Invited Review

Foot-and-mouth disease: an assessment of the risks facing New Zealand

HJ Pharo*

Abstract

Although New Zealand has never had a case of foot-and-mouth disease (FMD), the threat that this disease poses to the economy of this country has long been recognised. The unprecedented global spread of FMD caused by the type-O PanAsia strain, culminating in the outbreak that occurred in the United Kingdom in early 2001, has refocussed the concerns of biosecurity agencies worldwide. The 3 lines of defence against exotic disease incursions in this country are border controls, surveillance and incursion response capability. This article reviews the pathogenesis, virus survival, routes of infection and methods of spread of FMD virus, and in the light of recent international developments, presents a summary of the major risks of introduction and dissemination of FMD virus and the risk-management measures in place in this country.

KEY WORDS: *Foot-and-mouth disease, FMD, epidemiology, import risk analysis, risk assessment*

Key Points

- International spread of FMD virus is most commonly via the movement of live ruminants and pigs, followed by the movement of meat and meat products.
- The risk of introduction of FMD virus into New Zealand in legally imported animals and animal products is low due to the risk management measures currently in place that have been derived from risk analyses carried out over many years.
- The economic consequences of introduction would be extremely serious.
- The most likely route of introduction into New Zealand is illegally imported meat.
- The most likely outbreak scenario would be infection in pigs as a result of the feeding of infected meat.
- Border controls in this country are among the tightest in the world.
- Surveillance and response measures are aimed at early detection and eradication.

Introduction

FMD is an acute infection of cattle, sheep, pigs, goats, buffalo and many species of cloven-hoofed wildlife, caused by a single-stranded RNA virus belonging to the genus *Aphthovirus*, in the family Picornaviridae. There are 7 distinct serotypes of FMD virus, and within each serotype there are numerous strains (van Regenmortel et al 2000). Infections in humans are very rare and of minor clinical significance (Bauer 1997; Donaldson and Knowles 2001; Prempeh et al 2001).

Sanson (1994) reviewed the epidemiology of FMD, particularly from the point of view of how the disease would spread if introduced into New Zealand and the consequent implications for control. The significance of FMD is related not so much to its clinical effects, although these may be severe in livestock of high genetic merit and in immunologically naïve populations; in general there may be significant production losses and mortality of young animals (Thomson 1995). Rather, for countries that are free from FMD, the main economic implications of its introduction are related to the dramatic effect this would have on international trade. As the virus is highly contagious and may be transmitted by a variety of routes, countries in which the disease is present generally have restricted access to world markets for their animal products. The introduction of FMD into countries that are significant exporters of animals and animal products, such as New Zealand, would result in immediate and severe economic consequences (Davidson 1991).

The emergence of the type-O PanAsia strain

The study of the epidemiology of FMD has been transformed by the introduction of molecular techniques for characterising

* Animal Biosecurity, Biosecurity Authority, Ministry of Agriculture and Forestry, PO Box 2526, Wellington, New Zealand. Email: PharoH@maf.govt.nz

BSE	Bovine spongiform encephalopathy
ELISA	Enzyme linked immunosorbent assay
FMD	Foot-and-mouth disease
HTST	High temperature short time
IETS	International Embryo Transfer Society
MAF	Ministry of Agriculture and Forestry
NCDI	National Centre for Disease Investigation
OIE	Office International des Epizooties
RNA	Ribonucleic acid
$TCID_{50}$	Median tissue culture infective dose
UHT	Ultra-high temperature
UK	United Kingdom

strains of the virus (Kitching 1998), and phylogenetic analysis of virus nucleotide sequences is now the definitive technique for tracing the origins of viruses causing disease outbreaks. RNA viruses in general have very high mutation rates, and FMD virus is notoriously variable (Haydon et al 2000). Serotype O is the most prevalent of the 7 serotypes and occurs in many parts of the world. Within type O, genetic lineages fall into geographically distinct groups known as topotypes. The Middle East, South Asian (ME-SA) topotype comprises a grouping of genetically similar viruses that is endemic to that region, from which the PanAsia strain appears to have emerged (Samuel and Knowles 2001).

Although the exact origin of the PanAsia strain is uncertain, the virus was first identified in northern India around 1990. FMD is endemic on the Indian subcontinent, and approximately 90% of outbreaks there are caused by type-O viruses (Hemadri et al 2000). The PanAsia strain spread westward into Saudi Arabia and then through the Middle East, where it has appeared in most countries and has now largely replaced all other strains previously circulating (Samuel et al 1997). By 1996 it had reached Turkey, and spread into Greece and Bulgaria (Knowles et al 2001). During the 1990s the virus also moved eastward, being reported successively from Nepal, Bhutan and China. In 1999 it reached Taiwan, where only 2 years earlier a pig-adapted strain of type-O virus had devastated the Taiwanese pig industry. In early 2000, outbreaks due to the PanAsia strain were reported in the Republic of Korea and Japan. Later that year there were outbreaks in Russia, Mongolia, and South Africa, followed in February 2001 by the arrival of the virus in western Europe, first appearing in the United Kingdom (UK) and spreading from there to the Republic of Ireland and continental Europe (Knowles et al 2001).

Occurrence of virus in infected tissues, secretions and excretions

In domestic ruminants primary replication of FMD virus takes place in the mucosa of the pharynx and dorsal soft palate (Thomson 1997), and in pigs the pharyngeal, tracheal and nasal mucosa are the most important sites for both initial replication and aerosol generation (Alexandersen et al 2001). Spread from these sites occurs via the lymphoid system, resulting in a viraemia whereby the virus reaches a wide variety of organs and tissues where further replication gives rise at some sites to lesions characteristic of FMD (Thomson 1997).

The appearance of vesicles coincides with the peak of viraemia and the highest concentrations of virus in tissues where vesicles develop. The rupture of vesicles on the tongue and in the mouth results in the production of copious quantities of heavily contaminated saliva. The time from infection to this point can vary from 2–14 days, depending on the strain and dose of FMD virus, and the route of infection (Sanson 1994). The range of titres of FMD virus found in tissues of pigs and cattle at various stages of infection are shown in Table 1. The titres found in sheep are similar to those in cattle (Sellers 1971).

High concentrations of virus may occur in organs and tissues that do not generally develop gross lesions, such as lymph nodes, adrenal glands and skin of unaffected areas (Alexandersen et al 2001). There is also considerable replication in mammary tissue, so that milk from infected cattle may contain high titres of virus for up to 4 days before appearance of clinical signs (Burrows 1968). The same is true for semen (Sellers et al 1968), and peak titres in both semen and milk are seen on the day that clinical signs first become apparent (Sellers et al 1968; Burrows et al 1971).

The amount of virus excreted per rectum is small, especially in pigs. Fresh faeces collected from the floor have been found to contain small quantities of the virus for up to 12 days after infection in the case of cattle and 10 days for pigs. However, for sheep, only 1 faecal sample contained virus, on Day 2 of an 8-day observation period. As only 16% of rectal swabs from cattle and pigs were positive for virus, it is likely that infectivity in faeces was confounded by contamination with saliva (Parker 1971). Since urogenital secretions, particularly those of the prepuce, may contain high levels of virus it is possible that virus in urine may be the result of contamination by such secretions (Thomson 1994).

