infective aerosols released in the vicinity of infected animals. Since the 1967/68 FMD outbreak in the United Kingdom it has been recognised that FMDV can be spread over great distances by wind, and numerous research projects have been directed toward the study of this aspect of FMD epidemiology. Most of the research on airborne spread of FMD has been conducted in the United Kingdom where the climatic conditions may not be representative of Australian conditions. In addition, it is believed by some authors that the strain of FMDV involved in this outbreak was unusual and that it had an increased ability to survive in aerosols. A very limited analysis of the relative humidity in Melbourne during the 1993 summer period showed that the average relative humidity at 0300 hours was 73% compared to 90% which was reported during a summer in England by Gloster *et al.* (1981). For 0900 hours the average relative humidity was 67% in Melbourne compared to 75% in Britain; at 1500 hours the Melbourne average relative humidity was 48% compared to 60%; and at 2100 hours the average relative humidity in Melbourne was 62% compared to 77%.

The transmission of FMDV by aerosols between animals over short distances is probably influenced by relative humidity, presence of sunlight and temperature. Of these, the influence of relative humidity is best characterised with a relative humidity of 60% being considered the critical parameter. It is probable that in many parts of Australia the relative humidity for the greater part of a 24 hour period might be lower than 60%. The Australian Yearbook provides charts of relative humidities for 0900 hours throughout Australia. This reading is a general estimator of the average daily relative humidity and Figure 2, adapted from the Yearbook charts, shows the areas where the average daily relative humidity is greater than 60% for January and July. The other factors which influence survival of FMDV in aerosols are less well studied.

While there are a number of anecdotal accounts of the effect of temperature on virus survival and spread, there appears to be no good evidence to show whether temperatures higher than 27°C are detrimental to survival of FMDV in aerosols. Donaldson and Ferris (1975) failed to demonstrate any effect of temperature, to a maximum of 27°C, and strong sunlight on the survival of FMDV deposited from aerosols onto spider microthreads. However, Rumney (1986) noted that survival of FMDV in ultraviolet light depends to some extent on the nature of the aerosol.

Figure 2. Zones where average Relative Humidity is > 60% at 9 am



Ultraviolet light is detrimental to the survival of FMDV and it could be expected that, under Australian conditions, survival of FMDV in aerosols might be somewhat less than that recorded in the United Kingdom. Australian climatic conditions may be sufficient to severely reduce the spread of FMDV between animals, especially when they are extensively grazed, and therefore a much slower rate of spread might occur. Donaldson (1979) cites observations made by Schang (1960) who claimed that field observations showed that the spread of FMD could be controlled by separating groups of animals by fences 10 metres apart. These observations were made in Argentina but no indication is given of the climatic conditions.

Long distance spread of FMDV in aerosols is the result of wind action. The determinant climatic factors in wind-borne spread have been identified as: wind direction; wind spread; wind veer; atmospheric stability and humidity. FMDV must remain in the atmosphere in sufficiently high concentration while being transported by wind to infect other susceptible stock. The potential effects of humidity, temperature and sunlight have previously been discussed. As noted, the effects of these parameters on virus survival under Australian conditions have not been determined but it is likely that their effects will be more severe than under the conditions existing in the United Kingdom. Wind speed will affect the concentration of virus downwind. The greater the wind speed the greater the horizontal dispersal of virus particles, i.e. the virus will be diluted. For optimum dispersion of FMDV at concentrations sufficient to cause infection, light winds are required. Atmospheric stability is also an important component of wind-borne spread. An unstable atmosphere will result in considerable dilution of virus particles as they will be distributed over very large vertical distances. With an unstable atmosphere, which occurs when ground temperatures are higher than air temperatures, there is increased turbulence and the height of the boundary layer is increased. This results in the virus particles, excreted by infected animals, being mixed in large volumes of air. Unstable conditions often occur during daylight hours over land masses. In contrast, stable atmospheric conditions result in a reduced height of the boundary layer and less turbulence so that virus aerosols remain concentrated in the small volume of air. Stable atmospheric conditions are produced by warm air flowing over cooler surfaces. When this occurs there is usually an increase in the relative humidity, a greater chance of mist and poor vertical mixing of air. Neutral stable atmospheric conditions occur when there is no heat flux between air and land and there is an increasing windspeed with vertical height. Smith (1983) provided a nomogram for predicting the height of the boundary layer when there were neutral stable atmospheric conditions. As an example, at 1400 hours in summer with 4 octas cloud cover and a wind speed of 3 m/sec at 10 metres, the height of the boundary layer will 1080 metres. In winter with the same conditions the height of the boundary layer would be about 600 metres. Optimal dispersion of FMDV by wind occurs when there is a low boundary layer and light winds. To accomplish a low boundary layer there needs

to be overcast conditions and temperature profile showing a rise in air temperature with height. Typical boundary heights would range from a few hundred metres to more than 2000 metres during daylight hours, and from tens of metres to about 400 metres at night.

Under English and European climatic conditions the farthest distance over which airborne spread of FMDV is believed to have occurred is around 250 km for spread over the sea and 60 km for spread over the land (Donaldson, 1986).

The strain of FMDV appears to affect both its production and survival in aerosols. Viruses isolated from European FMD outbreaks produced more infective aerosols than those isolated from outbreaks in the Near East (Donaldson *et al.*, 1970 and Sellers *et al.*, 1971). It was postulated that this may reflect selection for ability to spread by the airborne route or by animal movement depending on the climatic conditions. Rumney (1986) noted that virus strains originating from drier climates survived better in aerosols than those from more temperate climates. While Donaldson (1972) noted that virus strains which were less stable in aerosols appeared to be those which are excreted in greater amounts by infected animals, and conversely the more stable strains are those excreted in the lowest amounts. Donaldson *et al.* (1970) noted, however, that the amount of virus released by a species of animal is only one of the factors influencing the degree of airborne spread after an initial outbreak. The numbers of animals involved, the period before the disease is reported as well as the topography of the area, the livestock density and the climatic conditions are all important.

The species of animal is also important in the spread of FMDV by aerosols as pigs produce about 30 times more virus than either sheep or cattle from their respiratory tract. The presence of pig farms has been observed to play a role in the epidemiology of FMD. In the United Kingdom FMD outbreak, it was observed that when all pig farms in an area had been infected, and the animals on them slaughtered, the spread of FMD was much reduced.

There has only been a superficial examination of the possibility of wind-borne spread of FMDV under Australian conditions. An examination of the relative humidity profiles noted above suggests that virus survival in aerosols may be much less under Australian conditions than in English and European climates. It should be possible to use weather records to examine the average atmospheric stability and light wind patterns to predict the possibility of wind-borne spread of FMD in Australia. The importance of climatic factors in the epidemiology of FMD can be seen in the 1975 outbreak of FMD on Malta when only 37% of the outbreaks could be attributed to the airborne route whereas approximately 80% of outbreaks in the 1967/68 United

Kingdom outbreak could be attributed to this cause (Sellers *et al.*, 1981). The authors noted that in the Malta outbreak there were several occasions when any virus in aerosols would have been exposed to periods of low relative humidity and that spread of the disease halted after those periods.

This aspect requires further study in Australia with a retrospective study of climatic data to determine, for representative areas of Australia, the probability of sustained wind-borne spread of FMDV during each month of the year. The probability of wind-borne spread of FMDV will influence the planning and utilisation of resources for any FMD outbreak in Australia.

(b). Artificial Insemination

Cottral *et al.* (1968) artificially inseminated 16 heifers with semen from bulls that had been infected with FMDV by intradermal inoculation into the tongue. The semen was demonstrated to contain FMDV by animal inoculation. Within 2 to 16 days after insemination, 5 of the heifers had clinical signs and lesions of FMD. Four of these were in oestrus at the time of insemination. The remainder of the heifers were not in oestrus. Two of the heifers which did not show clinical signs or lesions developed an immune response to FMDV after insemination. Animals which developed clinical signs and/or lesions received estimated doses of FMDV in semen between 2.3 and 2500 x 10^3 MID_{50}. A further 10 heifers were inseminated with infected semen in tryptose phosphate broth or egg yolk extender. Five of these heifers developed clinical signs and lesions of FMD after an incubation period of 4 to 5 days.

Vaccinated donor bulls, exposed to FMDV, may shed virus from the skin of the prepuce with possible contamination of semen (Philpott, 1993).

Artificial insemination has been shown to be one of the ways by which FMDV may be spread. Although it is unlikely that semen would be collected from animals showing clinical signs of FMD, there is the possibility of semen collected during the incubation period of the disease could spread FMD. However, as most semen used in Australia is stored before use, the possibility of widespread dissemination of FMD through artificial insemination is probably not high.

It has been recommended (Philpott,1993) that, to reduce the risk of importing FMDV in the semen of bulls or boars, collections should take place at least 30 days after vaccination to ensure that the vaccine used was not contaminated with virulent virus; that probang samples as well as semen are taken at the time of collection and cultured

for the presence of FMDV; that semen should be held for a least 30 days before distribution to ensure that no clinical signs of disease have occurred since collection. No collections should be made from bulls in areas where FMD has occurred in the previous 3 months.

It is unlikely that cattle semen used for artificial insemination in Australia would spread FMDV as most semen is collected from bulls held permanently at artificial breeding centres. The semen is processed, checked for sterility and usually held for prolonged periods before use. There is greater potential for the spread of FMD by semen in pigs because boar semen is used for artificial insemination within a few days of collection. Ram semen may be used immediately for artificial insemination, particularly in an embryo transfer program, or may be stored for prolonged periods before use. In the former case, and with pigs, it might be possible for semen, collected from an animal incubating FMD, to spread FMD. It is most likley that animals in a single flock or herd will be infected.

(c). Embryo Transfer

Mebus and Singh (1991) collected 436 embryos from 30 FMD viraemic cattle which had been infected by intravenous inoculation. Collection of embryos took place approximately 22 hours after inoculation to maximise the exposure of the embryos to viraemia while minimising any effect of elevated body temperature. Only 8 of the 30 viraemic donors had FMDV in uterine flush fluids or uterine sediment with virus titres of between $10^{1.7}$ and $10^{2.18}$ pfu/ml. The embryos were washed and assayed by tissue culture, intradermolingual inoculation of susceptible steers or transferred to recipient cows. FMDV was not detected in 287 embryos by tissue culture or intradermolingual inoculation. Only 22 of 112 recipients became pregnant. None of the recipients or calves born from the embryo transfers of 149 embryos showed evidence of FMD and all remained FMD-seronegative.

After washing, no FMDV was isolated from 169 zona-pellucida intact cattle embryos incubated with FMDV (Singh *et al.*, 1986). Virus was isolated from washes of the embryos up to the 4th wash and occasionally after the 4th wash but not from 10th wash. FMDV was isolated from 14 of 22 hatched cattle embryos and virus was detected up to the 6th wash. Of 100 pig embryos exposed to FMD only 2 were positive for FMDV after washing. A small number of zona-pellucida intact pig embryos retained FMD after washing. This may have been due to defects in the zona pellucida.

McVicar *et al.* (1986) collected 48 embryos from cattle in the acute stages of FMD and was not able to detect FMDV by tissue culture or by animal inoculation after the embryos were washed. Forty-two embryos were also collected from cattle 21 days after infection with FMD and were found to have no detectable FMDV after washing.

The Research Committee of the International Embryo Transfer Society (IETS) Import/Export Committee has classified FMD in cattle as a Category 1 disease for embryo transfer (anon, 1992). This category is for diseases or disease agents for which sufficient evidence has been accrued to show that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer. FMD in sheep, goats and pigs is considered to be a Category 3 disease. This is a disease or disease agent for which preliminary evidence indicates that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer but for which additional *in vitro* and *in-vivo* experimental data are required to substantiate the preliminary findings.

Although various workers have shown that uterine fluids and embryos from infected animals may be contaminated with FMDV it is unlikely that embryo transfer, using adequately washed embryos, will be important in the spread of FMD by the international movement of genetic material. Embryo transfer could be a method of spread of FMDV in Australia since extensive washing of embryos is unlikely, unless specified by the client. Thus embryos could be collected from animals incubating FMD and then be transported to other farms and implanted into recipients, infecting them with FMDV.

(d). Animal Movement

In an analysis of the Northumberland FMD outbreak in the United Kingdom during 1966/67 it was considered that on 9 of 32 farms affected, the movement of cattle, sheep, people or vehicles were involved in the spread of FMD (Sellers and Gloster, 1980). The rest of the farm outbreaks were considered due to airborne spread.

Several countries have reported that movement of animals has been associated with the spread of FMD. In the Sudan, where FMD is endemic, Eisa and Rweyemamu (1977) reported that the disease was disseminated principally by animal movement along traditional routes. Casas-Olascoaga (1978) noted that cattle movement control under South American conditions continued to be a weak point in the fight against FMD. The continuous movement of large numbers of cattle, especially, in some regions, young animals with low population immunity, represented a permanent risk. Sarma *et al.* (1985) reported that in northern India the maximum number of FMD outbreaks

occurred mainly in the plains areas and that restricted movement of animals and man probably accounted for the low incidence of FMD in the hill states of the region. Also in India, Ahuja *et al.* (1981) reported that two-thirds of the 1268 outbreaks of FMD reported to the Regional Centre at Hissar (northern India) between 1972 and 1978 occurred between 1976 and 1978 when floods caused widespread movement of livestock. The same authors considered that spread of FMD in northern India was enhanced by cattle fairs, common grazing, seasonal migration and movement of technical personnel working with diseased animals. Joseph (1986) reported that the most important method of spread of FMD in the ASEAN region was the movement of infected animals. This author considered that airborne spread was less important and that mechanical transmission was less likely to occur in tropical climates because of poorer virus survival in hot weather. In Sri Lanka, the spread of FMD was found to be mainly by movement of herds within villages, cart traffic between villages and transportation of stock from diseased areas to disease free areas for slaughter (Fernando, 1985). Rweyemamu *et al.* (1982) considered that animal movement was the principal mode of spread of FMD in the areas of South America, Africa and Asia where the disease is endemic.

Hugh-Jones *et al.* (1983) analysed animal movements in Brazil in an attempt to predict the spatial incidence of FMD. The amount of movement of breeding animals between herds in a region appeared to be important in the spread of FMD.

