NEW ZEALAND AND OVERSEAS STATUS

CSF is an OIE listed disease\(^\text{2}\) and listed in New Zealand as an unwanted notifiable organism\(^\text{3}\). Reports of a disease diagnosed as swine fever were not uncommon in New Zealand between 1894 and 1904 although it has been subsequently suggested that these were more likely to be due to pasteurellosis or salmonellosis\(^\text{4}\). Outbreaks of CSF were recorded in 13 New Zealand herds in 1933 and an outbreak affecting a single herd was described in 1953. In both these cases infection was linked to the feeding of imported food waste\(^\text{4, 5}\). CSF last occurred in New Zealand in 1953\(^\text{6}\).

Overseas, CSF is recognised in Asia (Russia, People’s Republic of China, Indonesia, Malaysia, Philippines, Thailand, Vietnam, India, Nepal, Sri Lanka, and Israel), South America (Bolivia, Brazil, Ecuador, and Peru), Central America (El Salvador), the Caribbean (Cuba, Dominican Republic, and Haiti), the Balkans (Bulgaria, Croatia, the Former Yugoslav Republic of Macedonia, and Serbia and Montenegro), Eastern Europe (Hungary, Lithuania, and Slovakia) and Germany\(^\text{6}\). In Europe, disease is usually limited to wild boars\(^\text{7, 8, 9, 10, 11}\), although a recent outbreak in Lithuania was described in domestic pigs with infection believed to be associated with trade in animal products rather than spread from the local wild boar population\(^\text{10}\).

TRANSMISSION

In areas free from the disease, introduction is usually associated with the importation of infected pigs or the feeding of garbage containing uncooked pork scraps\(^\text{1}\).

Wild and domestic pigs are the only natural reservoirs of CSF virus (CSFV)\(^\text{7}\). All breeds and ages are susceptible, although adults are more likely to survive an acute infection\(^\text{1}\). Transmission of CSFV occurs primarily by direct or indirect contact with infected wild or domestic pigs or through ingestion of contaminated food\(^\text{8,9,10}\). Virus can be recovered from the faeces and saliva of clinically diseased individuals\(^\text{11}\). Infected boars shed virus in their semen, and transmission of CSFV associated with artificial insemination has been demonstrated and was thought to be a major feature of the 1997–98 CSF epidemic in the Netherlands\(^\text{12,13,14}\).

Classical swine fever (CSF) is a highly infectious pestivirus disease of pigs. Although previously considered to be an acute fatal disease associated with pathological lesions consistent with a severe viraemia, chronic and inapparent forms of the disease are now recognised, including persistent congenital infection of newborn pigs\(^\text{1}\). The acute form of CSF is characterised by a high temperature (>40.5°C), accompanied by lethargy and inappetance. Breeding herds often show an increase in abortions, stillbirths and weak piglets. Affected individuals may be inappetent, hot to touch, and show weakness or lameness in their hindquarters. If you suspect CSF infection call the free Exotic Pest and Disease hotline on 0800 80 99 66.

Aerosol transmission of CSFV has been demonstrated experimentally\(^\text{15}\) although studies in the Netherlands demonstrated that airborne spread may be limited to distances of no more than 250 metres in areas with a dense pig population\(^\text{16}\). Given the low pig density in New Zealand\(^\text{17}\), aerosol transmission would be unlikely to be a significant component of an outbreak in this country.

As with other pestiviruses, CSFV can cross the placenta to infect foetuses, resulting in abortion and stillbirths. However, infection at 50–70 days gestation can lead to the birth of persistently infected piglets which develop congenital tremors and wasting\(^\text{18}\) and shed large amounts of virus into their environment\(^\text{7}\).

Transmission via vehicles or individuals has historically been associated with the spread of CSF between farms\(^\text{19}\) although more recent evidence suggests that the role of mechanical transmission may have been overestimated\(^\text{20}\).

