

## RINDERPEST

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### Definition [top](#)

Rinderpest (RP) is a contagious viral disease of cattle, domestic buffalo, and some species of wildlife. It is characterized by fever, oral erosions, diarrhea, lymphoid necrosis, and high mortality.

### Etiology [top](#)

Rinderpest virus (RPV) is a single-stranded RNA virus in the family Paramyxoviridae, genus *Morbillivirus*. It is immunologically related to canine distemper virus, human measles virus, peste des petits ruminants virus, and marine mammal morbilliviruses. There is only one serotype of rinderpest virus, but field strains vary widely in virulence, ease of transmission, and host affinity.

Rinderpest virus is a relatively fragile virus. Sunlight is lethal, and the vaccine must therefore be kept in a brown bottle and protected from light; virus in a thin layer of blood is inactivated in 2 hours. Moderate relative humidity inactivates the virus more quickly than either high or low humidity. The virus is very sensitive to

heat, and both lyophilized and reconstituted virus should therefore be kept cold; lyophilized virus stored at  $-20^{\circ}\text{C}$  is viable for years. Vaccine reconstituted in pure water quickly loses potency. Vaccine is more stable in a saline solution; reconstitution in a molar concentration of sulfate ions greatly increases resistance to heat.

Rinderpest virus is rapidly inactivated at pH 2 and 12 (10 minutes); optimal for survival is a pH of 6.5-7. The virus is inactivated by glycerol and lipid solvents.

### Transmission [top](#)

Rinderpest was established as an infectious disease in 1754 when susceptible animals were infected by placing bits of material previously dipped in morbid discharge into an incision made in the dewlap. In 1899, cattle were infected with a bacteria-free filtrate.

Secretions and excretions, particularly nasal-ocular discharges and feces, 1 to 2 days before clinical signs to 8 to 9 days after onset of clinical signs contain large quantities of virus. Spread of RP is by direct and indirect (contaminated ground, waters, equipment, clothing) contact with infected animals; aerosol transmission is not a significant means of transmission (it occurs only in a confined area and over a short distance). A major reason RP spreads in Africa is that the herds are nomadic. Cattle follow the grass and thus move great distances, and during the dry season, many herds will use the same well or watering area, and thus there is ample opportunity for cross-infection. It is said that a good fence will control RP.

There is only one serotype of RPV; recovered or properly vaccinated animals are immune for life, and there is no vertical transmission, arthropod vector, or carrier state. For these reasons, RPV is an ideal virus to be targeted for eradication.

Highly virulent strains of RPV are responsible for epizootics in susceptible animals and tend to die out. Milder strains tend to persist in an area, and the disease is not recognized as RP unless serology is performed.

The roles the various hosts can play in the disease are as follows:

Cattle and domestic buffalo — highly susceptible

Sheep and goats in Africa — subclinical infection and seroconversion, but there is no transmission to other animals.

Sheep and goats in India — when infected by low-passage goat RP

vaccine will transmit to domestic buffalo.

Pigs — Swayback pigs in Thailand and the Malay peninsula can be naturally infected and may die. European pigs can be infected by ingestion of RPV-infected meat and will transmit to cattle and other pigs.

### **Host Range** [top](#)

Most wild and domestic cloven-footed animals can be infected.

### **Geographic Distribution** [top](#)

Rinderpest is present in the Indian subcontinent, Near East, and sub-Saharan Africa.

#### **Wild ungulates**

Highly susceptible — African buffalo, wildebeest, kudu, eland, giraffe, warthog

Fairly susceptible — Thompson gazelle, hippopotamus

Wild ungulates are infected by contact with cattle and can transmit to cattle. In the absence of RP in cattle, the disease dies out in wildlife.

### **Incubation Period** [top](#)

The incubation period varies with the strain of virus, dosage, and route of exposure. Following natural exposure, the incubation period ranges from 3 to 15 days but is usually 4 to 5 days.

### **Clinical Signs** [top](#)

Depending on the strain of virus, resistance of the animal affected, and concurrent infection, RP can appear as a peracute, acute, or mild infection.

#### **Peracute Form**

This form is seen in highly susceptible and young animals. The only signs of illness are a fever of 104-107° F (40-41.7° C), congested mucous membranes, and death

within 2 to 3 days after the onset of fever.

## Acute or Classic Form

This form of the disease progresses as follows:

Small amounts of virus may be in nasal and ocular secretions before the onset of fever

Fever of 104-106° F (40-41.1° C)  
Serous to mucopurulent ocular discharge (Fig. 92)  
Serous to mucopurulent nasal discharge.  
Leukopenia  
Depression  
Anorexia  
Constipation  
Oral erosions — Salivation may be abundant and frothy (Fig. 93).  
Fever decreases and viral titer drops.  
Diarrhea — May be very watery or hemorrhagic, or both.  
Dehydration, emaciation  
Prostration and death 6 to 12 days after onset of illness.

### Gross Lesions [top](#)

Oral lesions are variable; some isolates cause good oral lesions and with others there is no oral lesion. Oral lesions start as small grey foci that may coalesce. The grey (necrotic) epithelium then sloughs off and leaves a red erosion.

