

LUMPY SKIN DISEASE

(Pseudo-urticaria, Neethling virus disease, exanthema nodularis bovis, knopvelsiekte)

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Definition [top](#)

Lumpy skin disease (LSD) is an acute to chronic viral disease of cattle characterized by skin nodules that may have inverted conical necrosis (sitfast) with lymphadenitis accompanied by a persistent fever.

Etiology [top](#)

The causative agent of LSD is a capripoxvirus. The prototype strain of LSD is the Neethling virus (1). The LSD virus (LSDV) is one of the largest viruses (170-260 by 300-450 nm) (16). There is only one serotype of LSDV. The LSDV is very closely related serologically to the virus of sheep and goat pox (SGP) from which it cannot be distinguished by routine virus neutralization or other serological tests (3). Restriction endonuclease studies of capripoxviruses indicate that LSDV strains

are essentially identical with each other and with a Kenyan strain (O 240/KSGP) of sheep and goat pox virus (SGPV). Other strains of SGPV from Kenya were different from the O 240/KSGP strain but similar to each other and resembled strains of SGPV from the Arabian Peninsula. The Kenyan group of SGPV strains showed differences when compared with ones from India, Iraq, and Nigeria (13).

The LSDV is very resistant to physical and chemical agents. The virus persists in necrotic skin for at least 33 days and remains viable in lesions in air-dried hides for at least 18 days at ambient temperature (22).

Host Range [top](#)

Lumpy skin disease is a disorder of cattle. There is inconclusive evidence regarding the infection of water buffalo (*Bubalus*) with LSDV. The African Cape buffalo (*Synercus caffer*) and other wild ungulates have not been infected during epizootics of LSD in Africa. Experimental infection of some species is possible (7).

Geographic Distribution [top](#)

Lumpy skin disease was first described in Northern Rhodesia in 1929 (17). Since then, the disease has spread over most of Africa in a series of epizootics (7, 11). The most recently affected countries include Kuwait in 1986-88 (2) and Egypt in 1988 (20). An outbreak of LSD occurred in Israel in 1989 (21). For the first time, the disease was eradicated by slaughter and vaccination.

Transmission [top](#)

Biting insects play the major role in the transmission of LSDV (5,15). Epidemics of LSD are associated with rainy seasons. The disease spreads in river basins and areas conducive to insect multiplication (6,10,15,22). *Stomoxys calcitrans* experimentally transmitted LSDV, but biting lice (*Mallophaga* spp.), sucking lice (*Damalinia* spp.), or *Culicoides nubeculosus* did not (14). In Kenya, *Culex mirificus* but also *Aedes natronius* were in heavy concentration during an LSD epizootic and were associated with transmission (15). Direct contact seems to play a minor role in the spread of LSD.

Incubation Period [top](#)

In the field the incubation period is 2 to 5 weeks (10). Following experimental infection by intradermal inoculation, a lesion usually develops at the inoculation site within 6 to 20 days.

Clinical Signs [top](#)

Lumpy skin disease virus causes inapparent to severe disease in cattle. All ages of cattle can be affected, but young calves are usually more severely affected (Fig. 72). The severity of the disease depends on the dose of the inoculum as well as the susceptibility of the host (*Bos taurus* is more susceptible than *Bos indicus*) and the route of exposure. A fever 104 to 107° F (40-41.5° C) can occur and can be transitory or last up to 4 weeks. Generally within 2 days after the appearance of the fever, swellings or nodules 1 to 5 cm in diameter appear in the skin and generalization occurs. Depression, anorexia, excessive salivation, oculonasal discharge, agalactia, and emaciation are presented. Nodules 1 to 7 cm in diameter may occur anywhere on the body but especially in the skin of the muzzle, nares, back, legs, scrotum, perineum, eyelids, lower ear, nasal and oral mucosa, and tail. The hair stands erect over early skin lesions. The nodules are painful and involve the epidermis, dermis, and subcutaneous tissue and may even involve the musculature. As the disease progresses, the nodules become necrotic, and eventually a deep scab forms; this lesion is called a sitfast (Fig. 71). Secondary bacterial infection can complicate healing and recovery. Lesions on the teats can result in severe secondary bacterial infection with loss of the quarter owing to mastitis.