Table 1. Foot-and-mouth virus titres in different tissues, secretions and excretions.

Animal	Tissue/excretion	Stage of disease	Titre	Reference
Pig	skin (histologically normal)	pre–clinical (1–4 days post–exposure)	10^9 $TCID_{50}$/g	Alexandersen et al 2001
Pig	pharynx (soft palate, tonsil, floor of pharynx)	pre–clinical (1–4 days post–exposure)	10^5 –10^6 $TCID_{50}$/g	Alexandersen et al 2001
Cattle	vesicular epithelium	peak clinical signs	$10^{9.6}$ $TCID_{50}$/g	Hyslop 1965a
Cattle	saliva	several hours before clinical signs	10^2 –$10^{3.75}$ $TCID_{50}$/ml	Hyslop 1965a
Cattle	saliva	peak clinical signs (copious production of saliva)	$10^{5.25}$ –$10^{8.5}$ $TCID_{50}$/ml	Hyslop 1965a
Cattle	milk	pre–clinical (up to 4 days before clinical signs)	$10^{6.6}$ $TCID_{50}$/g	Hyslop 1970
Cattle	semen	peak clinical signs	$10^{6.2}$ $TCID_{50}$/ml	Sellers et al 1968
Cattle	heart muscle adrenal retropharyngeal lymph node blood liver	peak clinical signs	$10^{10.0}$ pfu/g $10^{10.6}$ pfu/g $10^{8.2}$ pfu/g $10^{5.6}$ $TCID_{50}$/g $10^{3.6}$ $TCID_{50}$/g	Burrows et al 1981
Cattle	skin	up to 5 days after cessation of viraemia	$10^{3.6}$ pfu/g	Gailiunas and Cottral 1966

In ruminants, FMD virus may persist for long periods in the pharyngeal mucosa, but it does not persist in this tissue in pigs (Thomson 1997; Moonen and Schrijver 2000).

Virus survival in animal products and the environment

FMD virus is most stable at near-neutral pH and is sensitive to even mild acidity. Although temperatures above 50°C destroy most infectivity, it is likely that a small proportion of particles are relatively resistant to the effects of heat and pH in most populations (Hyslop 1970). The effect of temperature and pH on time for 90% virus inactivation is shown in Table 2.

FMD virus is very sensitive to desiccation. In aerosols it survives best when the relative humidity exceeds 70%, and there is a critical humidity level of 55–60%, below which survival is poor (Sellers 1971). Sunlight and ultraviolet radiation have little effect on virus persistence (Donaldson and Ferris 1975).

Since faeces are approximately neutral in pH, the survival of virus in slurry depends largely on temperature and humidity (Parker 1971). As long as the humidity remains above 55%, temperature is the major determinant of virus survival; the lower the temperature, the longer the survival. Russian experiments conducted in the early 20th century apparently demonstrated the survival of virus in frozen or liquid manure for periods of more than 6 months in winter (Cottral 1969), but it is not clear how long infectivity persists in dry faecal material under different environmental conditions.

Routes of infection and methods of spread

Transmission of FMD virus most commonly occurs during physical or close contact between acutely infected and susceptible animals, often following the movement of infected animals. The next most common transmission pathway is the consumption of contaminated animal products such as meat, offal, or milk which may be fed to pigs or to calves (Donaldson et al 2001).

It is generally accepted that primary infection of ruminants is usually by the respiratory route, whereas for pigs it is usually by the oral route (Donaldson and Alexandersen 2001), which reflects differences between these animal types in the dose of virus required to establish infection via the respiratory route and in the exposure pathways by which virus is usually introduced into their environments. The infectious dose of virus by the oral route is similar for cattle and pigs, being 10^6 $TCID_{50}$ and 10^5 $TCID_{50}$, respectively. However, the dose required to infect cattle and sheep by the respiratory route, about $10^{1.0}$ $TCID_{50}$, is considerably less than the $10^{2.6}$ $TCID_{50}$ required for pigs (Donaldson 1997).

The amount of airborne virus produced by infected animals varies with strain of virus, stage of infection and species of animal (Sanson 1994). Pigs are the most potent emitters of airborne virus, producing up to $10^{8.6}$ $TCID_{50}$ of airborne virus/pig/day (Donaldson and Ferris 1982), more than 1500 times the $10^{5.4}$ $TCID_{50}$ of airborne virus/animal/day produced by cattle and sheep (Sellers 1971).

The sensitivity of cattle to infection by the respiratory route and the high levels of airborne virus produced by infected pigs raises the possibility of spread of the virus to cattle as a result of aerosols of virus generated by infections in piggeries located upwind (Donaldson 1983). Airborne spread over land has been considered important within the UK during past outbreaks (Smith and Hugh-Jones 1969; Hugh-Jones and Wright 1970). However, it is probably not a common event as it would require the simultaneous occurrence of specific epidemiological and climatic conditions, including a large number of infected pigs at the same stage of infection, a constant wind direction at a steady speed of at least 5 m/sec, a high degree of atmospheric stability, no precipitation, a relative humidity above 55%, and no obstacles such as hills, mountains or urban areas (Donaldson et al 2001). It is generally considered that airborne spread is probably restricted to distances up to about 10 km under most circumstances (Sanson 1994).

In the case of the PanAsia strain, the potential for airborne spread appears to be very limited (Brownlie 2001). Using one of the UK 2001 isolates, the amount of airborne virus recovered per pig over a 24 h period was $10^{6.1}$ $TCID_{50}$, 300 times less than the peak output obtained from other strains of the virus. Using this level of airborne virus production from pigs, computer modelling suggested that pigs would not be at risk from airborne spread from any species, and that cattle would be at risk of infection only up to 6 km downwind of a pig herd that contained at least 1000 infected animals (Donaldson et al 2001). Although short distance airborne spread due to virus dispersal from the burning of animals

Table 2. The effect of temperature and pH on time for 90% foot-and-mouth virus inactivation[a].

Effect of temperature (at pH 7.5)		Effect of pH (at 4°C)	
Temperature	**Inactivation time (90%)**	**pH**	**Inactivation time (90%)**
61°C	30 seconds	10.0	14 hours
55°C	2 minutes	9.0	1 week
49°C	1 hour	8.0	3 weeks
43°C	7 hour	7.0–7.5	>5 weeks
37°C	21 hour	6.5	14 hours
20°C	11 days	6.0	1 minute
4°C	18 weeks	5.0	1 second

[a] data extracted from Bachrach et al (1975)

in open pyres was suspected following the 1967–68 UK outbreak (Smith and Hugh-Jones 1969), an analysis of outbreaks around pyres during the 2001 UK outbreak concluded that such spread was unlikely (Gloster et al 2001).

The possibility of long distance airborne spread of FMD virus, particularly over water, was raised on a number of occasions prior to the 1967–68 UK outbreak (Hyslop 1965b; Hurst 1968), and computer models were subsequently developed to assess the risk of airborne spread from Europe to the UK (Gloster et al 1982). The most convincing support for long distance airborne spread arose through the use of such models immediately prior to the outbreak on the Isle of Wight in 1981, when it was predicted that spread from infected piggeries in Brittany was likely – a distance of 250 km (Donaldson et al 1982). This notwithstanding, recent re-simulation of this event using a more sophisticated and accurate atmospheric dispersion model has suggested that the amount of virus arriving at the Isle of Wight would have been 500 times lower than the threshold value considered necessary to initiate infection in cattle, and that airborne spread could have been responsible only if there had been substantial under-reporting of the number of infected pigs in Brittany: only 63 pigs were reported with clinical signs on the 2 critical days, and the model suggested that at least 1500 affected pigs would have had to be present for sufficient aerosolised virus to have been generated (Sørensen et al 2000).

