

NAIROBI SHEEP DISEASE

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Definition [top](#)

Nairobi sheep disease (NSD) is a noncontagious, tick-borne, viral infection of sheep and goats characterized by hemorrhagic gastroenteritis and high mortality.

Etiology [top](#)

Nairobi sheep disease virus (NSDV) is transmitted primarily by the brown tick, *Rhipicephalus appendiculatus*. The causative agent is an RNA-containing virus having structural and chemical characteristics common within the *Bunyaviridae* viruses (16). However, it is antigenically independent of this group but is closely related to the Ganjam virus of goats in India (7). Ganjam virus is antigenically related to Dugbe virus isolated from cattle in west Africa. A new genus, *Nairovirus*, has been proposed for these three viruses (19).

Host Range [top](#)

Laboratory and domesticated animals other than sheep and goats are resistant to infection with NSDV (17). Davies (5) was unable to isolate NSDV from blood or tissues of a wide range of wild ruminants and rodents. However, it has been suggested (4) that the African field rat (*Arvicathus abyssinicus nubilans*) might be a reservoir host.

Geographic Distribution [top](#)

Nairobi sheep disease is usually confined to countries in east Africa, where the principal vector, *R. appendiculatus*, is endemic. The disease has been reported most frequently in Kenya in Kikuyu country between Nairobi and Mount Kenya as well as in Uganda, Tanzania, and Somalia (1). A disease similar to NSD called Kisenyi sheep disease has been described in the Republic of the Congo (3). Sera positive for NSDV antibody were confirmed in an outbreak in haired sheep in Harar Province of Ethiopia.

Transmission [top](#)

Nairobi sheep disease is not contagious and is only transmitted by ticks. Transmission by contact does not occur. Experimentally, NSD can be transmitted by the inoculation of infectious blood, serum, or organ suspensions into susceptible animals. Large doses (50 cc) of virulent blood or serum given to sheep by mouth may also cause infection.

The major vector of NSD is *R. appendiculatus* and is generally thought to be the only species of tick in which transovarial transmission of NSD is known to occur; however, there is firm evidence that a population of *R. pulchellus* in Somalia also transovarially transmits the virus. The African bont tick (*Amblyomma variegatum*) is believed to have been responsible for one large outbreak of NSD in Kenya (3). However, in a laboratory investigation, *A. variegatum* was found to be a less efficient vector than *R. appendiculatus* (4). More recently in Kenya, a closer correlation has been shown to exist between the presence of NSD antibody in sheep and goats and infestations with *R. appendiculatus* than with *A. variegatum* (14). Of eight species of ticks representing three genera (*Amblyomma*, *Hyalomma*, and *Rhipicephalus*) collected in Kenya, NSDV was isolated only from *R. appendiculatus* (5). Unfed adult ticks are infective for over 2 years. It has previously been stated by Daubney and Hudson (3) that infected *R. appendiculatus* lose their infectivity when allowed to feed on immune sheep or on nonsusceptible animals; however, this was later shown not to be the case (11).

Incubation Period [top](#)

The incubation period in natural infections is 4 to 15 days. Experimental inoculation of sheep and goats with virus results in a shorter incubation period of 1 to 3 days (19).

Clinical Signs [top](#)

Nairobi sheep disease is characterized by an acute hemorrhagic gastroenteritis (17). Clinical signs of NSD begin with a temperature rise to 40 to 41° C (104 to 106° F) and, during this stage, a prominent clinical depression develops followed by a temperature decline and diarrhea. There is an abundant mucopurulent nasal discharge, and breathing may become rapid and painful. Leukopenia is prominent during the period of hyperthermia (18). Initially the feces are thin and watery but later they may contain mucus and blood. In less acute cases the course of the disease is slower, and sheep become anorexic, weak, and recumbent with signs of diarrhea. There may be abortions. In hyperacute infections there is a sudden rise in body temperature that abruptly declines on the third to the sixth day followed by collapse and death within a few hours.

Gross Lesions [top](#)

The most obvious lesions are those associated with hemorrhagic gastroenteritis. The abomasal mucosa is hyperemic and may be covered with petechial hemorrhages. Intestinal lesions are most severe in the cecum and the anterior part of the colon. Hemorrhages in the mucosa of the large intestine are numerous, and the intestinal contents are blood stained.