Buik (1979) has analysed cattle movements in Australia and noted that they can be used to identify key points in disease transmission such as major source regions of non-slaughter cattle, major recipient regions and the principal sale/purchase points through which cattle move. Calculation of a contact index is suggested which could provide a weighting, in terms of probability of being infected, for all properties within a region. Recently, Sanson *et al.* (1993) studied the movement of animals and animal products in a limited area of New Zealand to determine the size of control areas needed to contain the spread of FMD. With the concept of zoning which requires that free zones must be totally free of a disease, it will be necessary to have larger control areas to ensure that disease is not present in other areas of the country. Just how large these zones will need to be will depend upon the magnitude of animal movements. For example, in the New Zealand analysis, a control zone of at least 100 kilometres radius would have been necessary to contain 95% of animal and product movements.

Similar analyses are needed for Australian livestock production areas to determine the potential role of animal movement in the spread of FMD and the potential for zoning in the event of an FMD outbreak.

(e). Live vectors

(i). Man

The possibility that man may be involved in the spread of FMD has been studied by several workers. Close contact with infected animals appears to be important.

Hyslop (1973) noted that FMD may produce painful, but transient, clinical signs in man. Exposure through inhalation of virus resulted in virus being recovered from the upper respiratory tract. Sellers *et al.* (1970) using samples taken from persons who had been in contact with animals infected with FMDV showed that virus could be recovered from the nose, throat, saliva and from air expelled during coughing, sneezing, talking and breathing. The highest recovery of airborne virus resulted from contact with infected pigs. In the majority of subjects, there was a $10^{1.5}$ fall in virus titre 3.5 hours after exposure. Nose blowing or washing the nostrils did not remove virus completely, nor were cloth or industrial masks completely effective in preventing inhalation of virus. FMDV was transmitted from infected subjects to another person on one occasion. No clinical cases resulted, nor was there a rise in antibody titres although virus was recovered from nasal swabs. The virus titres in nasal swabs were $10^{3.4}$ $TCID_{50}$ per swab for persons operating air samplers in the surrounds of pigs and $10^{4.1}$ $TCID_{50}$ per swab for persons examining pigs infected with FMDV $O_{1\ BFS\ 1860}$ subtype. For infections with A_5 FMDV, the titres were $10^{3.3}$ $TCID_{50}$ per swab and $10^{3.7}$ $TCID_{50}$ per swab for collectors and examiners respectively. With sheep infected with FMDV subtype O_2 the titres were $10^{1.2}$ $TCID_{50}$ per swab for collectors and $10^{1.6}$ $TCID_{50}$ per swab for examiners. The corresponding titres for sheep infected with FMDV type C were $10^{1.4}$ $TCID_{50}$ per swab and $10^{3.9}$ $TCID_{50}$ per swab. With cattle infected with subtype A_5 the titres were $10^{0.9}$ $TCID_{50}$ per swab and $10^{1.5}$ $TCID_{50}$ per swab for collectors and examiners respectively. For cattle infected with subtype C the titres were $10^{1.0}$ $TCID_{50}$ per swab and $10^{2.3}$ $TCID_{50}$ per swab.

Sellers *et al.* (1971) reported that FMDV was excreted by persons after handling infected pigs in amounts ranging from $10^{0.36}$ to $10^{3.25}$ $TCID_{50}$ by sneezing, snorting, coughing and breathing. Infection of a steer occurred when it was exposed to breathing, sneezing, snorting and coughing by these persons who concentrated their efforts at the muzzle of the cattle. Cattle were only exposed to about 2.5 minutes of aerosol generated by the persons. It was estimated that the infected steer received a dose of about $10^{2.0}$ $TCID_{50}$. FMDV disappeared from the nose of the subjects after 24 hours.

Information provided from this research suggests that man is unlikely to be an important vector in the spread of FMD in Australia.

(ii). cats and dogs

Dogs and cats can be infected with FMDV experimentally. For example, cats were able to be infected with FMDV when they were kept in contact with infected cats or fed milk contaminated with FMDV (anon, 1931). Saliva from an infected cat was found to remain infective for 13 days. It was also reported that both dogs and cats could be infected with FMDV by inoculation and that the disease could be passed from one to another of the same species by contact infection (anon, 1937). However, outbreaks of FMD in dogs and cats have never been reported in field outbreaks (Hyslop, 1969) and the role of domestic pets in the epidemiology of FMD is probably unimportant. It is possible that dogs could carry bones and meat from infected animals on an infected premise to farms where there would be pigs, the only species likely to be infected, by ingestion, from such a source. Alternatively, it has been suggested that cattle sniffing infected objects may inhale sufficient virus to become infected (Sellers *et al.*, 1971). Circumstantial evidence of transmission of FMD by scavenging dogs has been reported (Pereira, 1978).

The role of the fox in the epidemiology is likely to be similar to the domestic dog, i.e. it would be likely to spread carrion between farms.

The role of dogs, cats and foxes in the spread of FMD in Australia is likely to be small if carcasses are disposed of in a manner which ensures that these species do not scavenge on them.

(iii). rodents and small mammals

A variety of rodents and small mammals have been infected experimentally with FMDV. Rats were infected by feeding infected meat and by contact with infected rats (anon, 1931). The same workers failed to produce FMD in house mice but could infect white mice. Clinical lesions were only seen in Brown rats after inoculation and not after infection via feed or water or by contact (Capel-Edwards, 1970). Lesions were most severe when the virus had been passaged in guinea pigs. FMDV was detected for several months in the faeces of inoculated, virus-fed and in-contact rats. Tissue recovery studies showed virus in several tissues in inoculated rats but only in the alimentary tract of the orally-infected and in-contact rats. The virus appeared to remain as a commensal in the alimentary tract without stimulating antibody in the rat and continued to be excreted in small amounts over a long period. Virus titres ranged from 100 to 1000 $TCID_{50}$ per 3 ml of faecal suspension. No evidence was found of infection following feeding of mice with infective material from animals with FMD

(anon, 1925). Sellers (1971) calculated that an animal would have to ingest the faeces of 160 infected rats to become infected. If the virus could survive drying then infection from inhalation of dust particles, contaminated by rat faeces or urine would be possible. Clinical FMD has been observed in rats on infected premises and it possible that disease control operations on infected premises could drive rats away to other farms where they could spread the disease. However, in the 1967/68 U.K. outbreak that there was no difference in the number of subsequent outbreaks about rat infested farms nor was there any apparent benefit to be gained, in relation to spread of FMD, by killing rats (Hugh-Jones, 1970). In India, Odend'hal (1979) failed to detect infection in 47 rats trapped on or around FMD outbreaks in a small village in India.

The role of rats and mice in the spread of FMD is probably of little importance if adequate vermin control is instituted during FMD outbreaks.

Hedgehogs have been observed to be infected with FMDV on infected premises in the United Kingdom (anon, 1937). They have been found to be highly susceptible to infection with type O virus and can spread FMD to other hedgehogs by natural means (anon, 1931) and to cattle by contact (anon, 1937). It has also been demonstrated that hedgehogs can be infected by contact with infected cattle. The relevance of this finding to the Australian situation is not clear except that it is possible that small mammals in Australia may be similarly infected. To be important in the spread of FMD such animals would need to have intimate contact with domestic stock.

Wild rabbits were not readily infected with type O virus in early work (anon, 1931). It is unlikely that wild rabbits would be involved in the spread of FMD in Australia.

Experimental infection has been reported in chinchilla, armadillos, nutria, coypu, Indian squirrels, grey squirrels, water voles and moles (Capel-Edwards, 1971; Tewari *et al.*, 1976). While there is no direct equivalent of any of these species present in Australia the findings highlight the need to be aware that small mammals may play a minor role in the epidemiology of FMD. Snowdon (1968) demonstrated that water rats (*Hydromys chrysogaster*) can be infected with FMD but the susceptibility of most of the small native Australian mammals has not been studied. A review of FMDV infection in small mammals has been provided by (Capel-Edwards, 1971).

The potential role of small native mammals in the spread of FMD in Australia is difficult to assess. Based on experiences with other small mammals their role is likely to be minor.

(iv). feral animals

Pigs

Feral pigs may play a potentially important role in the epidemiology of FMD in Australia. Hone and Pech (1990) have estimated the probability of detecting just one case of FMD in feral pig populations in Australia with the current opportunistic surveillance, i.e. hunting, is less than 0.0015. They estimate that between 28 and 3077 cases of FMD would occur in a feral pig population before an outbreak was detected. They also estimated that if a feral pig population occupied 100 km^2 at a population density of 15/km^2, then it would take between 23 and 358 days to detect the occurrence of an FMD outbreak. This has major implications for the spread of FMD if a primary outbreak occurred in a feral pig population. It is likely that the presence of FMD would only be detected by the occurrence of infection in domestic stock populations in proximity to the feral pig population. The presence of FMD in large numbers of pigs is likely to provide a large aerosol source of virus. Depending on the climatic conditions the spread of the aerosol virus may only be over a limited distance. If under Australian conditions the distance over which FMDV may be spread by airborne transmission is only of the order of a few hundred metres, the presence of FMD may only be detected if domestic stock have a close association with feral pigs such as at waterholes or other areas of congregation. The population density of feral pigs will also influence the ability of FMD to persist in such populations. Pech and Hone (1988) estimate that if the population density of feral pigs was 3 pigs/km^2 then FMD may disappear about 60 days after initial infection. Thus FMDV infection could occur in feral pig populations and disappear again before it was detected by hunting or other activities.

Besides providing a problem in the detection of FMD, feral pig populations provide major problems for disease control. For example, Saunders and Bryant (1988) after a trial eradication program concluded that eradication of FMD from even a small population of feral pigs occupying an area of 120 km^2 might be impossible. Pech and Hone (1988) suggested that FMD might spread at a rate of 2 km/day in western N.S.W. feral pig populations and that if it took 29 or 49 days to detect the presence of the disease the area involved in the outbreak might cover 10,000 or 30,000 km^2 respectively.

Outbreaks of FMD in feral pig populations in Australia would present major problems for the control of FMD with eradication of disease in such populations being almost impossible. Techniques for detection of FMD outbreaks in feral pig populations and ways to reduce contact between feral pig populations and domestic stock need to be reviewed and developed.

Camels

FMDV infection has been reported in camels in Egypt (Moussa *et al.*, 1987). However, in a survey of oesophago-pharyngeal fluid from 454 camels, in areas where FMD had been reported, no evidence of carrier camels was found (Tantawi *et al.*, 1984). The role of this species in the spread of FMDV in Australia is likely to be limited given the relatively small populations of feral camels and their limited contact with domestic stock.

Buffalo

A definite role for the African Buffalo in the epidemiology of FMD appears to have been established. The situation with the Indian buffalo, the species which is present in Australia, is less clear.

Hedger *et al.* (1973) demonstrated that there is a persistent high prevalence (up to 60 per cent) of carriers of serotypes SAT 1, SAT 2 and SAT 3 in African buffalo populations. The virus appears to be maintained by cycling within these herds. In southern Africa, clinical disease has not been observed in surveys of buffalo populations, but the virus has been isolated from the throats of apparently healthy animals in titres up to $10^{4.2}$ $TCID_{50}$ per ml (Anderson *et al.*, 1979). These authors exposed susceptible cattle for periods of up to 5 months to buffalo infected with SAT 2 FMDV previously and to carrier buffalo that had been infected with SAT 1 FMDV for 5 months without developing infection. However, Young *et al.* (1972) observed typical mouth and foot lesions from which SAT 2 virus was isolated in African buffalo during an outbreak in the Kruger National Park. Animals with distinguishable lesions ranged from 25 to 43% when 1285 carcasses were examined. Natural transmission of the disease from infected buffalo to cattle occurred when 3 steers in a pen adjoining buffalo in the acute stages of FMD became infected.

Condy and Hedger (1974) reported that African buffalo may carry both SAT 1 and SAT 2 virus for periods as long as 28 months. Bengis *et al.* (1987) noted that transmission of FMD from carrier buffalo to susceptible cattle had been shown to be an inefficient and rare event. Actively, usually asymptomatic, infected buffalo, however, following primary exposure to a specific SAT virus type, shed substantial amounts of virus in their saliva, nasal secretions and respiratory aerosols. During this period, which may have lasted up to 4 weeks after infection, they were capable of infecting cattle in close contact. Condy *et al.* (1985) demonstrated that transmission of SAT 3 from carrier buffalo to cattle could occur. In addition, SAT 3 FMDV was shown to persist in an individual African buffalo for up to at least 5 years.

However, Anderson (1986) collected virus isolates in Africa from clinically infected cattle or convalescent carrier animals and compared them with viruses isolated at the same time from buffalo in the same area, using oligonucleotide mapping. There was no similarity between any of the viruses indicating that the different populations of virus were unique to each species and that no interspecies transmission had occurred.

The epidemiology of FMD in Indian buffalo has not been studied as extensively as in the African species. Sharma *et al.* (1985) reported that FMD occurred in Murrah buffaloes in Vietnam, however, only mouth lesions were seen and the feet were never involved. FMD is reported to be widespread in Indian Buffalo throughout India (Dutta *et al.*, 1983). Of 615 FMD outbreaks recorded in one area, 70 involved buffalo but in 51 of these outbreaks cattle and pigs were also involved. In 12 outbreaks, buffalo acquired infection from diseased cattle and transmitted it to in-contact goats. Sheep were infected in 3 outbreaks and in another 3 outbreaks in buffalo, the disease was simultaneously observed in pigs goats and cattle. In Brazil, it has been reported by Samara and Pinto (1983) that, after an FMD outbreak in cattle, 23% of 379 water buffalo surveyed had virus infection associated (VIA) antibody, and that in addition, virus was isolated from the pharynx of some animals. Another survey found 52% of 157 water buffalo sera with FMD VIA antibody. Transmission of infection from cattle and pigs to water buffalo occurred during an outbreak with FMDV being isolated from the pharynx of buffalo 21-30 days after the occurrence of FMD on a farm. No lesions were observed in the buffalo but generalised lesions were evident in the cattle and pigs.

It would appear that buffalo populations in northern Australia could play a role in the epidemiology of FMDV similar to that seen in Africa. In this situation, the virus appears to persist in feral buffalo populations producing few, if any, clinical signs. When conditions such as drought cause the co-mingling of cattle and feral buffalo at watering points and other locations the opportunity for spread of FMD is heightened. However, the culling of feral water buffalo populations in northern Australia in recent years as part of the BTEC program has probably reduced the risk of infection being transmitted between this species and domestic cattle.