Most of the work on the persistence of FMD in the environment was carried out early in the 20th century, at a time when FMD was endemic in most of Europe and local spread between farms was of primary concern. Potential contamination of fomites and feedstuffs, including concentrates, hay and straw, by saliva, faeces and urine, were considered responsible for a certain amount of spread (Parker 1971). Saliva of infected animals contains large quantities of virus, and while the amount of virus in faeces and urine is probably variable but low, the intensification of animal production raises questions regarding how barn wastes can be safely handled in an outbreak, in particular when it might be safe to spread stored manure onto pasture as fertiliser (Sellers 1971). While it is somewhat difficult to imagine how mechanically harvested hay and straw could become contaminated with the virus under natural conditions, opportunities for contamination may exist in certain countries where fodder is cut by hand and transported by infected draft animals. However, until further work is undertaken to clarify the likelihood of transmission via such items, it is difficult to assess the importance of these transmission pathways.

Thirty years ago, Cottral (1969) summarised a large number of papers on virus persistence, most of which were in languages other than English and therefore relatively inaccessible in the West. It is not completely clear which of those papers relate to experiments and which result from studies carried out under field conditions. Little has changed since Hyslop (1970) summarised the past research as follows:

> "The observations of numerous investigators during the past 60 years indicate that virus secreted in the saliva of infected animals may remain viable for up to 2 days at 37°C, 3 weeks at 26°C, and for 5 weeks at 4°C, while Russian workers have claimed that virus in animal excretions may remain detectable inside a contaminated building for at least a month during warm weather and for longer than 2 months during the winter. Reports dating from the second and third decades of this century suggest that virus may occasionally survive on wood, hay, straw, etc., for about 15 weeks."

Routes of recent international spread

While it is widely accepted that the majority of spread of FMD virus is by the movement of infected animals and their products (Cottral 1969), the importance of other potential routes of transmission is less clear. Since primary infection of ruminants is usually by the respiratory route, while for pigs it is usually by the oral route, it is generally accepted that if the index case is in pigs then the most probable source was some sort of animal product which found its way into their feed. On the other hand, if the index case is in ruminants then the movement of infected animals is usually considered to have been responsible. Within endemic regions of the world both of these major routes can be important, whereas for countries which experience sudden outbreaks after long periods of freedom, the source of the virus is frequently attributed to feeding waste food to pigs. For example, the most likely source of infection for the 1967–68 UK epidemic was meat from South America, but even during the period when FMD was endemic in the UK, more than half of the outbreaks (from 1938–1953) were associated with waste food (Donaldson and Doel 1992).

Nevertheless, it is often not possible to determine the exact source of a particular introduction because of incomplete information, especially where the feeding of illegally imported meat is concerned. Therefore, consideration of the origin of a virus responsible for a new outbreak usually requires piecing together whatever often circumstantial evidence can be found after the event, and increasing reliance is now placed on molecular methods to determine relationships with other known viruses (Samuel and Knowles 2001).

The pig-adapted virus that caused the devastating 1997 outbreak in Taiwan, the first since 1929 (Yang et al 1999), was not the PanAsia strain, but a virus belonging to the Cathay topotype, and was genetically very similar to viruses seen earlier from outbreaks in Hong Kong and the Philippines (Kitching 1999). The introduction of this virus to Taiwan was considered most likely to have resulted from smuggling meat or live animals from China via fishing boats (Ogawa and Matsuda 2000; Tsai et al 2000).

When the PanAsia strain appeared in Taiwan in February 1999, it was first reported on Kinmen Island, which is very close to the Chinese mainland. Since initial infections were in native yellow cattle, which did not show obvious clinical signs, by the time the disease was recognised, cattle had been moved to the main island of Taiwan where the virus caused typical FMD in dairy cattle and severe disease in goats (Knowles et al 2001).

The PanAsia strain was responsible for outbreaks in Siberia and Mongolia in April 2000. The Siberian outbreak occurred only in pigs and it was thought most likely to have resulted from the feeding of contaminated meat from the port of Vladivostock or from contaminated vehicles crossing the border with China (Garner 2000). In the case of Mongolia, which had been free of FMD since 1974, a range of ruminant species in 26 communally-grazed herds was affected, most likely as a result of illegal movements of livestock from China (Garner 2000).

The September 2000 outbreak in South Africa, which was the first outbreak in domestic livestock there since 1956, was attributed to the illegal feeding of pigs with garbage from a ship which origi-

nated somewhere in Asia (Mogajane 2000; Knowles et al 2001). The very close genetic match between the South African virus and the virus that appeared in the UK in February 2001 suggests a strong link if not a common source (A Samuel, pers. comm.)[1]. In the case of the UK epidemic, the index case was a swill-feeding piggery, and considering the very large quantities of meat that is illegally imported into the UK, and the limited border controls in place (Anonymous 2001), there is a strong possibility that illegally imported meat was the vehicle.

However, with much of the recent spread of the PanAsia strain in Asia it has not been possible to identify the route of introduction (Garner 2000). In late March 2000, South Korea experienced its first outbreak of FMD since 1934, within 5 km of the demilitarised zone. All outbreaks were in cattle (mostly beef cattle), and several routes of introduction were suspected (Anonymous 2000). Similarly, when FMD was reported in May 2000 in cattle in Japan (the first outbreak in Japan since 1908), several possible routes of introduction were suspected but the Japanese authorities were only able to conclude that the most likely vehicle was imported Chinese straw (Sugiura et al 2001).

Risk of introduction of FMD virus into New Zealand

Airborne spread

The longest distance claimed for airborne spread is 250 km from Brittany to the Isle of Wight in 1981, and that was apparently possible only under precise meteorological and epidemiological conditions. Therefore, for an isolated island nation such as New Zealand, the risk of introduction of FMD virus by the airborne route is negligible. Nevertheless, if a suitable strain of FMD virus was introduced into large piggeries in New Zealand, under certain circumstances the airborne route might be responsible for some spread within this country.

Live animals

The risk posed by movement of live animals from countries or zones that are not free from FMD has long been recognised for both regional and international spread of the disease (Cottral 1969). Large amounts of the virus are present in the secretions and excretions of preclinical infectious and clinically affected animals. Cattle and sheep may be sources of the virus for up to 5 days prior to the development of clinical signs, and pigs may harbour the virus for up to 10 days before clinical signs appear (Burrows 1968).

Persistent infection in the mucosa of the soft palate, pharynx and cranial oesophagus occurs in cattle, sheep, goats, buffalo, and various wild ruminant species, but apparently not in pigs (Thomson 1994). Approximately 50% of cattle infected with FMD virus become carriers, irrespective of vaccination status. Sheep and goats may remain carriers for up to 9 months, cattle for as long as 2 years, and African buffalo for more than 5 years (Moonen and Schrijver 2000). However, the role of carrier animals in the epidemiology of FMD is unclear. There is field evidence that carrier animals may play a role in the spread of the virus, especially if they are incompletely immunised (Moonen and Schrijver 2000; Brownlie 2001), but there are no published accounts demonstrating transmission of FMD from carrier to susceptible animals under controlled conditions (Barnett and Cox 1999; Moonen and Schrijver 2000).