There is nonspecific congestion and petechial and ecchymotic hemorrhages in most organs and tissues.

Generalized hyperplasia of lymphoid tissue is a prominent lesion. Lymph nodes are enlarged and edematous. The spleen may be several times its normal size and engorged with blood.

In pregnant ewes, the genital tract may be very hyperemic, which is indicative of inflammation, and fetal membranes may be swollen and edematous, and contain hemorrhages. The aborted fetus has numerous hemorrhages in its tissues and organs.

Morbidity and Mortality [top](#)

Early studies (17) revealed that sheep and goats resident in endemic areas were generally immune, whereas severe outbreaks occurred in susceptible animals moved into these region. Davies (5) in a 9-year study discovered that NSD outbreaks were mainly associated with the trading of livestock in the vicinity of Kenya's major cities. Sporadic outbreaks in nonendemic regions were usually preceded by excessive amounts of rainfall and the appearance of the tick vector.

Prognosis in susceptible sheep and goats is poor, although mild infection may occur. Mortality in Merino and Merino crossbreeds is about 40 percent, but mortality in Masai sheep is much higher.

Diagnosis [top](#)

Field Diagnosis [top](#)

An outbreak of NSD is nearly always associated with movement of susceptible animals into an endemic area where *R. appendiculatus* is abundant. When recently introduced small ruminants become ill with signs of severe enteritis and nasal discharge within an NSD endemic area and sheep native to the area do not, it is a likely assumption that NSD is the current problem. This is particularly true if the incidence of illness in sheep is high, is low in goats, and is absent in cattle and other animals. Susceptibility of goats may depend on breed (6).

Specimens for the Laboratory [top](#)

Heparinized blood is the best source of NSDV during the febrile stage. During later stages of illness, when body temperature has declined or is normal and the amount of virus in the blood stream is low or absent, spleen and mesenteric lymph nodes are the best tissues for virus isolation (2,17). Also submit serum, preferably paired specimens, for serology.

Laboratory Diagnosis [top](#)

Laboratory confirmation is necessary for a definitive diagnosis. Inoculation of cell culture with suspensions of infected organs or plasma and subsequent staining of the cell culture by the direct (FAT) or indirect fluorescein conjugated antibody test (IFAT) provide the most reliable means of identifying NSDV. The use of a fluorescein conjugated antibody test allows detection virus in 24 to 48 hours after inoculation of cell cultures and thus is not dependent on cytopathogenic effects in tissue culture cells (10).

Intracerebral inoculation of suckling mice is an excellent method of isolating NSDV. The brain material from infected mice can be used as a source of viral antigen, and its identity can be determined by FAT or a complement fixation test (10).

Differential Diagnosis [top](#)

The disease must be differentiated from heartwater, Rift Valley fever, anthrax, some types of plant and heavy metal poisoning, peste des petits ruminants, and coccidiosis.

Differentiation from other viral or rickettsial diseases is based on geographic location of the outbreak, species of animal affected, cross-immunity studies, serologic investigations, and viral isolation.

The following criteria may be of assistance in arriving at a diagnosis:

1. Nairobi sheep disease

- a. Nairobi sheep disease causes severe illness in sheep — an affliction that is characterized by diarrhea, often hemorrhagic.
- b. There is high mortality in Masai sheep, low mortality in Merino or Merino crosses, low mortality in goats, and no mortality in other ruminants, including wildlife.
- c. *R. appendiculatus* ticks are abundant in the region.
- d. Intracerebral inoculation of mice with blood or tissue suspensions causes rodent death.
- e. The Nairobi sheep disease virus can be isolated and propagated in tissue culture.
- f. The fluorescein antibody test and serum neutralization and complement fixation tests will identify the causative agent.