(v). wild animals

Australian Fauna

Snowdon (1968) examined the susceptibility of Australian fauna including red kangaroo, grey kangaroo, tree kangaroo, wombat, brush tail possum, long-nosed bandicoot, potoroo, water rat, echidna, brown marsupial mouse, Bennett's wallabies and wild rabbits to infection with FMDV. All animals were inoculated initially, but red kangaroos and wombats were used in contact experiments with cattle. FMDV multiplied in most of the species but clinical lesions were only seen in the tree kangaroo, 4 water rats and 2 echidnas. Suspicious lesions were seen in a red kangaroo. It was not possible to demonstrate a carrier state in red kangaroos. When red kangaroos and wombats were closely confined with cattle it was found that:

(1). a proportion of red kangaroos infected by intramuscular inoculation were capable of infecting cattle;

(2). infected cattle were capable of infecting red kangaroos and wombats; and

(3). after being confined with infected cattle for 48 hours, red kangaroos subsequently did not infect susceptible cattle when confined with them.

It was not possible to demonstrate spread of infection from infected red kangaroos to previously unexposed red kangaroos, or persistence of FMDV in red kangaroos.

Snowdon's work indicated that there is a wide range of susceptibility to infection with FMDV between Australian fauna. The assessment of the possibility of transmission between domestic animals and native fauna was primarily based on the excretion of virus in lesions. It was believed at the time of the work that lesion fluids were the most likely source of virus for FMDV transmission. Later work suggests that the respiratory route is the primary portal of entry of FMDV. While there is, in general, little close contact between native Australian fauna and domestic stock there could arise in certain locations sufficient concentrations of native fauna and domestic stock to allow aerosol transmission of FMD. This is an aspect that probably needs to be further explored. Snowdon's work only had one experiment where native fauna and domestic animals were run in close contact.

Murray and Snowdon (1976) report on possibility of spread in Australia of FMD by feral cattle, feral buffalo, feral pigs, feral goats, introduced deer, feral camels with distribution maps of these species in Australia.

Non-Australian Fauna

A variety of species, of which deer and llama are probably the most relevant to the Australian situation, have been shown to become naturally infected with FMDV.

White-tailed deer were shown to be susceptible to infection with FMDV type O_1 (McVicar *et al.*, 1974). The disease was transmitted by contact from deer to other deer, from deer to cattle, and from cattle to deer. One deer remained a carrier for 11 weeks after infection. Forman *et al.* (1974) and Gibbs *et al.* (1975a, 1975b) infected red, fallow, roe, sika and muntjac deer by exposing them to cattle with FMD. Each species transmitted disease to its own species and to cattle and sheep. The fallow and sika deer carried virus in the pharynx beyond 28 days after exposure. Some red deer also became carriers. In some of the red and fallow deer, no immune response was detectable although the animals developed a viraemia and/or pharyngeal infection was demonstrated. The disease was severe in the muntjac deer and by day 10 post-exposure, 6 of 9 deer had died. Virus was excreted as an aerosol from deer that did not develop clinical disease. Virus excretion in aerosols were similar in the deer, sheep and cattle.

Wild deer populations in Australia tend to be fairly small. Their role in the epidemiology of FMD in Australia is likely to be minor, similar to the situation in Europe where, in spite of their widespread distribution, deer have played an insignificant role in the spread of FMDV (Murray and Snowdon, 1976).

Lubroth *et al.* (1990) reported infection of 2 llamas by intradermolingual inoculation and 2 by exposure to a calf affected with FMDV type A_{24}. Two other llamas exposed by contact did not become infected. FMDV was shown to be transmitted from either cattle to llamas, llamas to swine, and llamas to llamas. FMDV was not isolated from the llamas beyond 1 week post-inoculation or post-contact. Lubroth and Yedloutschnig (1987) showed that llamas were susceptible to FMDV by direct inoculation and by contact with infected pigs, cattle or llamas.

(vi). birds

The role of birds in the spread of FMD is controversial. Before wind-borne spread of aerosols was established as an important method of FMD spread they were considered to be the most likely means of spreading the disease over long distances. There are no reports of the isolation of FMDV from birds captured in the wild (Hyslop, 1969). Experimental evidence suggests that birds could act as mechanical carriers of FMDV. For example, 85 Jackdaws captured in an area free from FMD were infected with a

lapinized A_{22} FMDV by the application of a viral suspension to skin and by oral dosing (Obidor and Pankov, 1975). The virus persisted on the beak, feathers and feet for 5 days, in liver and kidney for three days and in the heart and gastrointestinal system for two days. Infection resulted in catarrhal inflammation of digestive tract mucosa, which lasted 3-4 days. Sviridov *et al.* (1972) infected Jackdaws (*Corvus monedula*), starlings (*Sturnus vulgaris*) and sparrows (*Passer domesticus*) by oral doses of a lapinized type A_{22} virus. Some birds were infected by spreading the virus with a brush onto the feathers. The virus remained viable in the stomach, intestine, heart muscle and liver and on the feathers for 2-5 days, without being affected by fluctuations of ambient temperature. Svidorov *et al.* (1974) further reported that they were able to demonstrate the transmission of FMD virus type A_{22} from infected to susceptible cattle by contact with starlings. The starlings remained infective for susceptible cattle for 2-4 days after contact with infected cattle, but none of them transmitted virus to susceptible cattle after 8 days. It was considered that if starlings are carriers, they are purely mechanical ones. No evidence of multiplication of FMDV in various avian species, including sea gulls, was found in early British studies (anon, 1937). Carrion-eating birds, like the crow, might be able to spread infected parts of carcases over some distance.

A consideration of these findings suggests that birds are likely to be of minor importance in the spread of FMD in Australia.

(vii). arthropods

Bachrach (1968) noted that no biological insect vector has been identified as being important in the spread of FMD and that the likelihood that such transmission would occur is slight because of FMDV does not have the properties of an arthropod-borne virus. The only role for insects might be mechanical transmission. Long residence and transovarial passage of FMDV have been observed in ticks which have fed on infected animals but there are no reports of ticks transmitting FMDV to animals. In an experiment where *Rhipicephalus zambeziensis* ticks were allowed to feed on viraemic cattle, with virus titres in their blood ranging from 10^1 to 10^4 $TCID_{50}$ per ml, van Vuuren *et al* (1993) found that FMDV survived for at least 3 days in engorged nymphs, but less than 7 days after engorgement. Virus titres ranged from $10^{1.0}$ to $10^{1.2}$ $TCID_{50}$ per tick. The potential for ticks to transmit FMD was examined by feeding pools of 100 newly moulted ticks on susceptible cattle. The ticks were fed on the cattle 30 days after they had engorged and subsequently detached and most were found to have fed when examined 1 week later. No clinical or serological evidence of FMD infection was found in the cattle. *Boophilus annulatus* ticks collected from cattle in the febrile stage of FMD contained FMDV (anon, 1937).

Thomson *et al.* (1988) reported failure to transmit FMDV by biting insects. Newly emerged arthropod adults were viraemic with SAT 1 virus but susceptible cattle failed to develop FMDV after being housed with the flies for 48 hours. No serum antibody was found in the cattle when they were tested 3 weeks after exposure. FMDV was found to remain on the bodies of flies (Musca domestica) for 48 hours and for 18 hours in their digestive tract (anon, 1937). Attempts to transmit FMDV between guinea pigs by Cimex bugs which were allowed to feed on infected guinea pigs, and then to feed on susceptible guinea pigs, failed and even when the bugs were crushed and injected into susceptible guinea pigs no evidence of infection was observed (anon, 1925). Hyslop (1969) reported that FMDV had been detected in *Ixodes ricinus* and *Melophagus ovinus* which had been allowed to feed on infected animals.

The role of arthropods in the epidemiology of FMD remains uncertain with little information available on the possibility that biting insects could inject sufficient virus to initiate infection in a susceptible host.

(viii). earthworms

It has been reported (Hyslop, 1969) that earthworms maintained in infected soil for several days appeared to harbour FMDV because they produced infection in susceptible cattle when inoculated intradermally into the tongue epithelium after having been washed and ground before inoculation. Earlier work reported by Olitsky *et al.* (1928) suggested that earthworms did not harbour FMDV when they inhabit FMDV contaminated soil. There exists the possibility that earthworms might contain sufficient virus to produce infection if ingested by pigs. However, this aspect has not been investigated. Any role for earthworms in the spread of FMD would appear to be very small.

(f). Fomites

As noted in the section on virus survival it is difficult to assess the likelihood of fomites being involved in the spread of FMDV. There is little, if any, information on the titres of virus that might survive on fomites nor on the possibility of contact infection from fomites. In natural outbreaks, fomites do not appear to be important in the spread of FMD. For example, Hugh-Jones and Tinline (1976) reported that fomites were rarely observed to be the source of outbreaks during the 1967/68 United Kingdom FMD outbreak.

(i). vehicles

Vehicles could be a means of spread of FMD if there is contamination of surfaces with lesion material, faeces, saliva or other excretions. This could lead to infection of subsequent loads of stock if the vehicle is not adequately cleaned. This method of spread is most likely to be a problem in the early stages of an outbreak when animals incubating the disease may be carried. There has been at least one instance where a vehicle has been implicated in the spread of FMD. During the 1967/68 FMD outbreak in the United Kingdom a cattle truck was a common link between an affected pig farm and 2 cattle farms. The truck was used to carry cattle on successive days and cows came down with the disease 5 days later.

If fully enclosed vehicles are used to transport stock, there is the possibility of virus aerosols remaining in the air space. The section on buildings and Table 17 contains information on the persistence of FMDV in enclosed air spaces. These vehicles could not only retain airborne virus which could infect other stock carried in the vehicle but could also act as a source of aerosol virus when the enclosed space is ventilated.

It is possible that vehicles could play a major role in the spread of FMD in Australia because of the large amount of stock movements which are carried out by road transport. They would not only be the means of moving infected animals to non-infected areas but may also be a source of infection in themselves if they are not adequately cleaned. The lack of adequate truck washing facilities in many areas would increase the potential of vehicles to spread FMD.

(ii). utensils

The role of buckets, veterinary instruments, grooming equipment and other equipment is difficult to assess. There are a number of anecdotal accounts of infection resulting from transfer of FMDV on such objects. However, there is little information on the titre of virus that might survive on such objects and cause infection to susceptible stock. It is difficult to imagine their importance in the spread of FMDV in a situation such as a milking shed where aerosol virus is most likely to be the means of infection. Similarly, in any situation where there is a congregation of animals for some management procedure aerosol infection is most likely to be the major source of virus spread compared to mechanical or oral spread of the virus. The only situation where instruments might play a major role in the spread of FMD is where a mouth gag, or similar instrument, is used on an animal in the incubative stages of disease and is then used on a susceptible animal with damage to its tongue epithelium. Other situations where instruments might be involved in the spread of FMD are by injection

of virus. Amounts of virus needed to infect animals by the intramuscular, subcutaneous or intravenous route may be of the order of 10^2 $TCID_{50}$ (Sellers, 1971). Since titres of up to 10^5 $TCID_{50}$ may be found in skin (Gailiunas and Cottral, 1967) it is possible that an infective dose might be transferred via a syringe if it was not cleaned between its use on an infected animal and a susceptible one.

Animal equipment probably would have a minor role in the spread of FMD in Australia. However, the necessity for adequate hygiene precautions to be taken by people moving between farms needs to be reinforced. Farmers, veterinarians and other stock handlers need to be continually aware of the potential for the spread of infection on boots, clothing and equipment.

(iii). bedding, packing materials

As noted in the section on virus survival, FMDV has been reported to survive in hay stacks for 185-200 days in Russia. FMDV has also been reported to persist irregularly on straw for periods of between 5 days to 7 weeks (anon, 1928). Survival of FMDV on hay was reported to be at least 15 weeks provided it was stored under conditions of relative dryness and the absence of light (anon, 1931). Hay and straw soiled with FMDV-infected milk remained infective for 17 days. Once again, most of this work does not provide an indication of the virus titres expected on such materials. Infection following ingestion of such materials would be unlikely to be important in the spread of FMD. However, because of the relatively low doses of FMDV needed for infection by the respiratory route these materials could be important in the spread of FMD as animals may inhale dust and aerosols during their consumption. Infection by this route would require only a few virus particles to survive so that such materials could be involved in the spread of infection between farms.

(iv). water

FMDV has been found to survive for more than 20-50 days when the initial concentration of virus was 104 $TCID_{50}$ per ml of virus in both drinking and surface water held at 9°C (Mahnel *et al.*, 1977). Sellers (1971) calculated that a concentration of 32 $TCID_{50}$ per ml would be needed to produce infection of cattle by ingestion. The possibility of pollution of surface water by FMDV, mostly from affected animals buried after an outbreak, was examined by Lei (1985). The adsorption of the virus to various soil particles, and the rate of transport by surface water, were examined by using the analogy of liquid and solid phase chromatography. It was concluded that dissemination by water is unlikely but further research may be needed.

(v). fodder

The role of hay and straw has previously been discussed. Other foodstuffs may also play a part in the spread of FMD although oral infection is unlikely to be an important source of infection for the reasons discussed previously. FMDV has been reported to survive on bran for 20 weeks when it was stored under conditions of relative dryness and the absence of light.

(vi). buildings

Buildings have the potential to be involved in the spread of FMDV. Soiling of internal surfaces (floors, walls, pen railings, etc.,) with faeces, saliva and other secretions containing FMDV will provide foci of infection if susceptible animals are re-introduced to the building without adequate disinfection. Secondly, FMDV held in aerosols within a building, such as a stable or large piggery, may also serve as a source of infection for susceptible animals re-introduced to the building within a short time following the removal of infected animals.

Early work in England (anon, 1937) showed that susceptible cattle were not infected when they were placed in stalls vacated by highly infective cattle 3 or 4 days previously.

Sellers *et al.* (1971) used pigs and cattle held in loose-boxes and infected with either type $C_{NOVILLE}$ or $O_{1\ BFS\ 1860}$ FMDV to measure the amount of FMDV which remained in the air after the animals were killed or removed (Table 17). Killing or removal of pigs led to a reduction of 25-fold or more in virus recovered but virus persisted in the looseboxes for at least 24 hours although only to 1/5000 th of that originally present. Removal of cattle from the box did not lead to any significant fall in virus recovery. Their research showed that immediate slaughter of pigs brought about at least a 25-fold fall in the amount of FMDV present in air. These experiments were done in relative humidities of 90%. At lower values of relative humidity the amount of virus would decrease more rapidly, depending on virus type.