Because of the risk of introducing FMD virus in preclinical, clinical or recovered animals whatever their vaccination status, New Zealand does not import susceptible live animals from countries that are not free from FMD. Therefore the risk of introducing FMD virus in legally imported live animals is negligible.

Genetic material

The International Embryo Transfer Society (IETS) has classified FMD as a Category-1 disease, meaning that the risk of transmission via embryos is considered to be negligible provided the embryos are handled according to IETS protocols between collection and transfer (IETS 1992). However, cattle semen may contain high titres of the virus from several days prior to the presence of clinical signs until 10 days post-infection, and heifers inseminated with semen containing the virus may become infected (Cottral et al 1968). Therefore, New Zealand does not import genetic material from countries that are not free from FMD, unless a risk analysis concludes that the risks can be managed effectively using specific safeguards.

Meat and meat products

It is well accepted that meat from animals which are viraemic at the time of slaughter may harbour FMD virus, and many outbreaks of FMD have been traced to the feeding of waste food containing meat scraps to pigs. The New Zealand Ministry of Agriculture and Forestry (MAF) has examined the FMD risks posed by meat in general (MacDiarmid 1991) and sheep and goat meat in particular (MacDiarmid and Thompson 1997).

Following death, FMD virus is inactivated rapidly in skeletal muscle and heart muscle as a result of lactic acid formation that accompanies rigor mortis, through which the pH falls to 5.5–6.0. The inactivation rate for FMD virus at pH 6 is 90% per min, while at pH 5 it is 90% per sec (Bachrach et al 1975). Thus, in skeletal muscle kept at 4°C the virus is considered to be completely inactivated within 48 h, although infectivity may remain for over 4 months in lymph nodes, clotted blood, bone marrow and viscera where the virus is protected from pH changes (Blackwell 1984). Moreover, the muscle pH of animals slaughtered in the febrile state may not fall below 6.0, and some virus may be present in muscle tissue of such animals up to 96 h post-infection (MacDiarmid and Thompson 1997).

The International Animal Health Code of the Office International des Épizooties (OIE) recommends that the importation of boneless chilled or frozen beef from countries in which FMD is present may be permitted safely, provided the meat originates from deboned carcasses from which the major lymph nodes have been removed. Prior to deboning, carcasses have to be matured at a temperature above 2°C for at least 24 h and the pH must be below 6.0 when tested in the middle of the longissimus dorsi muscles. However, there are a number of reasons why the likelihood of sheep being viraemic at slaughter is greater than for cattle, and since it is generally considered impractical to debone sheep and goat carcasses prior to export, the recommendations of the OIE code are not considered applicable to these species.

Processing of meat to inactivate FMD virus is either by cooking to an internal core temperature of 70°C for 30 min, or by curing

[1] Alan Samuel, Institute for Animal Health, Pirbright, UK

at low pH. For example, the low pH of lactic-cured sausages, such as salamis, ensures that FMD virus is inactivated in a week, even if such products are made with meat of viraemic animals (MacDiarmid and Thompson 1997).

However, the level of protection that is considered appropriate for New Zealand with respect to FMD virus is apparent from the position this country takes not to import meat or meat products from countries not free from FMD unless the meat is cooked or cured using approved processes.

Milk and milk products

In a risk analysis on FMD in milk and dairy products, Donaldson (1997) noted that unpasteurised milk is a well-recognised vehicle for the spread of FMD, particularly during outbreaks and especially through the feeding of raw milk from infected animals to pigs. The disease risks posed by international trade of dairy products were subsequently analysed from a specifically New Zealand perspective by Christensen (1998).

Virus shed from infected mammary glands has been shown to be incorporated into milk micelles and fat droplets, which afford some protection against heat inactivation. In addition, there may be a heat-resistant fraction of the virus present in milk such that low titres of infectivity have been found in whole milk after heating at 72°C for 5 min, and in skim milk after 2 min at that temperature (Blackwell and Hyde 1976).

Low concentrations of infectivity have been found in milk after high temperature short time (HTST) pasteurisation at 72°C for 15 sec, or after acidification to pH 4.6 (Pirtle and Beran 1991). Despite this, the risk posed by HTST pasteurised milk is considered to be negligible, since for there to be a high probability of infection, a single pig would have to drink between 125 and 1,250 litres or inhale 500 to 5,000 ml of HTST pasteurised milk. Similarly, for a high probability of infection a calf would have to drink between 1,250 and 12,500 litres, or inhale between 12.5 ml and 125 ml of such milk (Donaldson 1997).

Products made from raw milk are recognised as potential vehicles for the spread of FMD. For example, the virus may survive for up to 2 months in dried casein. However, its survival in cheese made from raw milk depends on the pH achieved during manufacture; if the pH drops to 4.0, the virus is inactivated in seconds, while in cheeses which have a final pH of 6.0 the virus will not survive longer than 30 days. In cheeses that are cured at temperatures of not less than 2°C, the virus will not survive more than 120 days (Christensen 1998).

New Zealand's very cautious approach to the risk of FMD introduction is reflected by the policy that dairy products may be imported from countries that have not been free from FMD for a period of 12 months only if they are made from milk that has been subjected to one of the following treatments prior to being used for manufacture: double HTST, HTST plus another treatment such as ultra-high temperature (UHT) or UHT treatment plus another treatment such as pH<6.0 for at least 1 h.

Meat and dairy products in passenger luggage and mail

It has been recognised for many decades that meat and dairy products carried on ships or aircraft are a potential vehicle by which various animal disease agents could be introduced into this country, and that if such products were discarded into garbage that was subsequently fed to pigs, outbreaks of disease could result. Therefore, ever since the last outbreak of swine fever in New Zealand, which occurred in 1953, all garbage from arriving ships and aircraft must be either incinerated or steam sterilised (Davidson 1991).

Due to the risks posed by animal or plant disease agents in passenger luggage, incoming passengers are required on arrival to declare goods of plant or animal origin, and any restricted items found by border staff are seized. In the year 2000, approximately 3.5 million passengers and crew arrived in New Zealand by air, and another 20,000 arrived by sea. Air passenger numbers have been doubling every 7 years (Whyte 2001).

Periodic random surveys of passengers are carried out to estimate the amounts of various goods (slippage) that are missed by normal MAF processing through international passenger arrivals. In 1996 the estimated quantity of meat slippage for New Zealand was 7.7 tonnes/annum. Following the 1997 incursion of fruit fly into Auckland, X-ray machines for passenger luggage were introduced to the 3 largest international airports (Auckland, Wellington and Christchurch). At the same time the use of detector dog teams ('sniffer dogs') was initiated in arrival halls. Although the number of X-ray machines available at each of these airports meant that not all arriving baggage could be examined, the effect of these measures was that 85% of meat products were being detected by MAF systems, and annual slippage of meat products was estimated at 1.6 tonnes (C Whyte, pers. comm.)[2]. Passengers arriving by sea may present a small additional risk.