2. Heartwater

- a. Heartwater causes severe illness in sheep and is characterized by CNS signs followed by death. Pulmonary edema and an abundance of

fluid in the pericardial sac and pleural cavities may be seen in the more prolonged cases. Gastroenteritis is rare.

b. There is a high incidence of illness and mortality in exotic breeds of sheep, goats, and cattle in contrast to a lower incidence and mortality in indigenous breeds.

c. *Amblyomma hebraeum* or *A. variegatum* ticks are abundant in the affected area.

d. The rickettsia may be passaged in mice, often without any evidence of illness in the affected mice.

e. *Cowdria ruminantium*, a rickettsia, can be demonstrated in endothelial cells of capillaries found in brain smears and endothelial cells of large blood vessels stained with Giemsa.

f. The rickettsia cannot be isolated easily in tissue culture.

3. Rift Valley Fever (RVF)

a. In cattle, sheep, goats, and man, RVF is a very acute disease.

b. Rift Valley fever is characterized by a rapid course of infection, severe depression, diarrhea, massive liver necrosis, and widespread abortion.

c. The illness appears after periods of heavy rainfall when there is an abundance of mosquitoes, the arthropod vectors of the virus.

d. Ticks are not vectors of RVF and may be absent from the area of infection.

e. Mice, tissue cultures, and embryonated hen's eggs can be infected, and the isolated virus can be identified by serologic and immunologic methods.

4. Anthrax

a. Many species of mammals may be affected.

- b. The most prominent lesions are multiple hemorrhages, hemorrhagic enteritis, and prominent swelling of the spleen with failure of blood to clot.
- c. Blood or tissue smears stained with Giemsa reveal numerous encapsulated rod-shaped bacteria arranged in chains.
- d. Inoculated laboratory animals die, have numerous hemorrhages, and have an abundance of encapsulated bacteria in their tissues.
- e. *Bacillus anthracis* can be grown and identified on laboratory media.

5. Arsenic poisoning (from dips)

- a. Many species of animals may be affected.
- b. Signs: Profuse watery diarrhea, sometimes blood tinged, severe colic, dehydration, depression, weakness, and CNS signs; high fatality rates.
- c. Lesions: Edema and necrosis of gastric and intestinal epithelium and subepithelium. Diffuse degeneration of liver and other abdominal viscera.
- d. Arsenic detected in tissues.

6. Coccidiosis

- a Signs: Diarrhea (sometimes bloody), dehydration, fever, anorexia, and anemia.

The disease can be fatal — especially in lambs.

- b. Lesions: Edema, inflammation, and mucosal hemorrhage predominantly in the ileum, cecum, and upper colon.
- c. Thick white patches of oocysts may develop in small intestine. These oocysts can be demonstrated microscopically.

Treatment [top](#)

There is no specific treatment for NSD. Supportive treatment, protection from climatic adversities, and availability of good quality feed may reduce the mortality rate.

Vaccination [top](#)

Recovery from NSD leads to lifelong immunity. Because sheep and goats in endemic areas are constantly exposed to ticks carrying virus, they maintain good immunity and have no clinical signs of illness. It has been suggested (14) that lambs and kids are protected by colostrum antibody until they can acquire an active immunity through infection.

The Nairobi sheep disease virus can be propagated in cell culture (goat testes, goat kidneys, and hamster kidneys). When cell culture virus is attenuated, it is capable of protecting sheep and goats from NSD (9). The Entebbe strain of NSDV passaged 140 to 150 times through mouse brain is also used as vaccine. However, because of variability of breed responses to modified live virus vaccines and their adverse effects, they are generally not recommended.

Control and Eradication [top](#)

Susceptible sheep and goats must be protected from the vector by weekly acaricide dipping and spraying. Movement of animals into endemic areas must be controlled unless sheep and goats are naturally immune or have been vaccinated.

Because the infection is not transmitted by contact, there is little need for strict quarantine procedures. Dead sheep should be buried or incinerated. Livestock on the premises should be dipped or sprayed with acaricides to reduce the existing tick population.

Public Health [top](#)

Antibodies against NSDV have been detected in human blood serum, but it is not known if these antibodies are the result of NSDV infection or have been caused by a yet unidentified agent. An apparently naturally acquired clinical case was reported from Uganda in which a young man from whom virus was isolated experienced transient clinical signs (15). However no serological conversion has been demonstrated in investigators working with the virus (6).

GUIDE TO THE LITERATURE [top](#)

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