SHEEP POX AND GOAT POX

AETIOLOGY

Classification of the causative agent

Virus family *Poxviridae*, genus *Capripoxvirus*

Sheep pox virus (SPV) and goat pox virus (GPV) were once believed to be strains of the same virus, but genetic sequencing has now demonstrated them to be separate viruses. Most strains are host specific and cause severe clinical disease in either sheep or goats, while some strains have equal virulence in both species. Further complicating this is that recombination can occur between sheep and goat strains, which produce a spectrum with intermediate host preference and range of virulence

SPV and GPV cannot be distinguished from each other with serological techniques, including viral neutralisation. SPV and GPV are also closely related to lumpy skin disease virus in cattle (LSDV), but there is no evidence LSDV causes disease in sheep and goats. It has a different transmission mechanism (insects) and partially different geographic distribution.

Resistance to physical and chemical action

Temperature:	Susceptible to 56°C/2 hours; 65°C/30 minutes. Some isolates inactivated at 56°C/60 minutes
pH:	Susceptible to highly alkaline or acid pH (hydrochloric or sulphuric acid at 2% for 15 minutes)
Disinfectants/chemicals:	Inactivated by phenol (2%) in 15 minutes. Sensitive to detergents, e.g. sodium dodecyl sulphate. Sensitive to ether (20%), chloroform, formalin (1%), and sodium hypochlorite (2-3%), iodine compounds (1:33 dilution), Virkon® 2%, quarternary ammonium compounds 0.5%.
Survival:	Susceptible to sunlight, but remains viable in wool/hair and dry scabs on skin for up to 3 months. Persists in unclean shaded pens for as long as 6 months. Survives freeze–thaw cycles, but infectivity may be reduced

EPIDEMIOLOGY

- Morbidity rate: Endemic areas 70–90%
- Mortality rate: Endemic areas 5–10%, although can approach 100% in imported animals

Hosts

- All breeds of domestic and wild sheep and goats, although most strains cause more severe clinical disease in only one species
- Native breeds in endemic areas are far less susceptible than introduced breeds of European or Australian origin – morbidity and mortality may approach 100%

Transmission

- Transmission is usually by aerosol after close contact with severely affected animals containing ulcerated papules on the mucous membranes. There is no transmission in the prepapular stage, e.g. animals early in disease or those dying peracutely (e.g. Soay breed of European sheep). There is reduced transmission once papules have become necrotic and neutralising antibody produced (about one week after onset). Animals with mild localised infections also rarely transmit disease.
- Infection may also occur through other mucous membranes or abraded skin
- Chronically infected carriers do not occur

- Indirect transmission by contaminated implements, vehicles or products (litter, fodder) occurs
- Indirect transmission by insects (mechanical vectors) has been established (minor role)

Sources of virus

- Ulcerated papules on mucous membranes prior to necrosis
- Skin lesions with scabs: contain large amounts of virus in association with antibody but infectivity is not known
- Saliva, nasal and ocular secretions
- Milk, urine, faeces
- Semen or embryos: transmission not yet established

Occurrence

Capripox is endemic in Africa north of the Equator, the Middle East, Turkey, Iran, Iraq, Afghanistan, Pakistan, India, Nepal, parts of the People's Republic of China, Bangladesh.The most recent outbreaks occurred in Vietnam in 2005, Mongolia in 2008 and 2009, and Azerbaijan in 2009. The first outbreak in Chinese Taipei occurred in 2008 and was eradicated by stamping out and movement control.
For more recent, detailed information on the occurrence of this disease worldwide, see the OIE *World Animal Health Information Database* (WAHID) Interface [http://www.oie.int/wahis/public.php?page=home] or refer to the latest issues of the *World Animal Health* and the OIE *Bulletin*.

DIAGNOSIS

Incubation period is 8–13 days. It may be as short as 4 days following experimental infection by intradermal inoculation or mechanical transmission by insects

Clinical diagnosis

Clinical signs vary from mild to severe, depending on host factors (e.g. age, breed, immunity) and viral factors (e.g. species predilection and virulence of viral strain). Inapparent infections also occur

- Early clinical signs
  - rise in rectal temperature to above 40°C
  - macules develop in 2-5 days – small circumscribed areas of hyperaemia, most obvious on unpigmented skin
  - papules develop from macules – hard swellings of between 0.5 and 1 cm in diameter – which may cover the body or be restricted to the groin, axilla and perineum. Papules may be covered by fluid-filled vesicles, but this is rare. A flat haemorrhagic form of capripox has been observed in some breeds of European goat, in which all the papules appear to coalesce over the body; this form is always fatal.
- Acute phase: within 24 hours after appearance of generalised papules
  - affected animals develop rhinitis, conjunctivitis and enlargement of all superficial lymph nodes, especially prescapular lymph nodes
  - papules on the eyelids cause blepharitis of varying severity
  - papules on the mucous membranes of the eyes and nose ulcerate, creating mucopurulent discharge
  - mucosae of the mouth, anus, and prepuce or vagina become necrotic
  - breathing may become laboured and noisy due to pressure on the upper respiratory tract from the swollen retropharyngeal lymph nodes draining developing lung lesions.
- If animal survives acute phase
  - papules become necrotic from vascular thrombosis and ischaemic necrosis
  - papules form scabs in the next 5–10 days, which persist for up to 6 weeks, leaving small scars
  - skin lesions are susceptible to fly strike
  - secondary pneumonia is common
  - anorexia is unusual unless mouth lesions physically interfere with feeding
  - abortion is rare.