As a result of the public and political concern generated by the FMD epidemic in the UK in 2001, further funding was made available by the New Zealand government in early 2001 to enable the deployment of an extra 9 detector dogs and 11 X-ray machines, with the effect that now all baggage arriving at international airports in New Zealand is either X-rayed or hand searched. There is now also close to 100% X-ray of arriving mail (Whyte 2001).

Meat slippage now is limited to whatever may be carried on the person, as well as very small quantities that escape detection, or meat products that are so dry that X-ray imaging is ineffective. The recent imposition of $200 instant fines for passengers found with undeclared risk goods is intended to further discourage passengers from bringing such materials into this country.

New Zealand's very tight border controls are comparable to those in Australia, where, also in response to the 2001 UK outbreak, additional government funding of almost half a billion dollars has recently been made available for further detector dog teams and machines (Garner et al 2002). However, they stand in stark contrast to controls present in the UK. Indeed, until recently this route of introduction appears to have been given little consideration in assessing the FMD risk to the UK (Donaldson and Doel 1992). Approximately 67 million passengers arrive at UK airports per year (Department of the Environment, Transport and the Regions 2000), and huge quantities of illegally imported meat arrive regularly in their luggage, apparently with few, if any, border controls in place. Over an 8-month period in the year 2000, a total of 14 incoming flights from Africa were searched by UK customs officials, revealing a staggering 5.5 tonnes of meat and fish in passenger luggage. Moreover, these amounts in personal luggage are said to be eclipsed by the organised smuggling of 'bush meats' and

[2]Carolyn Whyte, MAF Quarantine Service, Auckland, New Zealand

apparently deliberate misdeclaration of cargo shipments into that country (Anonymous 2001).

Even prior to the considerable tightening of border measures in New Zealand following the recent UK epidemic, the small quantity of meat slippage items entering this country (less than 5 kg/day) and their type (generally small quantities of processed or cooked meat for personal consumption), meant that there was little likelihood of any being fed to pigs and the FMD risk was already relatively low. Although it was widely perceived that the risk had increased as a result of the recent spread of the PanAsia strain, the additional measures that were introduced in April 2001 had the effect of reducing the amount of illegal material now entering the country with air passengers to a very low level, and the resulting risk of FMD introduction through such material must now be considered to be minimal.

Carriage by humans

The risk of humans spreading infection between animals as a result of contamination of their hands and clothing, especially by saliva from infected animals, has long been recognised (Sellers 1971). However, a series of experiments carried out 30 years ago at the Animal Virus Research Institute in Pirbright raised the possibility that humans might be able to spread the virus more widely via their nasal passages, even after taking standard precautions including showering and changing clothing. The first set of experiments indicated that it is possible for humans examining the head area of clinically affected pigs to harbour the FMD virus in their nasal cavity, but the longest duration of carriage reported in 1 person was 28 h (Sellers et al 1970). Further experiments showed that infection can occasionally be transmitted by humans to cattle by "sneezing, snorting, coughing and breathing" for 30 sec into the mouths of susceptible steers within 15 to 20 min of examining clinically affected pigs (Sellers et al 1971). The significance of this in terms of the risk of international spread is difficult to evaluate. However, this is presumably where the concept of the "stand-down" period comes from for humans involved with infected animals in an outbreak situation, but how the stand-down period came to be set at 7 days (commonly used for disease control personnel) is less clear. The 7-day period is probably conservative, and it may be appropriate for people actually examining infected pigs, but for people that have been involved with sheep and cattle the risk is presumably less. In the UK over the course of the recent outbreak, the stand-down period was gradually reduced from 7 days to 48 h, presumably for logistical reasons (G Mackereth, pers. comm.)[3]. It is concluded that the risk of introduction of FMD virus on humans or their clothing is remote.

Hides and skins

FMD virus has been found in the skin of steers from all areas of the body, irrespective of the presence of hair. In skins the virus persists for as long as 5 days after cessation of viraemia, at titres of up to $10^{3.6}$ pfu/g of skin (Gailiunas and Cottral 1966). FMD virus may survive for up to 352 days in fresh hides, depending on temperature, and is capable of surviving in salted hides for up to 46 days at ambient temperature (Cottral 1969). It may also be found in dry-salted hides and skins (Gailiunas and Cottral 1966).

A MAF risk analysis on imported hides and skins concluded that FMD virus has the potential to be associated with hides and skins of animals that were infected at the time of death (Pharo, unpublished). The addition of 2% sodium carbonate to salt has been shown to inactivate all virus in heavily contaminated ox hides, provided they are stored for 4 weeks (Schjerning-Thiesen 1972). On that basis, the OIE International Animal Health Code recommends as a safeguard against FMD virus the treating of hides by salting for at least 28 days in sea salt containing 2% sodium carbonate. This raises the pH in the salt to at least pH 9.5, a level that is lethal to the FMD virus. Furthermore, the extreme pH levels attained during processing of hides and skins, particularly liming (pH 12.5–13) or pickling and tanning (pH<3), would result in rapid inactivation of any FMD virus present (Bachrach et al 1975).

The MAF risk analysis concluded that the risk of introducing FMD on or in imported pickled pelts or 'wet-blue' (chromium-tanned) hides is negligible, and that provided the OIE recommended standards are followed, and provided the hides are stored for at least 4 weeks, then even if the hides are derived from viraemic or recovering animals, the likelihood of introducing FMD virus with hides and skins imported into this country is very low. Moreover, since the risk of exposure of FMD virus on imported hides and skins to susceptible animal species in this country is remote, the risk analysis concluded that the risk of introduction was negligible.

Wool

In a MAF analysis of the biosecurity risks posed by the importation of unscoured wool (Pharo 1998), it was concluded that despite the scarcity of studies carried out under natural conditions, it is theoretically possible for FMD virus to be present on unscoured wool as a result of contamination by saliva of infected animals, and perhaps by urine or faeces. It was considered that within a wool bale the humidity (approximately 60%) and pH (pH 6–9, depending on wool type) would be conducive to virus survival, and that the duration of survival would therefore depend largely on temperature.

An experiment carried out in Kazakhstan in 1952–53 showed that the survival of FMD virus on the wool of sheep was dependent on air temperature. In winter, when the mean 24 h temperature was around minus 6°C, the virus survived between 15 and 20 days. In summer, when the mean 24 h temperature was from 12–22°C, the virus was inactivated within 3 days (Gizitdinov 1957). Another experiment carried out in central Asia in 1968 concluded that the virus will survive on wool for only 3–8 days in spring/summer conditions (Voinov 1968). Studies carried out at Plum Island in the USA in the 1970s found that FMD virus on wool stored at 21°C survived for 7 days but not for 14 days (Eisner and McVicar 1980). In a more recent Australian study, wool that was contaminated with faeces, urine or blood from infected animals was found to contain detectable quantities of the virus for 5–11 days at 18°C. The same study demonstrated that scouring contaminated wool at 60–70°C resulted in inactivation of FMD virus (McColl et al 1995).

The MAF risk analysis accepted the recommendation of McColl et al (1995) that storage of wool for 4 weeks at temperatures of 18°C or higher or scouring of wool at 60–70°C would be sufficient to remove whatever threat was posed by possible FMD virus contamination of imported greasy wool. It was also considered, similarly to imported hides and skins, that the risk of exposure of any infectious agents on imported wool to susceptible animals in this country is extremely remote.