In Australia, aerosol contamination of buildings is unlikely to be important in the epidemiology of FMD because, except for pigs, few animals are housed. In addition, it is difficult to see any situation where animals would be immediately introduced into a building after infected animals had been removed. It is recommended in AUSVETPLAN that restocking of premises not be undertaken for a period of 6 weeks.

Table 17. Persistence of FMDV in air in buildings after removal of infected animals

(a). Type O_1 FMDV in pigs: (amount of virus collected over 30 minute period)

1	Box containing live pigs	$10^{6.4}$ $TCID_{50}$
	Same box 30 min. after pigs killed	$10^{4.8}$ $TCID_{50}$
2	Box which held pigs for 45 min.	$10^{7.2}$ $TCID_{50}$
	New box in which the killed pigs were placed	$10^{3.0}$ $TCID_{50}$
3	Box containing live pigs	$10^{5.5}$ $TCID_{50}$
	Same box 30 min after pigs removed	$10^{4.0}$ $TCID_{50}$
4	Box which held pigs for 45 min	$10^{5.5}$ $TCID_{50}$
	Same box 30 min after pigs removed	$10^{4.1}$ $TCID_{50}$

(b). Type C_{NOV} FMDV in pigs: (amount of virus collected over 30 minute period)

1	Box containing live pigs	$10^{7.2}$ $TCID_{50}$
	Same box 30 min. after pigs killed	$10^{4.8}$ $TCID_{50}$
2	Box which held pigs for 45 min.	$10^{6.2}$ $TCID_{50}$
	New box in which the killed pigs were placed	$10^{4.6}$ $TCID_{50}$
3	Box containing live pigs	$10^{6.6}$ $TCID_{50}$
	Same box 30 min after pigs removed	$10^{5.2}$ $TCID_{50}$

(c). Type O_1 FMDV in cattle: (amount of virus collected over 30 minute period)

1	Box containing live cattle	$10^{2.8}$ $TCID_{50}$
	Same box 30 min after cattle removed	$10^{2.4}$ $TCID_{50}$
2	Box containing live cattle	$10^{2.4}$ $TCID_{50}$
	Same box 30 min after cattle removed	$10^{2.3}$ $TCID_{50}$
3	Box which held cattle for 45 min	$10^{2.5}$ $TCID_{50}$
	Same box 30 min after cattle removed	$10^{2.2}$ $TCID_{50}$

(d). Persistence in looseboxes (log_{10} $TCID_{50}$ of virus collected over 30 minute period)

	0 h	+1 h	+4 h	+24 h
Box with pigs	5.8			
Box after removal of pigs	-	4.4	3.0	2.2
New box with dead pigs		3.1	2.6	2.1

In regard to soiled building surfaces, infection of animals is most likely to occur through animals sniffing contaminated surfaces, rather than from licking and oral ingestion of contaminated material. As noted previously, it is difficult to assess the importance of research reporting the survival of FMDV in blood and other excretions on surfaces because these used sensitive measures to detect the virus. Data on the survival of FMDV in faeces would suggest that this material is important in the spread of FMD. There is no information on the possibility that animals could be infected by sniffing of faecal soiled surfaces. However, FMDV may survive in manure, both liquid and solid, at concentrations sufficient to infect susceptible animals highlighting the need for its removal from environments where animals are held. The disposal of liquid manure from buildings, especially by spray irrigation, may also present opportunities for spread of FMD.

(vii). pastures

It is probable that the survival of FMDV on pastures would be limited because of the action of ultraviolet light. Hyslop (1970), citing a number of authors, suggested that FMDV probably survived for 2-10 days on pasture. Since oral infection in cattle requires about 10^5 $TCID_{50}$, and daily intake of dry matter in cattle is between 8 and 20 kg, contamination rates on pasture of between 5 and 12 $TCID_{50}$ per gm on a dry matter basis would be needed to produce infection. Under Australian conditions the survival of FMDV on pastures would probably be limited but may be enough to allow for oral infection. However, if pasture contamination results from deposition of airborne particles it is more likely that animals will become infected from airborne aerosols than by consumption of contaminated pasture.

(g). Products

(i). Meat

Savi and Baldelli (1962) observed that the usual operations of slaughter and preservation of meat did not guarantee that all FMDV became inactivated and that fresh, salted, cooled and frozen meat represented a risk in the spread of FMD. On the other hand, these authors noted that manufactured meat products, made from FMDV-infected meat usually insured the inactivation of the virus because of physical, physio-chemical and enzymatic factors concerned with the preparation and ripening. They asserted that there has not been any reports of the spread of FMDV attributed to the trade of manufactured meats.

Brooksby (1962) considered that the technical data available indicated that FMDV may be present in carcases from infected animals and that it may persist for long periods under the conditions of the international meat trade. Sellers (1971) noted that many of the primary outbreaks of FMD in Great Britain had been attributed to animals feeding on or sniffing at uncooked meat, bones or offal. He calculated that in order to ingest $10^{5.0}$ $TCID_{50}$, the average dose needed for oral infection, a pig would need to consume 222 mg of pig liver containing $10^{5.6}$ $TCID_{50}$ per gm, 100 gm of offal or bone marrow containing $10^{3.0}$ $TCID_{50}$ per gm (i.e. about half the bone marrow from an ox femur) or 1 kg of liver containing $10^{3.0}$ $TCID_{50}$ per gm (i.e. half a pig's liver). The author noted that with virus concentrations of 10 $TCID_{50}$ per gm or less the amount to be consumed would be greater than a pig's daily food intake.

Lymph nodes from infected animals may contain at least 10^5 $TCID_{50}$ of FMDV per gram (anon, 1966) and the ingestion of a single lymph node by a pig would be sufficient to initiate infection. Information on the loss of virus titre in lymph nodes during storage was not available to assess the amount of lymph node material from frozen carcasses which would need to be ingested to initiate infection.

Sellers (1971) noted that in cattle, the dose required for infection by the oral route is greater than that for inhalation by a factor of 10^4 to 10^5. He considered that where cattle had been presumed to have been infected by contact with bone marrow or offal containing FMDV, it was more likely that infection resulted from sniffing rather than consuming these products. While there is good evidence that meat and meat products have been involved in the spread of FMD, the significance of prolonged survival times in these products is unresolved. Many of the experiments to determine the infectivity of stored or processed meat or meat products have used tissue culture or animal inoculation to detect the presence of FMDV. These techniques, as previously noted, are very sensitive but may not give a good indication of the infectiveness of such products when they are fed to susceptible species or when animals are allowed to inhale from their surfaces.

(ii). Milk –

Since milk can contain up to $10^{5.2}$ $TCID_{50}$ per ml (Burrows *et al.*, 1971) the amount of virus needed to infect pigs by the oral route, approximately 10^5 $TCID_{50}$, would be 1 ml. Sellers (1971) noted that a daily intake of 0.5 litre of milk would infect pigs if the milk contained $10^{2.3}$ $TCID_{50}$ per ml. For calves with a daily milk intake of 0.5 to 9 litres, milk with an FMDV content of $10^{3.3}$ to $10^{2.1}$ $TCID_{50}$ per ml would be sufficient to cause infection.

During the 1967/68 FMD outbreak in the United Kingdom, some concerns were expressed over the possibility of FMDV being spread by aerosols produced by bulk milk tankers exhausting air from the tanker space. However, Hugh-Jones (1976) used a spatial model to examine the likelihood that milk tankers spread FMD during the 1967/68 outbreak and concluded that the risk of milk tankers spreading FMD is certainly less than might have been previously thought.

Hugh-Jones and Tinline (1976) reported that skim milk appeared to have been involved in the spread of FMD to 3 pig farms during the 1967/68 United Kingdom FMD outbreak.

In Australia, milk is only likely to be involved in the spread of FMD from cows to calves on farms. It is possible that stored colostrum could be a means of interfarm spread of FMD if colostrum, collected from cows in the incubation stage of FMD, was provided to another farmer. It is unlikely that skim milk would be involved in the spread of FMD. There is little, or no, skim milk available from dairy factories in Australia for feeding to animals as it is used to produce skim milk powder and other dairy products. The possibility of dried milk being used in calf replacer rations is a potential source of infection with FMDV since the virus has been reported, by Cottral (1966), to survive at least 2 years in this product.

(iii). Whey and Casein

In the absence of information on the concentrations of FMDV in whey and casein, it is difficult to assess the likelihood of infections resulting from consumption of these products. However, available information on survival suggests that these products, if resulting from FMDV-infected milk, would be sources of infection. It might be that several days consumption might be necessary to accumulate an infective dose.

Acid whey, the by-product from casein manufacture, is unlikely to be a source of FMD since the acidification process is detrimental to the survival of FMDV. In addition, this product is unlikely to be fed to animals because of its adverse effect on the digestive tract.

FMDV can survive in sweet whey, produced from Cheddar cheese manufacture, and this product is more likely to be a source of FMDV. Most Australian dairy factories dispose of this by-product onto pasture but some may sell, or use, it for feeding to animals. In the event of an FMD outbreak this source of feed could be important in the spread of the disease, depending on the dilution of virus in the original milk.

(iv). Hides, Skins and Wool

FMDV may survive on wool and in hides but it is likely that infection in cattle and sheep would result from inhalation of virus from their surfaces, rather than by ingestion. In pigs, ingestion of hides could result in infection since FMDV may be present in concentrations of 10^5 pfu/gram in infected animals (Gailiunas and Cottral, 1967). Ingestion of only 1 gram of skin would be enough to produce infection in pigs.

(h). Latent Carrier Animals

Graves *et al.* (1971) found that concurrent infection with bovine enteroviruses could inhibit the multiplication of FMDV in cattle. This effect was discovered during an experiment to determine the time at which infected cattle were most infectious. In the experiment a donor steer was infected with O_1 FMDV by intranasal instillation and susceptible steers were exposed to it for a 24 hour period. The donor steer failed to develop lesions but when it was challenged with the same virus 33 days after the primary inoculation it was found to be fully susceptible to infection, developing classical FMD lesions. FMDV was isolated from the blood, but not from pharyngeal fluids, of the first 4 steers exposed to the donor animal. However, an enterovirus was isolated from the pharynx of these animals. Some of the contact steers which had been kept separate after their 24 hour exposure to the donor animal developed classical FMD after extended periods. Contact steers exposed on day 3 and day 5 developed FMD 42 days after the start of the experiment. The contact steer exposed on day 1 of the experiment remained clinically normal until day 119 when signs of FMD developed. The contact steer from day 4 had not developed clinical signs at day 160, the end of the experiment. No neutralising antibody to FMDV was detected in any of the contact steers before the appearance of lesions. It was postulated that, after infection via the respiratory tract, the FMDV replicated in pharyngeal cells simultaneously with the enterovirus. Because of low levels of FMDV the replication of the enterovirus-dominated and the bulk of the FMDV-RNA that was produced became genomically masked by encapsidation with coat protein of the enterovirus and it was not until FMDV became the predominant virus that disease resulted. Trautman and Sutmoller (1971) found that the type O FMD virus used in these studies would become encapsulated in the coat protein of bovine enterovirus in dually infected tissue cultures. They found that this was dependent on the relative amounts of the 2 viruses in the inoculum.

The significance of the findings of Graves *et al.* (1971) and Trautman and Sutmoller (1971) that concurrent infection of cattle with enterovirus could suppress the production of infectious FMDV, and the development of clinical signs, for long periods is unknown. Bovine enteroviruses have been isolated from apparently healthy adults and calves (Dunne *et al.*, 1974). A serological survey in Argentina found that 63.6% of cattle had antibody to bovine enterovirus (Carrillo *et al.*, 1986). The prevalence of bovine enterovirus in Australia is not known however, Spradbrow (1964) made 10 enterovirus isolates from 281 faecal samples collected from Queensland cattle. It appears that most calves (up to 55%) have been infected with bovine enterovirus by the age of 8 months (Stott *et al.*, 1980). It is probable that there are very few active enterovirus infections present in the cattle population at any time period and therefore the risks of latent carriage of FMDV, due to concurrent infection with enterovirus, is probably low. However, Stott *et al.* (1980) has reported that cattle may be reinfected with, or develop persistent infections with, bovine enteroviruses. These authors recovered enterovirus from 24 of 59 animals from which an enterovirus had previously been recovered.

(i). Recovered Carriers

FMDV infections have been found to persist in cattle, buffalo, sheep and a number of other animal species after lesions of the disease have resolved. In some cases FMDV has been recovered over extended periods, sometimes exceeding a year. As discussed later, the significance of prolonged carriage of FMDV in recovered animals is in doubt with differing results being obtained by various researchers.

The length of time that carrier animals excrete FMDV appears to be related to species. Hedger (1968) found that cattle may excrete FMDV for up to 30 months after infection whilst Pay (1988) found that sheep may excrete FMDV for up to 9 months. African buffalo may excrete FMDV for up to 5 years (Condy *et al.*, 1985)

Graves (1979) observed that up to 50% of recovered cattle, sheep, or goats may become FMDV carriers for periods as long as 6 months with the virus being present in their naso-pharynx. But Donaldson *et al.* (1981) found that although sheep, cattle and goats inoculated with FMDV $O_{1\ MALTA}$ became carriers, animals sampled on farms contiguous to infected farms in the FMD outbreak on Malta, from which this virus was isolated, were not convalescent carriers. However, no information is provided about the animals on the contiguous farms and whether they were ever infected with FMDV.

In Egypt, Tantawi *et al.* (1984) found that 32% of non-vaccinated cattle in endemic FMD areas were carrying FMDV and that 0.8% of non-vaccinated cattle in non-endemic areas also carried FMDV. The carrier rate in non-vaccinated buffalo was 29.8% in endemic areas and 0.8% in non-endemic areas.