Lesions

- Skin lesions: congestion, haemorrhage, oedema, vasculitis and necrosis. All the layers of epidermis, dermis and sometimes musculature are involved
- Lymph nodes draining infected areas: enlargement (up to eight times normal size), lymphoid proliferation, oedema, congestion, haemorrhage
- Pox lesions: on mucous membranes of the eyes, mouth, nose, pharynx, epiglottis, trachea, on the rumenal and abomasal mucosae, and on the muzzle, nares, in the vulva, prepuce, testicles, udder, and teats. Lesions may coalesce in severe cases
- Lung lesions: severe and extensive pox lesions, focal and uniformly distributed throughout the lungs; congestion, oedema, focal areas of proliferation with necrosis, lobular atelectasis. Enlargement, congestion, oedema and haemorrhages of mediastinal lymph nodes

Differential diagnosis

The clinical signs of severe sheep pox and goat pox are highly characteristic. However, in their mild form they can be confused with parapoxvirus causing orf or urticaria from multiple insect bites.

- Contagious ecthyma (contagious pustular dermatitis or orf)
- Insect bites
- Bluetongue
- Peste des petits ruminants
- Photosensitisation
- Dermatophilosis
- Parasitic pneumonia
- Caseous lymphadenitis
- Mange

Laboratory diagnosis

Samples

Samples for virus isolation must be sent to the laboratory as soon as possible. They should be kept cold and shipped on gel packs. If these samples must be shipped long distances without refrigeration, glycerol (10%) can be added; tissue samples must be large enough that glycerol does not penetrate into the centre of the tissue and destroy the virus.

Neutralising antibodies can interfere with virus isolation and some antigen-detection tests; samples for these tests must be collected during the first week of illness. Samples for PCR can be taken after neutralising antibodies have developed. Paired serum samples should be collected for serology.

- Live animals: Full skin thickness biopsies; vesicular fluid if available; scabs; skin scrapings; lymph node aspirates; whole blood collected into heparin or EDTA; paired sera
- Animals at necropsy: skin lesions; lymph nodes; lung lesions; histology: full set of tissues, especially those with lesions

Procedures

Identification of the agent

- Genome detection by polymerase chain reaction (PCR) using capripoxvirus-specific primers for the attachment protein gene is described in lumpy skin disease (chapter 2.4.14) of the *OIE Terrestrial Manual*
- Transmission electron microscopy: rapidly identifies typical capripox virions
- Virus isolation in cell culture (primary lamb testis or lamb kidney): the appearance of CPE may take 4–12 days, intracytoplasmic inclusions are clearly seen by haematoxylin and eosin staining, and antigen can be detected by immunoperoxidase or immunofluorescence staining techniques

- Agar gel immunodiffusion test (AGID): tests lymph gland biopsy material taken from an early case of capripox; cross-reacts with parapox
- Capripoxvirus antigen and inclusion bodies may also be seen in stained cryostat or paraffin sections of biopsy or post-mortem lesion material.
- Inhibition of cytopathic effect using positive serum
- Antigen-detection ELISA

Serological tests

- Virus neutralisation: most specific serological test, but not sufficiently sensitive since immunity to capripox infection is predominantly cell mediated – infected animals may only produce undetectable low levels of neutralising antibody.
- Indirect fluorescent antibody test: cross reacts with other poxviruses
- Agar gel immunodiffusion (AGID): cross reacts with other poxviruses
- Western blotting: uses P32 antigen of capripoxvirus for reaction with test sera; sensitive and specific, but is expensive and difficult to carry out
- ELISA : P32 antigen or another appropriate antigen expressed by a suitable vector could be used to develop an acceptable and standardised serological test

For more detailed information regarding laboratory diagnostic methodologies and vaccines, please refer to Chapter 2.7.14 Sheep pox and goat pox in the latest edition of the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Diagnostic Techniques".

PREVENTION AND CONTROL

No treatment

Sanitary prophylaxis

- If culling is not possible, isolation of infected herds and sick animals for at least 45 days after recovery
- Slaughtering of infected herd if possible
- Proper disposal of cadavers and products - burning or burial is often used
- Stringent cleaning and disinfection of farms and equipment
- Quarantine of new animals before introduction into herds
- Animal and vehicle movement controls within infected areas
- Vaccination may be considered when the disease has spread more widely

Medical prophylaxis

Live and inactivated vaccines have been used for the control of capripox. All strains of capripoxvirus so far examined share a major neutralisation site and will cross protect.

- There are several attenuated virus vaccines delivered by subcutaneous or intradermal route; conferred immunity lasts up to 2 years
- Inactivated vaccines give, at best, only short-term immunity
- Currently, no recombinant vaccines for capripoxviruses are commercially available. However, a new generation of capripox vaccines is being developed that uses the capripoxvirus genome as a vector for the genes of other ruminant pathogens, for instance genes of rinderpest and peste des petits ruminants (PPR) viruses.

For more detailed information regarding laboratory diagnostic methodologies and vaccines, please refer to Chapter 2.7.14 Sheep pox and goat pox in the latest edition of the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Requirements for Vaccines".

For more detailed information regarding safe international trade in terrestrial animals and their products, please refer to the latest edition of the OIE *Terrestrial Animal Health Code*.

REFERENCES AND OTHER INFORMATION

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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.