Where extensive generalization occurs, animals can become lame and reluctant to move because of edema. Lameness also may result from inflammation of the tendons, tendon sheaths (tendosynovitis), joints (synovitis), and laminae (laminitis). Severe edema in the brisket and legs can occur. If secondary bacterial infection develops in the tendon sheaths and joints, pemmanent lameness may result. Superficial lymph nodes such as the mandibular, parotid, prescapular, and prefemoral nodes, draining affected areas of skin become enlarged 4 to 10 times normal size.

Abortion may occur as the result of prolonged fever. Davies (7) has reported intrauterine infection of late-term fetuses in which calves are born with LSD lesions. Temporary or pemmanent sterility in bulls can result from the fever or lesions of the reproductive organs. Cows may not come into estrus for several months after LSD (7).

The lesions may persist in various stages over a course of 4 to 6 weeks. Final resolution of lesions may take 2 to 6 months, and nodules can remain visible 1 to 2 years. Permanent damage to the hide is inevitable in clinical cases.

Gross Lesions [top](#)

The gross lesions of LSD are well described (3,10,19,22). Skin nodules have congestion, hemorrhage, edema, and vasculitis with consequent necrosis and involve all layers of the epidermis, dermis, subcutaneous tissue, and often adjacent musculature. Lymph nodes draining affected areas are enlarged up to 10 times normal size with extensive lymphoid proliferation, edema, congestion, and hemorrhage.

Mucous membranes of the oral and nasal cavities can have pox lesions that coalesce in severe cases. Pox lesions may occur in the pharynx, epiglottis, and trachea (Fig. 73). Pox lesions are not easily visualized in the lungs but appear as focal areas of atelectasis and edema (Fig. 74). In severe cases, pleuritis can occur with enlargement of the mediastinal lymph nodes.

Synovitis and tendosynovitis with fibrin in the synovial fluid can occur. Pox lesions can be present in the testicles and urinary bladder.

Morbidity and Mortality [top](#)

Morbidity for LSD varies from 3 to 85 percent (10,15,22) and likely depends on prevalence of the mechanical insect vector and the susceptibility of the cattle. Mortality is generally low (1 to 3 percent). In one outbreak in South Africa, mortality was about 20 percent when an anaplasmosis vaccine was prepared from bovine blood contaminated with LSDV (9,10). Unusually high mortality (75 to 85 percent) in other outbreaks of LSD was not explained (9,10).

Diagnosis [top](#)

Field Diagnosis [top](#)

A tentative diagnosis of LSD can be made based upon clinical signs. A contagious disease with generalized skin nodules having a characteristic inverted conical necrosis of skin nodules (sitfast), persistent fever, emaciation, and low mortality suggests LSD.

Specimens for the Laboratory [top](#)

Skin biopsies of early lesions (ones where necrosis has not occurred) provide samples that can be used for virus isolation, histopathology, and electron microscopy. Samples should be taken from at least three animals. Samples aspirated from enlarged lymph nodes can be used for virus isolation. Samples for virus isolation should be shipped to the laboratory under wet ice if they will arrive

in 2 days and be shipped under dry ice if more time will be required. Samples for histopathology should be preserved in 10 percent buffered formalin (DO NOT FREEZE). Serum samples should be taken from acute and chronic cases. Followup serum samples (convalescent samples) should be taken 2 to 3 weeks after the first appearance of skin lesions.

Laboratory Diagnosis [top](#)

To confirm an initial diagnosis in an LSD-free area, the virus has to be isolated and identified. The laboratory procedures for the diagnosis of LSD include virus isolation in lamb testicle or fetal bovine lung cell cultures or both. Virions may be detected by electron microscopy (7). Herpesviruses may be present in bovine skin samples (1,12) and cause confusion in cell culture studies. Serological tests include virus neutralization and indirect fluorescent antibody (IFA) (8). The IFA test may measure group-reactive antibody that may be elicited by other pox viruses.