In India, Tewari and Singh (1975) collected pooled samples of oesophago-pharyngeal fluids from 15 animals (10 cattle, 2 buffalo, 2 sheep and 1 goat) that had recovered from FMD and was able to isolated FMDV from 11 of the animals. Type 0 virus was detected in 9 samples and types C and Asia I in one each. Type 0 virus could be isolated up to 8 weeks, but not at 16 weeks, after the onset of the disease. In Kenya, FMDV was isolated from cattle 10 months after an outbreak (Paling *et al.*, 1979). Hedger (1968) reported that an average of 20% of cattle sampled still carried FMDV in their oesophago-pharyngeal fluids 7 months after the last infection had been detected in a field outbreak of FMD in Botswana. In one region the rate of carrier animals was 20% twelve months after infection. Most of the carrier animals had high neutralising antibody titres but 4/50 animals did not have titres but still had FMDV in their oesophago-pharyngeal fluid. Burrows (1966) found that 5 out of 10 cattle infected with type A_{119} FMDV still had virus in their oesophago-pharyngeal fluids 26 weeks after infection by intradermal inoculation of the virus into tongue epithelium. The mean virus titre in oesophago-pharyngeal fluids was $10^{1.0}$ pfu/ml with a range of $10^{0.3}$ to $10^{2.0}$ pfu/ml. Following experimental infection with FMD virus type Asia 1 isolates of Indian origin the oesophageal pharyngeal fluid of 6 cattle was stated to yield virus for between 30 and 75 days (Prasad and Kumar, 1981). The virus was detected both by immunofluoresence and by isolation from pharyngeal tissue but not in any other tissues of one carrier animal.

Pay (1988) noted that it is generally considered that the carrier state does not persist in sheep with incidence levels as high or duration times as long as have been reported in cattle. Sheep infected by the intranasal route are more likely to become carriers than sheep infected by contact according to studies conducted by McVicar *et al.* (1972). In Egypt an incidence of 1.2% of virus carriers was found in a population of non-immunised sheep in one area without any history of previous FMD outbreaks (Tantawi *et al.*, 1984). Anderson *et al.* (1976) sampled sheep, cattle and goats at 5 locations where disease outbreaks had occurred in cattle. Virus carriers were found in the cattle at 3 of the 5 locations but no carrier sheep were detected. Carriers were found in one of the goat populations at a prevalence of 1.5%. Another author, Burrows (1968b), found that cross-bred Southdown sheep remained FMDV carriers for up to 5 months after exposure to type A_{IRAQ}. In a comparative study of experimental infection in sheep and goats it was found that at 4 weeks post-exposure, 83% of sheep and 44% of goats were virus carriers (McVicar *et al.*, 1972). Sheep and goats exposed to pigs infected with the Malta outbreak strain ($O_{1\ MALTA}$) for 1 hour were shown to carry virus in

oesophago-pharyngeal fluid for up to 5 weeks after exposure, however, virus titres were only at a trace level (Donaldson *et al.*, 1981). In a group of sheep infected by coronary band inoculation or by contact exposure FMDV was recovered from approximately 80% of the sheep 4 weeks after infection, from 45% after 12 weeks and from 1 animal after 20 weeks (Burrows, 1968b). The mean infectivity declined from approximately 150 pfu/oesophago-pharyngeal sample (0.1 - 0.3 ml) at 4 weeks to approximately 50 pfu/sample at 12 weeks. The highest titre recorded for individual animals was >1000 pfu/sample. FMDV was found to be localised in the tonsillar area, rather than in the pharynx as observed in cattle. It was postulated that persistence of FMDV in the tonsillar region of sheep might signify that the strains used for infecting them were not particularly virulent for sheep. In another experiment 56 sheep and goats were exposed to FMDV by intranasal instillation and 47 became infected and of these, 40 became carriers i.e. FMDV was isolated for at least 28 days following exposure (McVicar and Sutmoller, 1968).

Pigs are rarely found to be carriers of FMDV after recovery from the disease. However, Solyom and Horvath (1974) found that after intradermal inoculation of pigs with FMDV type C the same virus could be recovered from the metatarsal joints, thyroid gland and epiglottis on the 26th day post-inoculation, from the tonsil and epiglottis on the 50th day, from intestinal lymph nodes and bone marrow on the 57th day, and from tonsils on the 64th day. The same authors failed to isolate FMDV from 20 pigs that had survived an epidemic two years previously.

The role of recovered carrier animals in the spread of FMD remains obscure. FMDV recovered from oesophago-pharyngeal fluids collected from recovered animals is pathogenic when injected into susceptible animals, but it has been difficult to demonstrate natural transmission of infection from recovered carriers to susceptible animals. Pathogenicity tests of carrier virus strains present in oesophago-pharyngeal fluid samples showed that carrier virus retained pathogenicity for cattle as well as for pigs up to 30 days after infection (Tewari and Rao, 1983). Oesophago-pharyngeal fluid samples collected from cattle 40 days after infection proved pathogenic for pigs, but not when collected at 113 and 190 days. Titres of infectious virus in oesophago-pharyngeal fluids usually decrease over time and by 30 days after infection are often below the amounts considered necessary to initiate infection in susceptible animals by natural routes of infection (Donaldson and Kitching, 1989). Contact transmission trials showed that FMD-recovered cattle transmitted disease to healthy in-contact animals 7 days after infection but not 30 days after infection.

Bauer *et al.* (1977) conducted a trial where contact, for periods of up to 9 months, between cattle permanently excreting FMDV and unvaccinated and vaccinated cattle and unvaccinated swine gave no proof of transfer of virus. When sheep permanently excreting FMDV the virus were held in contact with unvaccinated sheep and cattle, one sheep which had been in contact with the carrier animals developed a specific increase in neutralising antibodies.

Sutmoller *et al.* (1967) reported that 3 FMDV strains isolated from carrier cattle 6 months after having FMD had reduced infectivity for cattle but their infectivity for pigs was not changed. However, after passage of the viruses in pigs, the infectivity of the strains for cattle was increased. Similar observations were made by Kaaden *et al.* (1975). These results seem to indicate that a reversible attenuation of FMDV may occur in carrier animals. A similar pattern was observed when rabbit-adapted live FMDV vaccines were used and there was a reversion to virulence in the field (Salt, 1993). It is therefore possible that FMDV excreted by carrier cattle may infect other susceptible species and that infection of pigs may provide the opportunity for further spread of FMD.

(j). Summary

A consideration of the ways in which FMD may be transmitted between farms in Australia suggests that animal movement and possibly airborne spread may be important. The importance of the latter mechanism still remains to be determined. However, a preliminary evaluation of the diverse climatic conditions which occur in Australia suggest that airborne spread of FMDV may only be of importance in some regions, and possibly only during some periods of the year.

In regard to animal movement, carrier animals may be important in the dissemination of FMDV in Australia. The role of sheep in the spread of FMDV in Australia may be particularly important because they may be infected, and excrete virus, but not develop clinical signs of FMD. It is possible to envisage a scenario where FMD occurs on a sheep property, remains undetected because few animals develop clinical signs, and is spread to other properties through the sale of animals.

Vehicles used to transport animals have been implicated in the spread of FMD in other countries and they may also be a method of spread of FMD in Australia through faecal and other contamination of floors and crates. Vehicles are unlikely to be cleaned between livestock loads, especially in areas where water is scarce, increasing the opportunities for spread of disease.

Embryo transfer is a possible means of disseminating FMDV within Australia because the embryo washing techniques are unlikely to be used, except when importing countries or clients demand such a protocol. It would be possible for embryos to be collected from animals incubating FMDV and be inserted into recipient animals on another farm or farms.

Of the remaining ways in which FMD may spread between farms, probably only the use of sweet whey from cheese manufacture, for feeding animals, is likely to be a major risk. Other dairy products, and milk, probably represent less of a risk since they are unlikely to be regularly fed to animals in Australia. Feeding of milk to calves may be one way in which FMDV may be spread on a farm.

The possibility that FMDV may survive for long periods in dried milk and casein highlights the need to ensure that milk from infected areas, if collected during an outbreak, is processed in such a manner that ensures that virus does not survive. On present evidence, this would appear to be a difficult task. Alternatively, its use for domestic milk supply in urban areas, leaving aside considerations of public perception, may be the least risk solution.

The feeding of meat, specifically trimmings and bones from butcher shops and boning rooms, would appear to be a potential way in which FMD may spread. However, in the Australian context such materials are unlikely to be fed to pigs because of current regulations on the feeding of such material, as swill, to pigs.

Artificial insemination of sheep and cattle would appear to be an unlikely method of spread of FMD in Australia because of the way in which semen is processed. There is a danger with ram and boar semen which may be used immediately after collection. This could be a means by which FMD could be spread within sheep flocks or pig herds.

The possibility of non-production animals, except for feral pigs and buffalo, being involved in the spread of FMD in Australia appears low. The role of buffalo in the spread of FMD may have been reduced by the BTEC program which has provided control of previously feral buffalo. This provides a greater opportunity to control FMD in this species. The possible role of feral pigs in the epidemiology of FMD in Australia needs further evaluation, especially as anecdotal evidence from other countries suggests that they play little part in the spread of FMD. Where opportunities exist for the intermingling of domestic stock and feral and native animals, such as at water sources, there may also be an increased risk of disease transmision between species. In such situations infection of feral and native animals may play a greater part in the maintenance and spread of FMD.

Based on overseas reports, the potential role of fomites, feed and water in the spread of FMD in Australia would appear to be small. However, the research examined in this review does not provide sufficient information on concentrations of virus likely to be present on such objects, nor on the concentrations surviving under climatic conditions likely to be experienced in Australia, to evaluate the role of fomites in the epidemiology of FMD in Australia.

A summary of the maximum amounts of FMDV reported in tissues and excretions and the amounts of these which would be needed to intiate infection are shown in Table 18. This data represents the worst-case scenario as the average virus concentrations would in most cases be lower.

Table 18. Sources of Foot-and-Mouth Disease virus and amounts of excretions or tissues needed to initiate infection by the oral or respiratory routes.

Source	maximum virus concentration reported	route of infection	minimum dose for infection of pigs & cattle	minimum volume for infection
respired air	10 $TCID_{50}$ /m^3 (cattle) 10^5 $TCID_{50}$ /m^3 (pigs)	resp.	10 $TCID_{50}$	12 minutes breathing
faeces	100 $TCID_{50}$ /g	oral	10^5 $TCID_{50}$	1 kg
urine	10^4 $TCID_{50}$ /ml	oral	10^5 $TCID_{50}$	10-100 ml
vaginal fluid	100 $TCID_{50}$ /ml	oral	10^5 $TCID_{50}$	1000 ml
saliva	10^7 pfu/ ml	oral	10^5 $TCID_{50}$	< 1 ml
milk	$10^{5.5}$ $TCID_{50}$ /ml	oral	10^5 $TCID_{50}$	1 ml
blood	10^4 $TCID_{50}$ /ml	oral	10^5 $TCID_{50}$	10 ml
muscle	unknown	oral	10^5 $TCID_{50}$	unlikley*
lymph node	10^6 $TCID_{50}$ /g	oral	10^5 $TCID_{50}$	< 1 g
bone marrow	10^6 $TCID_{50}$ /g	oral	10^5 $TCID_{50}$	1 g
liver/spleen	10^4 $TCID_{50}$ /g	oral	10^5 $TCID_{50}$	10 g
skin	10^5 pfu /g	oral	10^5 $TCID_{50}$	1 g

NOTE: Animals may be infected by sniffing at these secretions and tissues. No information is available on the amount of time that an animal must sniff at these fluids or tissues to become infected.

* The pH changes which occur in muscle after death are considered to be detrimental to the survival of FMD virus.

EPIDEMIC PARAMETERS REPORTED FROM NATURAL FMD OUTBREAKS

This section is an attempt to provide information from outbreaks of FMD where the disease had been introduced into a susceptible population. Such information is important for validation of disease models as it can show whether the models reflect the natural rates of spread of disease, and can therefore be used to estimate logistical requirements for disease control. Information on the spread of FMD in naive populations is available occasionally in official reports (Manuel *et al.*, 1974) and sometimes in the published literature. The majority of information available on the spread of FMD in susceptible populations is from the United Kingdom, both in official reports and the published literature. The majority of this information relates to the 1967/68 FMD outbreak. Other information reviewed here comes from Malaysia, Malta and Canada. Most other reports of FMD are of outbreaks due to the introduction of new strains of FMD into countries which already had endemic FMD and in which there is a vaccination program for the control of the endemic strain. It is difficult to relate this situation to the introduction of FMD into the susceptible livestock population of Australia.

(a). Sources of Infection

Except for the 1967/68 FMD outbreak in the United Kingdom, most reported outbreaks of FMD in susceptible populations have been relatively small with limited spread of disease. The Canadian (1952), Maltese (1975) and Malaysian (1974) outbreaks were confined to less than 50 farms in each outbreak. The 1967/68 FMD outbreak in the United Kingdom is an exception where large numbers of farms were infected. Much of Australia's planning has been based on this outbreak which may have resulted from a combination of circumstances including heavy stock densities and conditions favourable for the wind-borne spread of FMDV.

Sellers and Forman (1973) reported that the risk of spread of FMD, in the 1967/68 FMD outbreak in the United Kingdom, was associated more with the carriage of infected slaughterhouse waste, movement of animals and people or vehicles carrying animals, than through collection of milk, artificial insemination or movement of other types of vehicles. Outbreaks in pigs were more likely to result in spread of infection to other premises when compared with outbreaks in cattle. Secondary outbreaks attributable to airborne spread occurred only in ruminants. The decline in the epidemic was thought to be associated with the removal of pig sources of FMDV.

An analysis of the 1966 Northumberland (U.K.) outbreak showed that 9/32 outbreaks could be attributed to movement of cattle, sheep, people or vehicles; 5/32 outbreaks occurred where the animals were in the next field to either infected animals or direct contacts; and the remainder (18) were ascribed to airborne spread (Sellers and Gloster, 1980). In contrast, an analysis of the 1952 Canadian outbreak divided the method of spread into people movements (11), animal movement (5), airborne (11), meat (2) and milk (1) (Sellers and Daggupaty, 1990).

In the 1975 outbreak of FMD on Malta the most likely method of infection for the 24 outbreaks was assessed as contact of pigs with swill, offal and garbage (10); airborne spread involving sheep, cattle and goats (4); people movement (1); and adjacent to infected farm (1) (Sellers *et al.*, 1981). In another 7 outbreaks infection could be attributed to more than one method whilst in one outbreak the cause remained obscure. The disease epidemic started in a pig herd.