PATHOGENESIS

CSFV replicates in monocyte-macrophage cells and in the vascular endothelium. Leukopenia (especially
lymphopenia) is considered to be a classical early event following infection\(^\text{21}\). This lymphocyte depletion occurs 1–4 days before virus can be detected in serum by the reverse-transcriptase polymerase chain reaction\(^7\). Infection of endothelial cells is associated with the release of inflammatory cytokines that are believed to play a role in immunosuppression and viral dissemination\(^22\). Viral dissemination has also been linked to the ability of CSFV to infect and replicate in dendritic cells\(^23\). In\textit{ situ} hybridisation studies have shown that CSFV infection of spermatogonia and their progeny is likely to be responsible for the spread of the virus in semen from infected individuals\(^24\).

**PRESENTATION**

CSF is characterised by cutaneous hyperaemia and cyanosis together with non-specific clinical signs such as anorexia, pyrexia, lethargy, respiratory signs, and diarrhoea although these signs are far from pathognomonic and may also be associated with a number of other diseases including African swine fever, porcine reproductive and respiratory syndrome, porcine dermatitis and nephropathy syndrome, coumarin poisoning and salmonellosis\(^7, 26\). A pyrexia of 40.5–41.5°C is often seen before other clinical signs. Purple discoloration of the abdominal skin together with necrosis of the ears, tail and lips of the vulva may develop later. Conjunctivitis is also often described, with the eyelids stuck together by dried purulent exudate\(^1\).

A variant strain of CSFV has been reported in association with predominantly nervous signs, with pigs described in lateral recumbency with tetanic convulsion followed by clonic convulsion accompanied by loud squealing. Blindness, stumbling and pica have also been described\(^1\).

Acute CSF is associated with leukopenia and thrombocytopenia and diffuse petechiation and ecchymoses in the skin (Figure 1), lymph nodes, larynx (Figure 2), bladder, kidney (Figure 3) and ileocaecal junction. Multifocal infarction of the spleen is also described (Figure 4). In chronic CSF, button ulcers in the large intestine and caecum are often present (Figure 5), while haemorrhagic lesions may be less prominent or even absent.

Post-mortem examination is generally considered the most important aid to diagnosis of CSF\(^1\) although recent experience has demonstrated that this may be less valuable where there is a large variation in the presentation of individual cases\(^27, 28\). Acute cases are likely to be associated with submucosal and subserosal haemorrhages, although this may not be present in all individuals, whilst peracute cases may show no gross lesions at necropsy.

Epidemics associated with virulent strains of CSFV usually present with a morbidity of 100 percent and a case-fatality rate approaching 100 percent. Infection with a less virulent strain may be inapparent in growing and adult pigs, although perinatal mortality, abortions and mummified foetuses may be present\(^1\). It has been suggested that the genotype of pigs influences the clinical presentation following CSFV infection, with purebred pigs predisposed to acute fatal infections while crossbred pigs are more likely to develop chronic or transient disease\(^25\).

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Figure 1: Skin lesions in a pig caused by classical swine fever. Photo © Crown Copyright, 2009, reproduced with kind permission of the Department for Environment Food and Rural Affairs (UK).
DIAGNOSIS

There are no pathognomonic clinical signs associated with CSF, and diagnosis always requires laboratory confirmation. Any suspicion of CSF infection should be reported to MAFBNZ’s free Exotic Pest and Disease hotline on 0800 80 99 66 for further investigation.

PREVENTION AND CONTROL

Good biosecurity practices will significantly reduce the risk of CSFV introduction. Incursions of CSF are often linked to the feeding of illegally imported pig meat, such as in the United Kingdom in 2000\(^{29,30}\). Outbreaks in Croatia between 1997 and 2001 were associated with imported pig meat and contact with infected wild boars\(^{31}\). A Belgian outbreak in 1997 was linked to the movement of vehicles involved in CSF eradication in the Netherlands at that time\(^{32}\).

The three most important risk factors for the introduction of CSFV have been described as the importation of livestock, the presence of wild boars, and the feeding of imported swill\(^{33}\).

Any pig meat, porcine semen, or live pigs imported into New Zealand must comply with an Import Health Standard which contains measures to prevent the introduction of CSFV. The Biosecurity (Meat & Food Waste for Pigs) Regulations 2005 prohibit the feeding to pigs of untreated meat or untreated food waste, which must be heated throughout to 100°C for 1 hour or to an equivalent standard approved by MAF.
REFERENCES


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