---

[3] Graham Mackereth, NCDI, Wallaceville, New Zealand.

Other possible routes of introduction

Notwithstanding the many reports of survival of FMD virus on fomites (summarised by Cottral 1969), there is still considerable uncertainty regarding the length of time FMD virus might survive on clothing, shoes, and other inanimate objects following various forms of contamination. Although there is no evidence that contaminated fomites have ever been responsible for international spread of FMD, the rational assessment of risks in the face of such uncertainty is difficult, and when exposure is involuntary and the consequences are high, there is generally a natural tendency towards risk aversion (Slovic 1999). The widespread public anxiety at the height of the UK epidemic led to speculation that a range of items from used agricultural machinery to ships' ballast water might harbour FMD virus. Several countries even banned the importation of horses from the UK and Europe for fear that infectivity might be carried on soil attached to their hooves, and in many countries tourists disembarking from international flights were required to walk through disinfectant footbaths, creating the perception of the international public that there was indeed a risk that warranted addressing.

Although no formal analysis of the risk of introduction of FMD virus on hooves of animals or fomites has been undertaken by MAF, in line with many other countries New Zealand has traditionally taken a conservative approach. A lack of commercial incentives means that there has never been interest in importing hay or straw into this country, and the small quantities that are imported with live animal shipments are destroyed on arrival for plant quarantine reasons. Since the majority of international horse traffic to NZ is via Australia, and since most countries that have endemic FMD also have equine diseases of international trade significance, prior to the 2001 UK outbreak MAF had not been faced with the question of whether it was safe to import horses from countries with endemic FMD. For horses imported from the UK and Europe, MAF considered that the 30-day pre-export quarantine period, combined with washing of hooves, was enough to mitigate the risk of introducing FMD virus. Similarly, at international airports the footwear of passengers arriving from the UK was given special attention as a precautionary measure.

Finally, in view of rising international concern that agriculture could be targeted for economic sabotage by terrorist groups, the possibility of deliberate introduction of FMD virus cannot be ruled out. No formal analysis of this risk has been carried out by MAF, but the case of rabbit calicivirus showed that "a well-planned, organised criminal event" is extremely difficult for biosecurity authorities to prevent, even with enhanced border security (Wilson et al 2000). The same applies to the possibility of introduction of the virus through the smuggling of live animals or animal products. However, the combination of New Zealand's geographical isolation, the economics of its animal production systems and the rigour of its biosecurity systems means that there are few incentives to smuggle FMD-susceptible animals or their products from endemically infected countries. Nevertheless, if FMD virus was introduced, the response would be the same regardless of how the introduction occurred.

Surveillance and response

The surveillance and response systems that are in place for vesicular diseases in New Zealand are largely unchanged since reviewed by Sanson (1994). Primary surveillance continues to be provided by farmers and/or veterinary practitioners. A freephone number is now available to report any suspected cases, which result in an initial investigation by an approved veterinarian. In the case of a 'not-negative' diagnosis, an investigation team is sent from MAF's National Centre for Disease Investigation (NCDI) (Thornton 1999) to carry out a full epidemiological assessment and to collect appropriate samples for submission to the national reference laboratory. The NCDI reference laboratory has the capability to carry out initial screening for FMD with an antigen capture ELISA, and confirmatory testing would be carried out by the World Foot-and-Mouth Disease Reference Laboratory at Pirbright, UK. In the case of an outbreak of FMD in this country, an eradication policy would be followed, involving depopulation of infected herds and epidemic management based on the EpiMAN decision support system (Sanson et al 1999).

One significant change in New Zealand's biosecurity arrangements over the past decade is that there is no longer any regulation of garbage-feeding piggeries. In 1997 MAF reviewed its existing regulatory framework, which comprised the registration of garbage-feeding piggeries and the issuing of treatment licences to those piggeries feeding garbage containing any meat. It was concluded that the funding required to make the system function as originally envisaged was not available. One of the major costs of regulation was the maintenance of an up-to-date database of pig producers, and the review considered that the risk of introduction of animal disease in garbage lay more with small (unregistered) backyard pig units rather than large commercial (registered) piggeries. The review concluded that any FMD risk from garbage was likely to be associated with uncooked animal products, and since all legally imported meat was from FMD-free countries, it was argued that the real risk lay only with piggeries that had access to smuggled animal products. The risk to large commercial piggeries was judged to be minimal because the sources of garbage to such units were generally hotels, supermarkets and institutional kitchens, which were considered unlikely to be users of illegally imported meat. The review further concluded that the risk of exotic disease incursion could be managed more cost-effectively by tightening border controls to minimise the illegal importation of meat, and as is discussed above, this was the course of action taken. Nevertheless, as a result of concerns raised in several quarters following the 2001 outbreak of FMD in the UK, a further review of garbage-feeding piggeries has recently been initiated in this country.

Conclusions

New Zealand has long recognised its economic vulnerability to FMD, and it would not be an exaggeration to say that the exclusion of this disease has always been the primary focus of the state veterinary service. Systems to meet that end were designed over many years in the full knowledge that the virus remains endemic in Asia, Africa, the Middle East and South America. The emergence of the PanAsia strain illustrates that it is possible for new and significantly different FMD viruses to appear, and it is difficult to predict the impact that any new strain will have on world animal health. This notwithstanding, it remains clear that ruminants are most likely to be exposed to direct infection, such as by infected animals or semen, and pigs are most likely to be exposed by ingestion of contaminated animal products. New Zealand's isolation makes it an unlikely destination for smuggled live ruminants

and pigs, and given the safeguards that are in place to prevent the introduction of FMD virus in legally imported animals and animal products, the most likely route of introduction of the virus would be the illegal importation of animal products harbouring the virus and the subsequent feeding of such material to pigs.

The 2001 UK epidemic was the first-ever to be presented to the New Zealand public by modern mass media. Television images of slaughtered animals and funeral pyres shocked a country that, over the past 2 decades, has lost much of its formerly strong rural identity. An increasingly urbanised western society recoiled in horror from the concept of pre-emptive slaughter and holocaust-like disposal methods, while some of the more dramatic 'risk management' measures imposed by countries in various parts of the world created an impression that there were very real risks associated with a broad range of products traded internationally. At the same time, quite legitimate concerns were raised from an animal welfare perspective, including whether vaccination might be a more humane method of control.

As a direct result of the widespread anxiety expressed in New Zealand during the 2001 FMD outbreak in the UK, the borders in this country are now tighter than ever before, and it is difficult to assess exactly what risks remain for our livestock populations. Although the FMD risks related to trade in animals and their products are now generally well understood, the risks involved with contaminated fomites are not. If more informed decisions are to be made in the future, it will be necessary for further research to be undertaken, particularly on the role of faeces and urine and the mechanical carriage of FMD virus to either ruminants or pigs on various contaminated articles. In particular, its survivability must be assessed on clothing and shoes, in conditions equivalent to those in aircraft cabins for times reflecting intercontinental flights. In addition, experiments need to be repeated on the persistence of FMD, under realistic combinations of heat and humidity, on various types of fodder, soil, animal hooves, and used agricultural machinery.