The 540 FMD outbreaks recorded in the U.K. between 1938 and 1954 have been classified according to their presumed source (anon, 1954) as follows: birds (16%), swill (40%), contact with imported meat and bones (9%), unknown but probably swill (7%), origin not determined (28%). These data were collected before the recognition of airborne spread of FMDV and the 16% of outbreaks recorded as being due to the movement of birds were most probably due to wind-borne spread of FMDV.

(b). Geography

In a discussion of the epidemiology of FMD in the Americas, it was noted that in some countries where FMD is endemic, there are areas which are recognised to be free from FMD (anon., 1975). The infected areas are usually located in more densely populated sectors with a more intensive marketing system of animals, have more fertile soils and are located in the vicinity of large population centres. Foot and Mouth disease rarely occurs in the savannas which are exposed to sharp seasonal rainfall and drought conditions, and on the semi-arid plains which only support a small number of animals. Outbreaks in such areas usually follow the introduction of animals from endemic areas. In the southern part of South America these conditions involve relatively large areas and are relatively isolated from other areas. They also have a predominantly sheep population. The paper notes that in the regions where sheep are raised without other livestock the climatic and other conditions do not appear to favour the perpetuation of FMDV.

(c). Seasonal variation

Peralta *et al.* (1982) studied the seasonal variation of FMD outbreaks in Paraguay and found that it was due to vaccination measures. Because more personnel were in the field during the months when vaccination was undertaken, there was a greater chance for observing the occurrence of FMD. In addition, vaccination often involved the assembly of large groups of animals from neighbouring farms in one place to facilitate vaccination, increasing the contact between animals and the likelihood of disease spread. FMD outbreaks were also seen to occur in cycles of about 3-4 years duration which was probably caused by changes in cattle populations and changing population immunity. In India, a cycle of 6 years was observed between major outbreaks of FMD by Sharma and Singh (1993). This was also thought to be due to altered herd immunity resulting from population shifts. A similar 6 year cycle was observed by Boldrini (1978) in Europe before the era of systematic vaccination.

(d). Incubation Period

The incubation period appears to be important in the spread of FMDV as both its length and the time after infection when animals commence to excrete virus determine how rapidly FMD might spread. Virus production in animals often takes place before there is evidence of clinical signs. In some species, such as sheep or goats, there may only be limited, or no, evidence of lesions. By the time the first animal in a herd or flock has presented with clinical signs there may be a large number of its cohorts infected and excreting virus. There may be considerable amounts of virus excreted in various secretions or present in body tissues before lesions are present. Virus may be released in aerosols, generated by normal respiration, before lesions develop. Although lesions that develop during the clinical phase are rich in virus, the animal is less of a hazard than it is during the incubation period because ingestion of FMDV is a less effective means of infection compared to inhalation of virus particles . The length of the incubation period is affected by the strain of FMDV infecting an animal, the infecting dose and the route of infection. Some of the experimental routes of infection, such as intradermal inoculation of the tongue, produce much shorter incubation periods than those seen when animals are naturally exposed to infection. Hugh-Jones and Tinline (1976) reported that in the 1967/68 FMD outbreak in the United Kingdom the incubation period of the disease in cattle varied with age and the amount of exposure to infection. When there was a massive exposure to infection, the incubation period might be as short as 36+ hours but under usual field conditions the minimum incubation period was between 3 and 5 days. The incubation period for calves exposed to natural virus aerosols (3 - 7 days) were in most cases shorter than those for calves

which developed disease following exposure to low doses of artificially generated aerosols (Donaldson *et al.*, 1987).

In the 1967/68 FMD outbreak in the United Kingdom the incubation period in pigs was observed to be between 4 and 9 days, with a tendency for the incubation period in younger pigs to be in the longer part of this range (Hugh-Jones and Tinline, 1976). Very young piglets, within 2 to 3 weeks after birth, may die before lesions develop and their deaths may occur at the time when sows are just beginning to develop clinical lesions (Donaldson *et al.*, 1984). The maximum amount of virus was excreted by pigs before lesions were observed (Donaldson *et al.*, 1970; Sellers and Parker, 1969).

The incubation period in natural cases in sheep is usually between 3 and 8 days, but it has been claimed that the incubation period may be as long as 3 weeks (Geering, 1967). In the 1967/68 FMD outbreak in the United Kingdom the incubation period was considered to have been between 6 and 13 days with a median of 9 days (Hugh-Jones and Tinline, 1976). The mean times from exposure to the onset of viraemia, pyrexia and appearance of vesicular lesions were 2.5, 3.8 and 4.7 days respectively for sheep exposed by natural aerosols to infection with the O_1 strain of FMDV.

(e). Morbidity

Sarma *et al.* (1984) investigated the morbidity rates in various species when exposed to natural FMDV infection in north-eastern India. In cattle the morbidity rate was 43.55%, while for buffalo it was 14.08%, for pigs 13.67%, in sheep it was 2.67% and for goats 2.38%. The low morbidity rates for sheep and goats are probably related to the observation that often a high proportion of these species may be infected but show no clinical signs. For example, Sharma (1978) has shown that after experimental infection that up to 70% of infected sheep, as determined by excretion of FMDV, did not show clinical signs of FMD.

(f). Herd Size and Stocking Density

Herd size and stocking density have been observed to influence the spread of FMD. Hugh-Jones (1972) reported that during the 1967/68 FMD outbreak in the United Kingdom the number of dairy herds affected in an area was related to average herd size. Less than 1% of herds with less than 10 cows were affected but 29% of herds with more than 80 cows were affected. He postulated that large herds may be more efficient virus traps or samplers than smaller herds, or that subsequent spread of the disease is more efficient within larger herds ensuring that the disease is not missed in herds

where there has been only a clinically mild primary infection. However, there was no serological survey to back up this theory. The density of stock in an area also had an effect on the occurrence of FMD. It was suggested that at some particular density level a collection of dense smallholdings became the equivalent of one large farm, but one in which only one unit is affected.

Sellers and Daggupaty (1990) noted that in the 1952 Canadian FMD outbreak the average cattle density was low - an average of 4.5 per km^2 compared to 71 per km^2 in the 1967/68 United Kingdom outbreak in parts of Hampshire. The authors believe that the low stock density coupled with the fact that few pigs were kept reduced the possibility of large amounts of airborne virus, and hence limited the spread of the disease in Canada. In the 1974 Malaysian FMD outbreak, the density of the animal populations in affected areas was relatively low and usually the distances between animals was several hundred metres (Manuel *et al.*, 1974). These factors apparently resulted in a very limited spread of the disease from the initial focus.

Richards (1978) noted that the spread of FMD within a large piggery, of say 5000 head, or a large feedlot of cattle is likely to be rapid, especially if animals are kept inside where conditions favour survival of airborne virus. The likelihood of a massive manufacture of virus is enhanced.

THE ROLE OF VACCINATION IN THE PREVENTION OF FMD SPREAD

There are many factors involved in the use of vaccination for the prevention of spread of FMD in a country where there is a fully susceptible animal population. These include the identification of the virus strain involved in an outbreak, the selection of an appropriate virus strain for vaccination, the type of vaccine to be used, the type of vaccination strategy to be employed, the timing of vaccination in relation to the outbreak, whether vaccinates are allowed to move from vaccinated areas to other areas, and what to do with vaccinates after eradication.

There are a number of techniques available to select an appropriate vaccine strain which are outside the scope of this review. However, it is usually found that outbreak strains of FMDV are often not good for use in vaccines as they may not grow abundantly in the media used to manufacture vaccines.

(a). Vaccine types

The most effective commercially available vaccines are tissue culture grown chemically inactivated whole virus vaccines (Kitching, 1992). However, peptide, recombinant and vector expressed virus protein vaccines are currently under development and these may substantially alter the present approach to vaccination.

(b). Vaccination Strategies

There are 2 strategies for the use of vaccination to control the spread of FMD in an outbreak occurring in a country with a fully susceptible population. These are barrier vaccination and ring vaccination.

(i). Barrier vaccination

Barrier vaccination involves the vaccination of all susceptible animals in a buffer zone, usually selected on the basis of geographical features, to prevent the spread of FMD from one area to another. The size of the buffer area will depend on the epidemiology and likely methods of spread of FMD in the area. For example, if windborne spread of FMD was possible then a wide barrier would be needed to reduce the risk of the virus infecting animals outside the barrier zone. Several countries have used this technique to prevent the introduction of disease from neighbouring countries. Examples include

South Africa which maintains a vaccination buffer zone along its northern borders. This has been successful in preventing outbreaks of FMD in the rest of the country, even though outbreaks have occurred within the buffer zone. Barrier vaccination is necessarily extensive unless some geographical feature can be utilised to minimise the area.

(ii). Ring vaccination

Ring vaccination involves the vaccination of all susceptible livestock within a prescribed area around an outbreak and is carried out in conjunction with the slaughter of infected animals and those that have been in contact with them. The 1968 Committee of Enquiry on Foot-and-Mouth Disease in the United Kingdom has recommended that ring vaccination should be started both at the centre and the periphery of the area to be vaccinated. Ring vaccination has been practised by Denmark to combat isolated outbreaks of FMD. Their approach relied upon prompt identification of the outbreak strain of virus so that vaccination with a homologous vaccine could be implemented. All cattle and sheep, but not pigs, within a radius of at least 10 km of the FMD outbreak are vaccinated within 10 days of the start of the outbreak. Animals are allowed to move from the vaccination area after a period of 30 days has elapsed. For 5 months after the ring vaccination is completed, only vaccinated stock can be brought into the vaccination zone. There have been no outbreaks of FMD when vaccinated cattle have been released from the vaccination zone after 30 days, and allowed to mix with susceptible animals in the other parts of Denmark. Pigs are not vaccinated because they do not usually give a good immune response to vaccination and also because there is a rapid turn-over of pig populations so that there will always be a high percentage of un-vaccinated animals. On the other hand, the 1968 Committee of Enquiry on Foot-and-Mouth Disease in the United Kingdom recommended that all cattle and sheep over 3 months of age and all pigs over 2 months of age should be vaccinated if ring vaccination was used following an outbreak of FMD.

(c). Timing of the decision to vaccinate

The decision to use vaccination as an aid in the eradication of FMD involves cost-benefit analyses. Several computer models have been used to examine the cost-benefit of vaccination both in countries where FMD is endemic (Carpenter and Thieme, 1980; Stougaard, 1984; Lorenz, 1989), and in countries that are FMD-free (Power and Harris, 1973; James and Ellis, 1978; Miller, 1978; Davies, 1988; Berentsen *et al.*, 1992; Dijkhuizen, 1989). Some of the analyses of the use of vaccination when FMD has been

introduced into a fully susceptible population have demonstrated that once the disease has reached a certain incidence, vaccination is the most cost-effective option. Most of these models have not included in their analysis the costs of loss of export markets. **There would be merit in using or adapting existing models to examine when and where vaccination might be used to aid in the eradication of FMD in Australia.**

(d). Effect of vaccination on spread of FMD

Animals vaccinated during an outbreak will be protected against the development of clinical FMD but may still become infected and excrete virus.

The spread of FMD was only slightly reduced when vaccinated sheep were exposed to an estimated $10^{3.2}$ $TCID_{50}$ of FMDV during a 2 hour contact period with aerosols generated by infected pigs (Gibson *et al.*, 1984). Sheep in the experiment were vaccinated once or twice prior to exposure with the last vaccination taking place one week before challenge. Only 1 of 12 vaccinated sheep developed lesions while 4/4 un-vaccinated control developed lesions. Viraemia first occurred in the un-vaccinated animals between 1 and 3 days post exposure and lasted for 3 days. Viraemia was not detected in vaccinated sheep. Virus was detected intermittently in pharyngeal fluid from sheep vaccinated with a 6-fold dose of vaccine on one or two occasions. FMDV was consistently isolated from pharyngeal fluids from un-vaccinated sheep and from animals vaccinated once with a 3-fold dose. It was observed that neither 3-fold nor 6-fold doses of vaccine administered shortly before challenge produced enough protection to prevent airborne infection but would have some value in reducing the likelihood of further spread.

Donaldson and Kitching (1989) found that when 3 weeks elapsed, between the first vaccination of cattle and natural infection, the amount of virus excreted was unlikely to result in transmission of infection even to animals in close contact. With shorter periods between the first vaccination and exposure to infection, the amplification and excretion of large amounts of virus by partially immune animals may result in the transmission of infection by vaccinated animals. This has obvious implications for vaccination in the face of an outbreak in that it is desirable to include all livestock in a ring vaccination program to minimise spread.

It was estimated by the 1968 Committee of Enquiry on Foot-and-Mouth Disease in the United Kingdom that 70 to 80% of animals in an area would need to be vaccinated to prevent spread of FMD.

(e). Carrier animals

Anderson *et al.* (1974) reported that in Kenya the prevalence of carrier cattle in an area where 6-monthly vaccination with an inactivated vaccine was carried out was 0.49% compared to 3.4% of cattle in an unvaccinated enzootic area.

When cattle were vaccinated and presented with a severe natural field challenge with FMDV, nearly all animals (85%) with sufficient immunity to protect them against clinical disease were, however, susceptible to local pharyngeal infection and became virus carriers (Hedger, 1970). Donaldson and Kitching (1989) showed that vaccinated cattle, which become infected soon after vaccination, can transmit FMDV to susceptible animals for up to 7 days after they are infected. The likelihood of vaccinated animals becoming infected carrier animals, when exposed to FMDV, was related to the length of time between vaccination and exposure to infection. Donaldson and Kitching (1989) showed that a 3 week period between vaccination and infection with FMDV resulted in carrier animals which excreted only low titres of virus which was insufficient to infect in-contact susceptible animals. Recent evidence (Doel *et al.*, 1993) has shown that vaccination may protect animals against clinical disease, but not infection, when it is administered only 4 days prior to exposure to FMDV. Their observations suggest that vaccination in the face of an outbreak would result in the production of carrier animals with a range of infectivity depending on the period between vaccination and infection and the duration of infection.

The Danish experience, that movement of vaccinated animals from vaccination areas, after the elapse of 30 days from the last animal vaccinated, had not resulted in the spread of FMD to susceptible populations would suggest that, in terms of the epidemiology of FMD, the movement of vaccinated animals may present little risk. However, the Danish situation may be unique as they have only 6 major livestock markets and there is usually little movement of animals. The European Commission for the Control of Foot-and-Mouth Disease recommended that 3 months should elapse after vaccination before animals are exported from a barrier vaccinated area.

