SWINE VESICULAR DISEASE

AETIOLOGY

Classification of the causative agent

Swine vesicular disease (SVD) is classified as a pig enterovirus, in the family Picornaviridae. All isolates are classified in a single serotype, with four distinguishable antigenic/genomic variants, which evolved sequentially in different time-periods without overlapping, except for the third and fourth variants that were co-circulating in Italy during 1992-1993. All SVD viruses occurring since then diverge from a common origin and cluster in a unique antigenic/genomic lineage corresponding to the fourth and most recent group; however, two genomic sub-lineages are distinguishable within it. Antigenically, swine vesicular disease virus (SVDV) is related to the human coxsackie virus B5.

Resistance to physical and chemical action

Temperature:	Preserved by refrigeration and freezing, inactivated by 56°C/1 hour
pH:	SVDV is stable in the pH range 2.5–12.0
Disinfectants/chemicals:	In the presence of organic matter, inactivated by sodium hydroxide (1% combined with detergent). Direct treatment of swine waste with 1.5% (w/v) NaOH or $Ca(OH)_2$ for 30 minutes could inactivate SVDV at either 4°C (39°F) or 22°C (72°F). Mixture of didecydimethylammonium chloride and 0.1% NaOH for 30–60 minutes also demonstrates efficacy. For personal disinfection and in the absence of gross organic matter, disinfectants, such as oxidising agents, iodophores, acids etc., are suitable if combined with detergents.
Survival:	Resistant to fermentation and smoking processes. May remain in hams for 180 days, dried sausages for >1 year, and in processed intestinal casings for >2 years

EPIDEMIOLOGY

Movement of subclinically infected animals is the most common means of moving SVDV. Transport of large numbers of swine often results in small lesions and these provide a portal of entry for the SVDV. Introduction of susceptible swine into contaminated environments will also result in SVD outbreaks. Non-heat treated garbage fed to swine provides another means for infected meat to cause disease.

- Morbidity rate in herds may be low but high in groups of pigs (in pens)
- Does not cause death

Hosts

- Swine are the only natural host for SVDV

Transmission

- Virus infects swine via: lesions in skin and mucosa, ingestion and inhalation
- Direct contact among infected swine or with their excretions
    - very low titres of virus needed to infect animals across broken skin
    - faecal contamination is a major source of virus spread, often within contaminated vehicles or premises
- Meat scraps and ‘swill’ derived from infected pigs
    - SVDV not inactivated by normal pH change associated with *rigor mortis*

Sources of virus

- Affected pigs may excrete virus from the nose and mouth and in the faeces up to 48 hours before the onset of clinical signs
- Most virus is produced in the first 7 days after infection

    - virus excretion from the nose and mouth normally stops within 2 weeks
    - may continue to be shed for up to 3 months in the faeces
- All tissues contain virus during the viraemic period
- Ruptured vesicles (epithelium and fluid) are a high-titre source of virus; faeces are a lower-titre source of virus

Occurrence

The disease is reported occasionally from countries in Europe and is reported regularly from southern and sporadically from central Italy. The disease is likely present in various parts of eastern Asia.

DIAGNOSIS

The incubation period for SVD is between 2 and 7 days. For the purposes of the *OIE Terrestrial Animal Health Code*, the incubation period for the SVD is 28 days.

Clinical diagnosis

SVD can be a subclinical, mild or severe vesicular condition depending on the strain of virus involved, the age of pigs affected, the route and dose of infection, and the husbandry conditions under which the pigs are kept. The clinical signs of SVD may easily be confused with those of Foot and mouth disease (FMD) and any outbreaks of vesicular disease in pigs must be differentiated by laboratory confirmation. Recent outbreaks of SVD have been characterised by less severe or no clinical signs; infection has been detected when samples are tested for a serosurveillance programme or for export certification.

- The first sign of disease may be sudden appearance of lameness in several animals in a group in close contact and a transient fever of up to 41°C
    - off feed for a few days
- Vesicles then develop on the coronary band, typically at the junction with the heel, and interdigital spaces of the feet
    - may affect the whole coronary band resulting in loss of the hoof
- More rarely, vesicles may also appear on the snout, particularly on the dorsal surface, on the lips, tongue and teats, and shallow erosions may be seen on the knees
- On hard surfaces, animals may be observed to limp, stand with arched back, or refuse to move even in the presence of food
- Clinical signs are more severe in wet or unsanitary conditions and abrasive floors and conversely pigs kept on grass or housed on deep straw may demonstrate little or no clinical signs
- Nervous signs have been reported, but are unusual
- Young animals are usually more severely affected by SVD
- Abortion is not a typical feature of SVD
- Recovery occurs usually within 2–3 weeks; only evidence of infection being a dark, horizontal line on the hoof where growth has been temporarily interrupted
- Some strains produce only mild clinical signs or are subclinical
- Morbidity may reach 100% but usually no deaths are associated

Lesions

- Vesicle formation is the only known lesion directly attributable to the infection
    - these lesions are indistinguishable from FMD and other vesicular disease in pigs

The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated April 2013.