Differential Diagnosis [top](#)

Listed below are several diseases that should be considered in the differential diagnosis of LSD:

Bovine herpes mammillitis (also called Allerton virus infection caused by Bovid Herpesvirus-2) — The lesions are superficial (involving only the epidermis) and occur predominantly on the cooler parts of the body such as teats and muzzle. Generalized skin lesions can occur accompanied by a transient fever (1 to 3 days). Resolution of the lesion is rapid and results in focal alopecia but no hide damage.

Streptotrichosis (*Dermatophilus congolensis* infection) — lesions are superficial (often moist and appear as crusts) scabs or 0.5- to 2-cm diameter accumulations of keratinized material. Lesions are common in the skin of the neck, axillary region, inguinal region, and perineum. The organism can be demonstrated by Giemsa staining.

Ringworm — The lesions of ringworm in cattle are grayish, raised, plaque-like, and often pruritic. The organism can be demonstrated with a silver stain.

Hypoderma bovis infection — The parasitic fly larvae of this parasite have a predilection to migrate to the dorsal skin of the back. They cause a nodule with a small central hole through which the larva exits the body, which results in significant hide damage.

Photosensitization — Dry, flaky, inflamed areas are confined to the nonpigmented parts of the skin.

Bovine papular stomatitis — Pox-like lesions occur in the skin of the muzzle, oral cavity, and esophagus. There is no generalized disease.

Insect bites — The trauma from insect bites causes local inflammation, edema, and pruritus. Insects seldom bite mucous membranes.

Urticaria — Delayed hypersensitivity reactions can be confused with LSD. Such lesions generally resolve within 3 to 5 days. An example of this was described by Shimshony (1989) where allergic reactions occurred after vaccination with a foot-and-mouth disease vaccine.

Besnoitiosis (Globidiosis) — Thick-walled cysts in the skin are caused by sporozoan parasites of the genus *Besnoitia*, which are transmitted mechanically by certain biting flies. Histologic sections will reveal the parasites.

Treatment [top](#)

Treatment is directed at preventing or controlling secondary infection. Animals infected with LSDV generally recover (mortality is usually less than 3 percent). Complete recovery may take several months and may be prolonged where secondary bacterial infection occurs. Loss of production results from severe emaciation, lowered milk production, extensive damage to hides, and loss of draft from lameness. It may take up to 6 months for animals severely affected by LSDV to recover fully (9).

Vaccination [top](#)

In endemic areas, vaccination against LSD has been successfully practiced. In the Union of South Africa, an attenuated LSD vaccine is used. In Kenya, sheep and goat pox virus is used (4). In Egypt, the Romanian strain of sheep and goat pox vaccine has been used successfully for prophylaxis against LSD.

Control and Eradication [top](#)

The most likely way for LSD to enter a new area is by introduction of infected animals. Biting insects that have fed on infected cattle may travel and be blown for substantial distances. It is likely that LSD spread to Israel via contaminated insects blown across the Sinai Desert (21). The movement of contaminated hides

represents another potential means for this resistant virus to move.

If LSD is confirmed in a new area before extensive spread occurs, the area should be quarantined, infected and exposed animals slaughtered, and the premises cleaned and disinfected. Vaccination of susceptible animals within the quarantine should be considered.

If the disease has spread over a large area, the most effective means of controlling losses from LSD is vaccination. However, even with vaccination, consideration still should be given to eliminating infected and exposed herds by slaughter, proper disposal of animals and contaminated material, and by cleaning and disinfecting contaminated premises, equipment, and facilities.

In the Union of South Africa, the control of insects was not effective in preventing the spread of LSD, but current insecticides together with repellents aid in the prevention of the spread of LSD.

Public Health [top](#)

There is no evidence that LSDV infects humans.

GUIDE TO THE LITERATURE [top](#)

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