In terms of trade, the presence of FMD vaccinates in Australia might prejudice sale of animals and animal products. At the present time, the only way to identify vaccinated animals which are carrying virus in their pharyngeal region is by probang sampling and virus isolation. This is not a sensitive method for detecting virus carriers since FMDV may only be intermittently present in pharyngeal fluids. Alternative methods for detection of virus carriers are being developed, including an ELISA test for virus infection associated antigen and polymerase chain reaction (PCR) techniques (Kitching, 1992).

DISCUSSION

(a). Geographic distribution of FMD virus types and risks to Australia

The geographic distribution of FMDV strains is important in a consideration of the epidemiology of the spread of FMDV. For example, the virtual eradication of endemic FMD from most of Europe in recent times has substantially reduced one of the potential risks of introducing FMD into Australia by animal products carried in the luggage of travellers from the Mediterranean area. However, a new outbreak of FMD has been reported in Italy which occurred after the introduction of animals from Eastern Europe. This highlights the need for timely intelligence on the occurrence of FMD throughout the world to ensure that the risks of the introduction of this disease are minimised.

New Zealand authorities have conducted a risk analysis of the importation of various diseases into their country by travellers and in postal items which is based on traffic flows from various countries, the prevalence of the disease in the country, the risks of spread in New Zealand and the economic costs to the country. Australia should adapt or modify such an analysis to quantify the risks of introducing FMD on a country by country basis by travellers and other means. Such an analysis would be useful in targeting animal quarantine inspections of traveller's luggage and would also provide some indication of the strains of FMDV likely to be imported into this country. The latter information could help to identify the way FMDV might spread in Australia if the strain of virus is important in determining the potential for airborne spread. As previously noted, Donaldson (1972) reported that FMDV strains from regions with a dry climate were relatively more stable in saliva aerosols stored at 70% relative humidity and 55% relative humidity than those from more temperate climates. The strains involved in most of the recent epidemics in Europe since 1964 seem to produce more infective aerosols compared to strains isolated from outbreaks in the Near East. The latter isolates also appeared to be shed in lower numbers from the pharyngeal region. The differences may be due to selection for the ability to spread by the airborne route or by animal movement depend on climatic conditions. Anderson et al. (1974) reported that in Kenya the O and SAT 2 subtypes of FMDV had a distinct geographic distribution which appeared to be associated with livestock movement patterns. In South America, Rosenberg and Astudillo (1978) have reported that the three types of FMDV present in Paraguay have distinct epidemiological patterns. Outbreaks due to type O occur every 4 to 5 years and this epidemic cycle is probably related to the cattle population immunity half-life. Infection with type A FMDV is endemic and epidemic outbreaks are very rare. Extensive outbreaks caused by type C FMDV occur at unpredictable intervals and the virus appears to be inactive during inter-epidemic years.

These observations were made in Paraguay where 60% of farms have never vaccinated for FMD and only 26% have used FMD vaccine occasionally. In Botswana, Falconer (1972) has reported that the SAT 1 strain of FMDV causes generalised infection in sheep and goats but the SAT 3 strain is relatively non-pathogenic. Fernando (1985) has reported that although type O FMDV is endemic in Sri Lanka when the type C virus was introduced from India in 1970 it failed to become endemic. Many of the predictions about the epidemiology of FMDV in Australia have assumed that the disease will behave in a similar fashion to the outbreak in the United Kingdom during 1967/68. It has been suggested by Hyslop (1970) that the outbreak in Shropshire was unusual and that the type O strain of virus involved may have had a high degree of virulence and an increased ability to survive for long periods in aerosols. Donaldson (1979) cites work by Fogedby *et al.* (1960) showing that type O virus was readily transmitted between animals housed in separate looseboxes within an isolation compound but type A virus was not.

FMDV types A, O and C have the widest distribution throughout the world. Type A exists in Russia, the Near East, Central Asia, the Far East, Africa and South America. Type O virus continues to infect many countries in the Near East, Southern Asia, a part of Africa, and South America. Type C virus exists in Russia, Southern Asia, the Far East, Africa and South America. SAT 1, 2 and 3 viruses were only found in Africa until 1962 when SAT 1 caused an outbreak in the Middle East. The Asia 1 serotype has so far been confined to the continent of Asia. Countries reporting the occurrence of FMD to OIE/FAO in 1991 are shown in Figure 3. However it should be emphasised that not all countries are members of the OIE and they are not required to report on the occurrence of various diseases. An example is China which has FMD but is not a member of OIE, but is expected to become a member in the near future.

The nucleotide sequence of strains of FMD virus may evolve over a period of time to become specific to a geographical area (Vosloo *et al.*, 1992). This is probably due to a complex interaction of climatic conditions, vaccination effect on immune status of animals, animal movement and species present in an area. It is known that the strain of FMD virus affects its production and survival in aerosols. Whether this is reflected in the nucleotide sequence is not known. It could be postulated that an FMD virus strain which has become 'adapted' to a particular geographic area may produce a different epidemiological pattern when transferred to another area. The implications of these findings are that FMD virus strains may behave differently, epidemiologically, in Australia than in their country of origin. The presence of a animal population which has had little, or no, contact with FMD viruses may also influence the epidemiology of FMD in Australia.

Figure 3. Reported occurrence of FMD (from 1991 FAO/WHO Animal Health Yearbook)

(b). Significance of FMD Epidemiology for different Australian regions

The factors influencing the spread of FMD after an initial outbreak includes the number and species of animals involved, the period before the disease is reported, the topography of the area, the livestock density and the climatic conditions. Stock movements are likely to play an important role in the epidemiology of FMD in Australia. **An analysis of stock movements similar to that suggested by Buik (1979) or by Sanson *et al.* (1993) should be conducted to assess the areas of greatest risk from the spread of FMD by such movements.**

(i). Intensive Dairying area with climate potentially favourable for windborne spread of FMD

It has previously been assumed, in planning for the combat of FMD, that some of these areas might have climatic conditions which favour the long distance spread of FMDV by wind. Some of the dairying areas are probably similar to some of the areas affected with FMD in the 1967/68 United Kingdom outbreak, but with a milder climate. In the absence of climatic information, and using data such as the humidity maps shown in Figure 2, it could be speculated that windborne spread of FMDV is only likely to occur in the winter months. However, it is possible that the environmental temperatures and humidity will favour some survival of some strains of FMDV in respiratory aerosols and that there could be some short distance spread which might be from farm to farm or over a limited distance. **There is a need for the collection and analysis of climatic information in the intensive dairying areas to determine whether wind-borne spread of FMDV is likely to occur and the times of the year in which it is likely to occur**. Even without the agency of windborne spread of FMDV, the disease is likely to spread more rapidly in the intensive dairying areas because of the high stocking densities. Because animals are yarded twice a day for milking and because they are likely to be grazed at very high stocking rates, the opportunities for spread of the disease between individual animals in a herd are likely to be high. It is likely that several animals in a herd may be infected at the same time presenting a greater concentration of virus in respiratory aerosols and in products such as milk, prior to the occurrence of visible signs of disease. The aerosols produced as a result may be of sufficient concentration, and able to survive for long enough to infect animals on adjacent farms. The large movements of cull animals and calves at certain times of the year in these areas also constitute a greater hazard to the spread of FMD than in some other areas. Counter-balancing this is the likelihood that FMD will be detected at an early stage because animals are milked twice a day.

(ii). Intensive sheep raising area with climate potentially favourable for windborne spread of FMD

It is difficult to assess the likelihood of the spread of FMD in sheep raising areas. The fact that FMD can be difficult to diagnose in sheep and that infection with FMDV does not always cause clinical signs, means that observational studies may not provide a clear indication of the epidemiology of the disease in sheep-raising areas. Anecdotal evidence suggests that where both sheep and cattle are run, cattle are more likely to show clinical signs of FMD. However, it is difficult to determine whether sheep were infected but the disease was not diagnosed or whether there is a species effect. For example, Anderson *et al.* (1976) reported that in a confirmed SAT 2 outbreak in cattle in Kenya, no virus was isolated from sheep or goats in the same area although 56% had significant antibody titres. In addition, they reported that there are no confirmed reports of clinical FMD in sheep and goats in that country. In two reports from the 1967/68 FMD outbreak in the United Kingdom, sheep were not observed to be affected by, or spread, FMD. Sellers and Gloster (1980) noted that in the 1966 Northumberland outbreak there were many sheep in the area but only a very small percentage, compared with cattle, were described as having lesions of FMD. They attributed this observation to the difficulty of seeing lesions in sheep, but noted that in the absence of lesions, infected sheep can excrete airborne virus. Hugh-Jones and Wright (1970) reported that infected sheep flocks did not appear to frequently infect stock grazing adjoining fields. They noted that this may have been because adjacent groups were usually also sheep, which although becoming infected might have missed being diagnosed. In neither of these cases is there any information provided, such as serology results, to back-up the suggestion that sheep either were infected or alternatively remained un-infected.

Gallo (1969) reported that in Sicily, the re-occurrence of FMD on farms was associated with the presence of sheep. Serological investigations of sheep on 59 farms where FMD had been previously diagnosed in cattle demonstrated the presence of antibodies to the strain of FMDV which had been detected in the cattle. No clinical signs of FMD had been observed in the sheep on any of the farms.

Pay (1988) noted that there are differences in the pathogenicity of field virus strains for different hosts. In the SAT 1 outbreak in 1962/63 in the Middle East the virus was unusually pathogenic for sheep while the A_{22} subtype produced severe disease in sheep and goats in Iran and caused a severe outbreak in Sikkim. In Botswana, the SAT 1 virus caused generalised infection in sheep and goats, while the SAT 3 virus was almost non-pathogenic (Falconer, 1972). In field outbreaks, lesion rates as low as 7% have been reported in South America (Fernandez, 1970) but in India lesion rates as high as 78% were seen in indigenous breeds of sheep (Singh and Sharma, 1980).

However, lesion rates in exotic and cross-bred stock were lower, with 48% and 46% of sheep having lesions, respectively. Burrows (1968b) noted that the frequency and character of lesions in sheep can be altered by environmental and climatic conditions. Burrows (1968b) also reported that following experimental infection by coronary band inoculation or by exposure to infected donor sheep type A FMDV produced the most obvious signs of disease in susceptible sheep whilst type O produced the least.

Joseph (1986) reported that, in the ASEAN region, FMD is most commonly observed in cattle, buffalo and pigs and rarely in sheep and goats. Reported cases in sheep and goats are rare and these animals were considered by the author to play an insignificant role in the epidemiology of the disease in the region. However, this anecdotal information is different to that reported from India where Pay (1988) noted that there were large differences in the susceptibility of sheep and goats to FMDV in different regions of the country. Pay (1988) has also reported that sheep were thought to have served as a reservoir of FMD infection for cattle in Sicily. Nomadic movements of sheep and goats in Turkey have led to the extensive spread of the disease. The seasonal movements of sheep and goats in India were considered to give rise to the spread of infection during FMD epizootics.

An experimental observation that may be important in the assessment of the possible epidemiology of FMD in intensive sheep-raising areas of Australia is that of McVicar and Sutmoller (1972) who found that the infection rate was lower in sheep exposed to infected sheep than it was for sheep exposed to infected cattle.

Similar observations in relation to stock density as those made in regard to intensive dairying areas would also apply in intensive sheep-raising areas. Stock density and the close proximity of farms would aid in the spread of FMDV with or without the aid of wind-borne transmission.

These observations suggest that the epidemiology of FMD in an intensive sheep-raising area might vary with the strain of infecting virus and that the spread of disease may be slower than in cattle-raising areas. In particular, the observation of McVicar and Sutmoller (1972) suggests that, with some strains of FMDV, the spread of infection between flocks in a predominantly sheep-raising area might be less rapid than the spread in an area where there are both sheep and cattle enterprises. In the situation where infection was due to a virus strain which produced minimal or no clinical signs, sheep might serve as a reservoir of virus for other susceptible stock in the area. The fact that sheep are irregularly inspected increases the likelihood that when FMD is detected, irrespective of the ability of the infecting strain to produce clinical signs, there will be large numbers of infected animals in a flock when the disease is first diagnosed. The disease could smoulder in a flock until sufficient animals become

infected to produce infective aerosols capable of being spread over short or long distances on wind currents. This may be of greater importance if climatic conditions are favourable for survival of FMDV in aerosols. As with intensive dairying areas **there is a need for a detailed analysis of climatic data to determine the risks of airborne transmission of FMDV in the intensive sheep-raising areas.** The data in Table 6 for virus excretion in aerosols from sheep and cattle, infected after contact with infected donor animals, suggests that virus output in sheep would compensate for their smaller respiratory volume and that a group of infected sheep might produce a more concentrated aerosol than a single bovine. The potential for FMD in sheep to remain undetected until large numbers of sheep are incubating the disease could produce virus concentrations in the air which would result in an aerosol remaining infective over long distances down wind from the infected flock if climatic conditions are favourable for virus survival.

The other problem which may arise with undetected infection is the occurrence of carrier animals which could spread infection to other areas. Pay (1988) noted that the incidence and duration of the carrier state in sheep is not as great as those reported in cattle. In Egypt, the incidence of carrier sheep in a population of non-immunised sheep from an area which had no history of outbreaks of FMD was 1.2% (Tantawi *et al.*, 1984). Russian workers have reported that FMDV isolated from carrier sheep after 106 days were fully pathogenic for sheep and pigs by inoculation (Khukhorov *et al.*, 1973). American workers also found that FMDV isolated from carrier sheep were pathogenic for susceptible cattle (McVicar and Sutmoller, 1968). However, Pay (1988) reported that experiments carried out in Germany failed to transmit infection from carrier sheep to in-contact cattle. However, sub-clinical infection occurred in one susceptible sheep in contact with carrier sheep. Similar experiments in India failed to transmit infection from carrier sheep to in-contact sheep submitted to physical or chemical stress.

The worst scenario for the intensive sheep-raising areas would be infection with a virus strain which spreads readily between sheep but produces minimal clinical signs, coupled with the climatic conditions favourable for air-borne spread of FMDV. If this occurred in a period when there were seasonal movements of sheep between areas it could be expected that FMD could be spread widely.

Since the strain of virus might influence the epidemiology of FMD in sheep-raising areas there is a need to conduct a risk analysis of the likely sources, both country and product types, of introduced FMDV.