Following hard on the heels of the outbreak of bovine spongiform encephalopathy (BSE), the UK epidemic again raises important issues of public perception of risk and public trust in government. The perhaps inevitable restructuring of UK MAFF, even before the impending enquiries into the handling of the epidemic, reinforces the need for biosecurity agencies to be aware that the view of risk assessment as a purely scientific enterprise is no longer tenable, and to pay careful attention to the provision of information to a public that is now generally distrustful of technical experts (Slovic 1999).

Acknowledgements

The author would like to thank the following for their helpful comments and suggestions made on a draft version of this paper: Lawrence Gleeson, Martin Hugh-Jones, Nick Knowles, Dirk Pfeiffer, Alan Samuel, and MAF colleagues Stuart MacDiarmid, Mathew Stone and Joanne Thompson.

References

Anonymous. FMD in the Republic of Korea. *Australian Veterinary Journal* 78, 445–6, 2000

Anonymous. An open invitation to disease? *The Veterinary Times* 31, 1–2, 4 June 2001

Alexandersen S, Oleksiewicz MB, Donaldson AI. The early pathogenesis of foot-and-mouth disease in pigs infected by contact: a quantitative time-course study using TaqMan RT-PCR. *Journal of General Virology* 82, 747–55, 2001

Bachrach HL, Breese SS, Callis JJ, Hess WR, Patty RE. Inactivation of foot-and-mouth disease virus by pH and temperature changes and by formaldehyde. *Proceedings of the Society for Experimental Biology and Medicine* 95, 147–52, 1975

Barnett PV, Cox SJ. The role of small ruminants in the epidemiology and transmission of foot-and-mouth disease. *The Veterinary Journal* 158, 6–13, 1999

Bauer K. (1997) Foot-and-mouth disease as zoonosis. *Archives of Virology, Supplement* 13, 95–7, 1997

Blackwell JH. Foreign animal disease agent survival in animal products: recent developments. *Journal of the American Veterinary Medical Association* 184, 674–9, 1984

Blackwell JH, Hyde JL. Effect of heat on foot-and-mouth disease virus (FMDV) in the components of milk from FMDV-infected cows. *Journal of Hygiene, Cambridge* 77, 77–83, 1976

Brownlie J. Strategic decisions to evaluate before implementing a vaccine programme in the face of a foot-and-mouth disease (FMD) outbreak. *Veterinary Record* 148, 358–60, 2001

Burrows R. Excretion of foot-and-mouth disease virus prior to the development of lesions. *Veterinary Record* 82, 387–8, 1968

Burrows R, Mann JA, Greig A, Chapman WG, Goodridge D. The growth and persistence of foot-and-mouth disease virus in the bovine mammary gland. *Journal of Hygiene, Cambridge* 69, 307–21, 1971

Christensen B. *The Importation of Dairy Products: Risks to New Zealand Livestock*. MAF Regulatory Authority, Wellington, 1998

Cottral GE. Persistence of foot-and-mouth disease virus in animals, their products and the environment. *Bulletin de L'Office International des Épizooties* 71, 549–68, 1969

Cottral GE, Gailiunas P, Cox BF. Foot-and-mouth disease virus in semen of bulls and its transmission by artificial insemination. *Archiv für die gesamte Virusforschung* 23, 362–77, 1968

Davidson M. Foot and mouth disease. *Surveillance* 18(3), 13–5, 1991

Department of the Environment, Transport and the Regions. *Transport Statistics Great Britain 2000*. The Stationary Office, London, 2000

Donaldson AI. Quantitative data on airborne foot-and-mouth disease virus: its production, carriage and deposition. *Philosophical Transactions of the Royal Society of London* B302, 529–34, 1983

Donaldson AI. Risks of spreading foot-and-mouth disease through milk and dairy products. *Revue Scientifique et Technique de L'Office International des Épizooties* 16, 117–24, 1997

Donaldson AI, Alexandersen S. Relative resistance of pigs to infection by natural aerosols of FMD virus. *Veterinary Record* 148, 600–2, 2001

Donaldson AI, Doel TR. Foot-and-mouth disease: the risk for Great Britain after 1992. *Veterinary Record* 131, 141–20, 1992

Donaldson AI, Ferris NP. The survival of foot-and-mouth disease virus in open air conditions. *Journal of Hygiene, Cambridge* 74, 409–16, 1975

Donaldson AI, Ferris NP. Air sampling of pigs infected with foot-and-mouth disease virus: comparison of Litton and cyclone samplers. *Research in Veterinary Science* 33, 384–5, 1982

Donaldson AI, Knowles N. Foot-and-mouth disease in man. *Veterinary Record* 148, 319, 2001

Donaldson AI, Gloster J, Harvey LDJ, Deans DH. Use of prediction models to forecast and analyse spread during the foot-and-mouth disease outbreaks in Brittany, Jersey and the Isle of Wight in 1981. *Veterinary Record* 110, 53–7 1982

Donaldson AI, Alexandersen S, Sørensen JH, Mikkelsen T. Relative risks of uncontrollable (airborne) spread of FMD by different species. *Veterinary Record* 148, 602–4, 2001

Eisner RJ, McVicar JW. Foot-and-mouth disease virus on wool of infected sheep. *Bulletin de L'Office International des Épizooties* 92, 29–36, 1980

Garner MG. FMD in Asia – a growing threat. *Animal Health Surveillance Quarterly* 5, 1–3, 2000

Garner MG, Fisher BS, Murray JG. Economic aspects of foot-and-mouth disease: perspectives of a free country – Australia. *Revue Scientifique et Technique de L'Office International des Épizooties*, in press, 2002

Gailiunas P, Cottral GE. Presence and persistence of foot-and-mouth disease virus in bovine skin. *Journal of Bacteriology* 91, 2333–8, 1966

Gizitdinov NN. The survival of foot and mouth disease virus on the woolly coat of animals. *Trudy Inst. vet. (Kazakh.) NIVI* 9, 73–80, 1957 [Translated from Russian, NZ Translation Centre, 1998]

Gloster J, Sellers RF, Donaldson AI. Long distance transport of foot-and-mouth disease virus over the sea. *Veterinary Record* 110, 47–52, 1982

Gloster J, Hewson HJ, Mackay DKJ, Garland AJM, Donaldson AI, Mason IS, Brown RJ. Spread of foot-and-mouth disease from burning of animal carcases on open pyres. *Veterinary Record* 148, 585–9, 2001

Haydon DT, Samuel AR, Knowles NJ. The origin, generation and persistence of genetic variation in foot-and-mouth disease virus. *Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine, Edinburgh.* Pp 213–24, 2000

Hemadri D, Tosh C, Venkataramanan D, Sanyal A, Samuel AR, Knowles NJ, Kitching RP. Genetic analysis of foot-and-mouth disease virus type O isolates responsible for field outbreaks in India between 1993 and 1999. *Epidemiology and Infection* 125, 729–36, 2000

Hugh-Jones ME, Wright PB. Studies on the 1967–8 foot-and-mouth disease epidemic. *Journal of Hygiene, Cambridge* 68, 253–71, 1970

Hurst GW. Foot-and-mouth disease: the possibility of continental sources of the virus in England in epidemics of October 1967 and several other years. *Veterinary Record* 81, 610–4, 1968

Hyslop NStG. Secretion of foot-and-mouth disease virus and antibody in the saliva of infected and immunized cattle. *Journal of Comparative Pathology* 75, 111–7, 1965a

