# Sheep & Goat Pox

Capripoxvirus Infection

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# Etiology

Sheep pox and goat pox result from infection by sheeppox virus (SPV) or goatpox virus (GPV), closely related members of the *Capripox* genus in the family Poxviridae. Most isolates are host specific, with SPV mainly causing disease in sheep and GPV predominantly affecting goats. However, some isolates can cause serious disease in both species. SPV and GPV cannot be distinguished from each other with serological techniques (including serum neutralization), and were once thought to be strains of a single virus. Genetic sequencing has now demonstrated that these viruses are distinct, but recombination can occur between them. Recombinant strains usually have intermediate host specificity.

SPV and GPV are closely related to the virus that causes lumpy skin disease in cattle (LSDV). The relationships between these three capripoxviruses are still being established, but one recent analysis suggests that GPV and LSDV are more closely related to each other than SPV is to LSDV.

### **Species Affected**

Sheep and goat capripoxviruses cause disease only in these two species. Many SPV isolates are specific for sheep, and many GPV strains are specific for goats, but some strains of these viruses readily affect both species. Infections have not been reported in wild ungulates.

# **Geographic Distribution**

Sheep pox and goat pox are found in parts of Africa and Asia, the Middle East, and most of the Indian subcontinent.

# Transmission

SPV and GPV are often transmitted by the respiratory route during close contact, but they may also enter the body through other mucous membranes or abraded skin. These viruses can be found in saliva, nasal and conjunctival secretions, milk, urine and feces, as well as in skin lesions and their scabs. Ulcers on the mucous membranes are important sources of virus. Whether SPV and GVP can be transmitted in semen or embryos has not been established. Animals are most contagious before neutralizing antibodies develop, which occurs approximately a week after the onset of clinical signs. Experimentally infected sheep and goats can shed poxviruses in nasal, conjunctival and oral secretions for 1 to 2 months, but shedding peaks during the second week after inoculation, then declines rapidly. Chronically infected carriers are not seen.

SPV and GPV can also be spread on fomites or transmitted mechanically by insects such as stable flies (*Stomoxys calcitrans*), although the latter route may be uncommon. These viruses can remain infectious for up to six months in shaded sheep pens. They may also be found on the wool or hair for as long as three months after infection, and possibly longer in scabs. Whether the viruses in scabs are infectious is unknown; these viruses are complexed with antibodies and can be difficult to recover on tissue culture media.

# **Incubation Period**

The incubation period varies from four to 21 days, but it is usually 1 to 2 weeks. Clinical signs generally appear sooner when the virus is inoculated by insects than when it is transmitted in aerosols. After experimental inoculation into the dermis, primary lesions can develop at the site within 2 to 4 days.

# **Sheep and Goat Pox**

### **Clinical Signs**

The clinical signs vary from mild to severe, depending on the animal's age, breed, immunity and other factors. Inapparent infections also occur.

In affected animals, an initial fever is usually followed in one to five days by the characteristic skin lesions, which begin as erythematous macules, and develop into 0.5-1.5 cm hard papules. In the common, papulovesicular form of the disease, the centers of the papules become depressed, whitish gray and necrotic, and are surrounded by an area of hyperemia. Dark, hard, sharply demarcated scabs eventually form over the necrotic areas. Vesicles might be seen during the intermediate stage, but are uncommon. In the uncommon, nodular form of the disease ('stonepox'), the papules develop into nodules. These nodules can be found in the epidermis, dermis and subcutaneous tissues. They become necrotic and slough, leaving a hairless scar. Some European breeds of goats may develop a flat hemorrhagic form of goat pox. In this form, the papules seem to coalesce over the body, and the animal invariably dies. Capripox lesions have a predilection for areas of sparsely wooled/ haired skin such as the axillae, muzzle, eyelids, ears, mammary gland and inguinal area, but in more severe cases, they may cover the body. In animals with heavy wool, the lesions can be easier to find by palpation than visual inspection. Mild infections can easily be missed; only a few lesions may be present, often around the ears or the tail. All superficial lymph nodes usually become enlarged within a day of the appearance of generalized papules; the prescapular lymph nodes are particularly noticeable.

Lesions can also develop on the mucous membranes and internal organs, causing systemic signs. In some cases, these symptoms may precede the onset of skin lesions by a day or two. Lesions in the mouth, nares, eyes or eyelids can cause salivation or inappetence, as well as rhinitis, conjunctivitis or blepharitis with mucopurulent discharges. Affected mucous membranes may become necrotic and ulcerate or slough. Animals with lung lesions may have respiratory signs including coughing, nasal discharge and dyspnea. Nodules in the intestines can cause diarrhea. Depression and emaciation may be seen in some animals. Abortions can occur but are not common. Some breeds of sheep can die of acute disease before the characteristic skin lesions appear.

Capripox lesions can take several weeks to heal, and may leave permanent scars on the skin. During healing, they are susceptible to fly strike. Secondary bacterial infections, including pneumonia, are common, and death can occur at any stage of the disease. Recovery can be slow if the animal was severely affected.

### **Post Mortem Lesions**

The skin usually contains macules, papules and/or necrotic lesions and scabs, surrounded by areas of edema,

hemorrhage and congestion. The papules penetrate through both the dermis and epidermis; in severe cases, they may extend into the musculature. Skin lesions may not be as apparent at necropsy as they are in living animals. The mucous membranes of the eyes, nose, mouth, vulva and prepuce may be necrotic or ulcerated. The lungs often contain congested, edematous or consolidated areas, and firm gray or white nodules. Nodules in the lungs can be up to 5 cm in diameter, and are particularly common in the diaphragmatic lobes. In early stages of the disease, they may appear as red spots. Papules or ulcerated papules are common on the abomasal mucosa. They may also be found on the rumen, large intestine, pharynx, trachea and esophagus. Pale, discrete subcapsular foci are sometimes present on the surface of the kidney, liver and testes. Lymph nodes throughout the body are usually enlarged and edematous, and may be congested and hemorrhagic.

### **Morbidity and Mortality**

Morbidity and mortality vary with the breed of the animal, its immunity to capripoxviruses, and the strain of the virus. Mild infections are common among indigenous breeds in endemic areas, but more severe disease can be seen in young or stressed animals, animals with concurrent infections, or animals from areas where pox has not occurred for some time. Reported morbidity rates in indigenous breeds range from 1% to 75% or higher. Although the mortality rate is often less than 10%, case fatality rates of nearly 100% have been reported in some young animals.

Imported breeds of sheep and goats usually develop severe disease when they are moved into an endemic area. The morbidity and mortality rates can approach 100% in newly imported, highly susceptible flocks.

### Diagnosis

### Clinical

Sheep or goat pox should be suspected in febrile animals with the characteristic full-thickness skin lesions and enlarged lymph nodes. Dyspnea, conjunctivitis, nasal discharges and other signs may also be seen. The mortality rate is usually high in naïve animals. Although sheep pox and goat pox are usually distinctive in fully susceptible animals, these diseases can be subtler and more difficult to diagnose in indigenous animals.