(iii). Semi-intensive beef/sheep raising areas

The lower stock density and the probability that these areas have climatic conditions which are less favourable for the survival of FMDV in aerosols suggest that spread of FMD in these areas is likely to be less rapid than in areas of more intense livestock production. Since beef cattle are not under the same intensive surveillance as dairy cattle, it is possible that when the first outbreaks are detected, a number of animals in a herd will be infected with consequent greater virus concentrations in the environment. **This makes it important to conduct an analysis of climatic data from typical areas, and further studies on short distance spread of FMD, to define the epidemiology of FMD in these regions.** The role of sheep as modifiers of the spread of FMDV in these areas is not clear. However, the comments noted above also apply to sheep in these areas. That is, infection may be sub-clinical with large numbers of animals infected before FMD is diagnosed with the danger that FMDV may be transferred from one area to another by sheep which are infected but showing no clinical signs.

(iv). Pastoral zone

Stock densities are likely to be low in these areas and correspondingly the chances of spread of FMD lowered because of climatic conditions which are not optimum for virus survival in aerosols. Limited evidence suggests that spread of FMD in areas like the Australian pastoral zone might be minimal. For instance, Mariner *et al.* (1989) surveyed cattle in Niger for FMD after several outbreaks of FMD had been reported and the disease was considered to have become endemic to the region. A prevalence of 1.7% of sera positive for FMD by the AGID test was reported in 234 animals surveyed. Howell and Mansvelt (1972) in examining the situation in South Africa considered that short or long distance airborne spread of FMDV would not be expected to occur in hot and dry countries, since the virus would be inactivated in the presence of low relative humidity and sunlight. However, they noted that animals might become infected by sniffing people, animals or fomites contaminated with virus.

Stock movements may be important in the spread of FMD from the Pastoral zone to other areas and vice-versa. Most of these movements, prior to the BTEC program, were seasonal but are less so know with a well developed transport infrastructure. This allows increased mobility of cattle herds, especially in times of drought when cattle may be moved many hundreds of kilometres. This development has probably increased the likelihood of the spread of FMD from this zone to other areas. The limited surveillance of stock in these areas could allow disease to smoulder unchecked until large numbers of animals have been infected and carrier animals are present. If sold

for fattening, or if moved because of drought, carrier animals might be the source of infection for susceptible stock in other areas. It has been reported that circumstantial evidence suggests that FMD has been disseminated in South America by healthy bovine carriers (anon, 1975). However, Falconer (1972) has reported that experience in Botswana has shown no instances of outbreaks occurring after the release of animals from a quarantine area (and therefore possible carriers) into a susceptible area.

Observations in Africa suggest that FMD may only occur during the dry season in the Pastoral zone. Dawe (1978) found that in Malawi the majority of FMD outbreaks began in the dry season (July - November). The mean relative humidity during this period was 58.2% compared to 77.7% for the rest of the year. The mean temperature for the period was 24.8°C compared to 24.1°C for the rest of the year. Daily sunshine was 9.8 hours versus 7.2 hours. Maximum wind speeds were 8.42 knots versus 4.93 knots. Average monthly rainfall was 1.14 cm versus 15.82 cm. The dry season incidence of disease could be explained by the congregation of cattle and wild game at waterholes at night when proximity and local high relative humidity provided conditions for aerosol transfer of virus. The authors noted that animal movement increased in the dry season and that cattle and buffalo grazed in larger groups facilitating the generation of virus aerosols by diseased herds and the infection of susceptible herds.

With the fencing of many of the grazing areas in this zone following the BTEC program, there is probably a greater degree of control over cattle and a greater opportunity to limit the spread of disease because cattle are held in smaller, managed groups. The provision of permanent stock watering points, resulting from the BTEC program, may reduce the congregation of livestock around limited water supplies and reduce the possibility of transmission of FMD. However, permanent water points, on some properties, may result in greater concentrations of cattle and increased opportunities for contact with wildlife.

(v). The Wet Tropics

Henderson (1960) has noted that there appears to be a definite tendency in the tropics for the virus multiplication sequence to be less rapid, less severe, less dramatic than in colder latitudes. Outbreaks of FMD in Malaysia and Bali tend to confirm this observation with the disease appearing to spread only very slowly between livestock operations. The relative humidity is usually high in the wet tropics, but temperatures are also likely to be high and there is likely to be an increased intensity of sunlight. These factors would tend to reduce the likelihood of airborne spread. In addition, there is usually instability in the atmosphere in these areas for considerable parts of the

year. Such instability would produce increased mixing of the atmosphere and reduce the chances that adequate virus concentrations will occur in the air to permit any spread of infection down-wind.

Feral buffalo are unlikely to play a major role in these areas since the reduction of their numbers in the BTEC program. In areas where populations of feral buffalo exist, there is a small possibility that they could become carriers.

(vi). Areas with high risk enterprises

piggeries/feedlots

Any area in which there are large piggeries will have an increased risk of FMD transmission as pigs act as multipliers of FMDV. Pigs excrete about 30 times that amount of virus that sheep and cattle produce in aerosols. Piggeries are likely to act as massive virus factories, if they become infected, because the spread of infection within the enterprise is likely to be extensive because of the high stock densities and also because the conditions within the buildings housing the pigs are likely to be favourable for survival of FMDV in aerosols. If conditions in the external environment are also favourable for wind-borne spread of FMDV, infection is likely to be spread for considerable distances down-wind. Ventilation exits may become important as highly concentrated sources of FMDV in aerosols. The time taken to slaughter and dispose of the animals in a large piggery may increase the time that virus is available for dispersal. If there are limited areas for carcass disposal at the piggery site, then there may be a need for transportation of carcasses off-site with consequent dangers of dispersal of virus. There are increased dangers if faecal wastes are dispersed by spray irrigation after minimal treatment. Such waste disposal systems are likely to produce virus aerosols and, if climatic conditions are favourable for virus survival, there is likely to be considerable dispersal of virus.

These observations reinforce the need to conduct climatic analyses to identify areas within Australia where air-borne spread of FMDV is likely to occur. There is also a need to investigate ways to prevent infection occurring in housed pigs when an FMD outbreak has occurred in their vicinity. This may require engineering to ensure that virus does not enter buildings with ventilation air.

Feedlots present similar problems to piggeries because of the large concentrations of animals, although probably not to the same extent as piggeries. The lower concentrations of virus excreted in aerosols by cattle will reduce virus concentrations in air and lessen the chances of air-borne spread. However, because animals in feedlots

are not usually housed there will be diffuse dispersion of virus aerosols, unlike piggeries where aerosols will mainly disperse from ventilation exits. The large concentration of animals will increase the chances of animals becoming infected from wind-borne aerosols as there are greater numbers of animals sampling the contaminated air. Slaughter and disposal of animals will produce similar effects as FMD in piggeries, as will disposal of faecal wastes.

areas where there is feral animal/domestic animal contact

The epidemiology of FMD in areas where there are feral and native animals has been discussed previously. Feral pigs may play a potentially important role in the epidemiology of FMD in Australia because of the difficulty of detecting disease in feral populations and their high output of aerosol virus. The role of native fauna is less clear and further investigations are needed into the possibility of FMD being maintained in kangaroos.

(c). Summary

The extent to which FMD may spread in Australia will depend on climatic factors, the efficiency of detection and diagnosis of early cases, livestock movements, stock density and possibly the presence of feral and native animals. Sheep may also play an important role in the spread of FMD in Australia because they may be infected, excrete virus, but remain clinically normal.

Since the respiratory tract is the most efficient route of infection in FMD, survival and transport of aerosols is important in the spread of this disease. Climatic factors influence the survival of FMDV and also the dispersal of infectious aerosols. Stocking density will also influence the spread of disease by allowing more susceptible animals to be exposed to aerosols emitted by an infected animal. Thus in areas where climatic conditions favour survival of FMDV in aerosols and stocking densities are high the disease is likely to spread more rapidly. The reverse applies in areas where the climatic factors do not favour the survival of FMDV in aerosols and stock densities are low. These observations suggest that within Australia there will be a variety of expressions of FMD, ranging from limited spread of the disease to widespread dissemination. How widely FMD might spread under Australian conditions might be determined from a study of meteorological data and modelling of the disease, taking into account stock densities.

It is likely that, in many parts of Australia, climatic conditions will not favour airborne dispersal of FMDV over any great distance and that animal movement will become important in the spread of FMD. Livestock may be moved widely within Australia, usually following specific patterns related to production and marketing options, but occasionally in response to natural phenomena such as drought. A knowledge of such patterns would be useful in formulating options to limit the spread of FMD in the event of an incursion into Australia. Sheep movements may present the greatest risk in the spread of FMD, for the reasons outlined above.

The efficiency of detection and diagnosis of early cases of FMD may be influenced by livestock species and husbandry. There are greater opportunities for detection and diagnosis of FMD in dairy cattle which are individually handled daily, slightly lesser opportunities in housed and feedlot stock, limited opportunities in beef cattle and sheep which may be inspected at irregular intervals, and little opportunity in animals run under extensive conditions pertaining in Northern Australia.

Sheep may be important in the epidemiology of FMD in Australia. Some overseas observations suggest that sheep play little part in the spread of FMD whilst other observations suggest that they may form a reservoir of infection because of their tendency to become infected and excrete virus whilst remaining free of clinical signs. Since there is a large sheep population in Australia, often associated with cattle in some areas, further investigation of the potential role of sheep in the epidemiology of FMD, in climatic and geographic conditions similar to Australia appears warranted.

Feral pigs, buffalo and native fauna may be important in the epidemiology of FMD in some areas of Australia but requires further evaluation.

RECOMMENDATIONS

1. A small preliminary investigation of the possibility of windborne spread of FMD in Australia was carried out by the Bureau of Meteorology in 1982. Relative humidity and winds were tabulated for a small number of coastal sites. This aspect requires further study in Australia with a retrospective study of climatic data to determine, for representative areas of Australia, the probability of sustained wind-borne spread of FMDV during each month of the year. The probability of wind-borne spread of FMDV will influence the planning and utilisation of resources for any FMD outbreak in Australia.

2. Buik (1979) has analysed cattle movements in Australia and noted that they can be used to identify key points in disease transmission such as major source regions of non-slaughter cattle, major recipient regions and the principal sale/purchase points through which the cattle move. Calculation of a contact index is suggested which could provide a weighting, in terms of probability of being infected, for all properties within a region. Sanson *et al.* (1993) examined animal movements in a small area of New Zealand to determine the size of control areas needed to contain the spread of FMD. Analyses similar to these studies need to be carried out for representative areas in Australia to determine whether zoning can be implemented and to investigate the extent of movement controls which would be necessary to control the spread of FMD. There is particular need to document livestock movement patterns in Australia, and sources of information on such patterns, in advance of any outbreak so that basic information is available to rapidly implement analyses similar to those of Sanson *et al.* (1993). The movement patterns of sheep may be particularly important because of the possibility that they may be infected without showing clinical signs and their movement may result in the spread of FMDV to other farms.

3. The movement of animal products within Australia is extensive and may represent a potential source of FMDV during an outbreak. There is a need to assess the risk that FMDV may be spread by animal product movement within Australia. This would need to take into account the survival of FMDV in dairy products and meat and the likelihood of it reaching susceptible livestock. In addition, the handling of animal products originating from an outbreak area needs to be examined. For example, the studies examined in this review suggest that normal pasteurisation of milk may not be sufficient to eliminate FMDV from dairy products, and special processing may be needed to make these

products safe. In addition, the economic implications of restricting the movement of animal products needs examination.

4. The role of sheep in the spread of FMD needs further study. Many authors have observed that sheep often do not develop clinical signs after infection with FMDV but shed virus which may infect other susceptible animals. It has been suggested that FMD may spread slower, or not at all, in sheep raising areas. However, it is difficult to assess the significance of some of the observations. Further study of the role of sheep in the epidemiology of FMD is required, preferably in countries where there are major sheep enterprises.

5. The role of vaccination in combating an outbreak of FMD in Australia needs to examined in view of the potential for the development of carrier animals and the present difficulties of differentiating infected animals from vaccinates. Some analyses of the use of vaccination when FMD has been introduced into a fully susceptible population have demonstrated that, once the disease has reached a certain incidence, vaccination is the most cost-effective option. Most of these models have not included in their analysis the costs of loss of export markets. There would be merit in using or adapting existing models to examine when and where vaccination might be used to aid in the eradication of FMD in Australia.

6. Early studies by Australian workers indicated that there was a wide range of susceptibility to infection with FMDV between Australian fauna. The assessment of the possibility of transmission between domestic animals and native fauna was primarily based on the excretion of virus in lesions, although in one experiment native fauna and domestic animals were run in close contact. It was believed at the time of the work that lesion fluids were the most likely source of virus for FMDV transmission. Later work suggests that the respiratory route is the primary portal of entry of FMDV. While there is, in general, little close contact between native Australian fauna and domestic stock there could arise in certain locations sufficient concentrations of native fauna and domestic stock to allow aerosol transmission of FMD. This is an aspect that probably needs to be further explored.

7. A further assessment of the possible role of feral pigs in the maintenance and spread of FMD in Australia is warranted because of the amount of resources that would be needed to control this disease in feral pig populations. Anecdotal evidence suggests that the role of feral pigs in the epidemiology of FMD is minimal in Europe and South America. While the ecology of feral pigs in Europe is possibly different to that in Australia it would be useful to identify

countries where their ecology is similar to Australia. Their association with FMD in those countries could be further investigated and an assessment of their importance in the epidemiology of FMD made.

8. New Zealand authorities have conducted a risk analysis of the importation of various diseases into their country by travellers and in postal items which is based on traffic flows from various countries, the prevalence of the disease in the country, the risks of spread in New Zealand and the economic costs to the country. Australia should adapt or modify such an analysis to quantify the risks of introducing FMD on a country by country basis by travellers and other means. Such an analysis would be useful in targeting animal quarantine inspections of traveller's luggage and would also provide some indication of the strains of FMDV likely to be imported into this country.

8. It has been observed that concurrent infection with bovine enteroviruses may mask the clinical signs of FMD and the multiplication of FMDV in cattle. While bovine enteroviruses are present in Australia, little is known of the prevalence of such infections. Further studies on bovine enteroviruses might be of use in determining whether masked infection could represent a problem in the diagnosis of FMD.

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