Hyslop NStG. Airborne infection with the virus of foot-and-mouth disease. *Journal of Comparative Pathology* 75, 119–26, 1965b

Hyslop NStG. The epizootiology and epidemiology of foot and mouth disease. *Advances in Veterinary Science* 14, 261–307, 1970

IETS. Conclusions of the Research Subcommittee of the International Embryo Transfer Society (IETS) Import/Export Committee. *Revue Scientifique et Technique de L'Office International des Épizooties* 11, 937–8, 1992

Kitching RP. A recent history of foot-and-mouth disease. *Journal of Comparative Pathology* 118, 89–108, 1998

Kitching RP. Foot-and-mouth disease: current world situation. *Vaccine* 17, 1772–4, 1999

Knowles NJ, Samuel AR, Davies PR, Kitching P, Donaldson AI. Outbreak of foot-and-mouth disease virus serotype O in the UK caused by a pandemic strain. *Veterinary Record* 148, 258–9, 2001

MacDiarmid SC. *Importation Into New Zealand of Meat and Meat Products.* MAF Regulatory Authority, Wellington, 1991

MacDiarmid SC, Thompson EJ. The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de L'Office International des Épizooties* 16, 45–56, 1997

McColl KA, Westbury HA, Kitching RP, Lewis VM. The persistence of foot-and-mouth disease virus on wool. *Australian Veterinary Journal* 72, 286–92, 1995

Mogajane EM. Foot-and-mouth disease in South Africa. *Office International des Épizooties Disease Information* 13, 164–5, 2000

Moonen P, Schrijver R. Carriers of foot-and-mouth disease virus: a review. *Veterinary Quarterly* 22, 193–7, 2000

Ogawa T, Matsuda K. Potential risk of transmission and spread of foot-and-mouth disease in Kagoshima Prefecture, Japan. *Japan Agricultural Research Quarterly* 34, 203–8, 2000

Parker J. Presence and inactivation of foot-and-mouth disease virus in animal faeces. *Veterinary Record* 88, 659–62, 1971

Pharo H. *Import Risk Analysis: Unprocessed Fibre of Sheep and Goats.* MAF Biosecurity Authority, Wellington, 1998

Pirtle EC, Beran GW. Virus survival in the environment. *Revue Scientifique et Technique de L'Office International des Épizooties* 10, 733–48, 1991

Prempeh H, Smith R, Müller B. Foot-and-mouth disease: the human consequences. *British Medical Journal* 322, 565–6, 2001

Samuel AR, Knowles NJ. Foot-and-mouth disease type O viruses exhibit genetically and geographically distinct evolutionary lineages (topotypes). *Journal of General Virology* 82, 609–21, 2001

Samuel AR, Knowles NJ, Kitching RP, Hafez SM. Molecular analysis of type O foot-and-mouth disease viruses isolated in Saudi Arabia between 1983 and 1995. *Epidemiology and Infection* 119, 381–9, 1997

Sanson RL. The epidemiology of foot-and-mouth disease: implications for New Zealand. *New Zealand Veterinary Journal* 42, 41–53, 1994

Sanson RL, Morris RS, Stern MW. EpiMAN-FMD: a decision support system for managing epidemics of vesicular disease. *Revue Scientifique et Technique de L'Office International des Épizooties* 18, 593–605, 1999

Schjernin-Thiesen K. The inactivating effect of a mixture of sodium chloride and sodium carbonate on foot-and-mouth disease virus on ox hides. *Bulletin de L'Office International des Épizooties* 77, 1125–9, 1972

Sellers RF. Quantitative aspects of the spread of foot and mouth disease. *Veterinary Bulletin* 41, 431–9, 1971

Sellers RF, Burrows R, Mann JA, Dawe P. Recovery of virus from bulls affected with foot-and-mouth disease. *Veterinary Record* 83, 303, 1968

Sellers RF, Donaldson AI, Herniman KAJ. Inhalation, persistence and dispersal of foot-and-mouth disease virus by man. *Journal of Hygiene, Cambridge* 68, 565–73, 1970

Sellers RF, Herniman KAJ and Mann JA. Transfer of foot-and-mouth disease virus in the nose of man from infected to non-infected animals. *Veterinary Record* 89, 447–9, 1971

Slovic P. Trust, emotion, sex, politics, and science: surveying the risk-assessment battlefield. *Risk Analysis* 19, 689–701, 1999

Smith LP, Hugh–Jones ME. The weather factor and foot and mouth disease epidemics. *Nature* 223, 712–5, 1969

Sørensen JH, MacKay DKJ, Jensen CØ, Donaldson AI. An integrated model to predict the atmospheric spread of foot-and-mouth disease virus. *Epidemiology and Infection* 124, 577–90, 2000

Sugiura K, Ogura H, Ito K, Ishikawa K, Hoshino K, Sakamoto K. Eradication of foot and mouth disease in Japan. *Revue Scientifique et Technique de L'Office International des Épizooties* 20, 701–13, 2001

Thomson GR. Foot-and-mouth disease. In: Coetzer JAW, Thomson GR, Tustin RC (eds). *Infectious Diseases of Livestock with Special Reference to Southern Africa.* Pp 825–51. Oxford University Press Southern Africa, Capetown, 1994

Thomson GR. The role of carrier animals in the transmission of foot and mouth disease. In: *Comprehensive Reports on Technical Items Presented to the International Committee or To Regional Commissions [Office International des Épizooties]. Conference Proceedings of the 64th General Session, Paris, 20–24 May 1996.* Pp 87–103. Office International des Épizooties, Paris, 1997

Thornton R. The National Centre for Disease Investigation. *Surveillance* 26(1), 9–10, 1999

Tsai CP, Pan CH, Liu MY, Lin YL, Chen CM, Huang TS, Cheng IC, Jong MH, Yang PC. Molecular epidemiological studies on foot-and-mouth disease type O Taiwan viruses from the 1997 epidemic. *Veterinary Microbiology* 74, 207–16, 2000

van Regenmortel MHV, Fauquet CM, Bishop DHL, Carstens EB, Estes MK, Lemon SM, Manioff J, Mayo MA, McGeoch DJ, Pringle CR, Wickner RB (eds). *Virus Taxonomy: Classification and Nomenclature of Viruses. Seventh Report of the International Committee on Taxonomy of Viruses*. Academic Press, SanDiego, 2000

Voinov SI. Persistence of foot-and-mouth disease virus on the wool coat of animals under central Asian conditions. *Tr. Vses. Inst. Sanit.* 30, 45–50, 1968 [Translated from Russian. TT 81–53756, Translation 29762, USDA National Agricultural Library, 1984]

Whyte CF. *Annual Statistics, 1993/94 – 2000/01*. Auckland, MAF Quarantine Service, Auckland, 2001

Wilson TM, Logan-Henfrey L, Weller R, Kellman B. Agroterrorism, biological crimes, and biological warfare targeting animal agriculture. In: Brown C, Bolin C (eds). *Emerging Diseases of Animals.* Pp 23–57. ASM Press, Washington DC, 2000

Yang PC, Chu RM, Chung WB, Sung HT. Epidemiological characteristics and financial costs of the 1997 foot-and-mouth disease epidemic in Taiwan. *Veterinary Record* 145, 731–4, 1999

Accepted for publication 18 January 2002