Mouth — Lesions occur on the gums, lips, hard and soft palate, cheeks, and base of the tongue. Early lesions are grey, necrotic, pinhead-sized areas that later coalesce and erode and leave red areas (Fig. 94).

Esophagus — Brownish necrotic or eroded areas.

Rumen and reticulum — Lesions are rare.

Omasum — Erosions and hemorrhage are rare.

Abomasum — Congestion and edema.

Small intestine — Necrosis or erosion of Peyer's patches in the jejunum (Fig. 95); necrosis or erosions over the lymphoid area in the ileum (ingesta adhering to the intestinal mucosa indicates areas of necrotic epithelium).

Cecum and colon — The wall may be edematous, and there may be blood in the lumen and blood clots on the mucosa. Lesions are usually more severe in the upper colon (edema of the wall, erosions in the mucosa, and congestion) (Fig. 96). The lesions may be accentuated at the cecocolic junction (Fig. 97). Further down the colon, the colonic ridges may be congested; this is referred to as "tiger striping" (Fig. 98). Tiger striping can occur in other diarrheas and probably results from tenesmus.

Severity of intestinal lesions varies between isolates.

Lymph nodes — Generally swollen and edematous.

Liver — There may be petechial to ecchymotic hemorrhages in the gall bladder (Fig. 99).

Lung — There may be emphysema, congestion, and areas of pneumonia.

### **Diagnosis** [top](#)

### **Field Diagnosis** [top](#)

Rinderpest should be considered in all ages of cattle whenever there is a rapidly spreading acute febrile disease accompanied by the preceding clinical signs and lesions of RP. The all ages stipulation is important because this will be one of the major differences between bovine virus diarrhea-mucosal disease, which predominately affects animals between 4 and 24 months of age.

### **Specimens for the Laboratory** [top](#)

Because the viral titer drops when the fever falls and diarrhea starts, specimens should preferably be collected from animals with a high fever and oral lesions. The following samples should be collected from live animals:

- Blood in EDTA or heparin
- Blood for serum
- Swabs containing lacrimal fluid
- Necrotic tissue from the oral cavity
- Aspiration biopsies of superficial lymph nodes

For the best specimens, a febrile animal should be slaughtered and specimens collected. If this cannot be done, then collect specimens from moribund animals. Collect the blood samples listed above and sections of

- Spleen
- Lymph nodes
- Tonsil

The preceding samples should be transported to the laboratory on wet ice — NOT FROZEN.

A complete set of tissues, including sections of all lesions, should be collected in 10 percent formalin.

### **Laboratory Diagnosis** [top](#)

To confirm the initial diagnosis in a free area, the virus has to be isolated and identified.

### **Differential Diagnosis** [top](#)

The differential diagnosis for RP should include bovine virus diarrhea (mucosal disease), infectious bovine rhinotracheitis, malignant catarrhal fever, foot-and-mouth disease, vesicular stomatitis, salmonellosis, paratuberculosis, and arsenic poisoning.

### **Vaccination** [top](#)

The following types of RP vaccine have been used:

- Lapinized in China and Korea
- Avianized-lapinized in Korea
- Goat-adapted in India
- cell-culture-adapted in Africa, Middle East, and India.

An experimental vaccinia-vectored vaccine containing the F and H genes of RPV has protected against challenge inoculation of virulent virus

The two most commonly used vaccines today (1996) are the goat-adapted and cell-culture-adapted vaccines. The goat-adapted vaccine is only partially attenuated; it will cause disease in animals with low innate resistance or concurrent latent disease and kills sheep and goats. The cell-culture-attenuated vaccine was developed by Plowright in Kenya in the 1960's. This is a safe vaccine for many species and produces life-long immunity in cattle (animals challenge-inoculated 7 years after vaccination were protected). In endemic areas where cattle have been vaccinated, colostrum immunity will interfere with the vaccination of calves up to 11 to 12 months of age. Because the duration of colostrum immunity is variable, the recommendation is to vaccinate calves annually for 3 years.

One of the biggest problems with the cell-culture-adapted vaccine has been stability. The lyophilized virus has to be kept cold (cold chain) until used. The combination of maintenance of the cold chain and remoteness of vaccination sites made RP vaccination very expensive. Because of the uncertainty that the vaccine being used was viable, in areas of Africa it is and was the policy to vaccinate animals every year in the hope that one of the vaccinations would immunize the animal. Researchers at Plum Island in the early 1990's greatly increased the stability of the lyophilized vaccine by modifying the stabilizers and lyophilization process. This change in production is now being used in some production facilities in Africa.

Experimentally, the vaccinia-vectored RP vaccine protected cattle against challenge inoculation with RPV. This vaccine is undergoing field testing. This vaccine could be particularly useful in an eradication program because vaccinia-vectored-RP-vaccine immunized animals can be differentiated serologically from animals having antibody induced by live virus. The vaccinia-vectored vaccine would enable a country toward the end of an eradication program to maintain herd immunity to RP without using a live RP virus.

### **Control and Eradication [top](#)**

Countries and areas free of RP should prohibit unrestricted movement of RP-susceptible animals and uncooked meat products from areas infected by RP or practicing RP vaccination. Because recovered animals are not carriers, and there are good serological techniques, zoological ruminants and swine can be imported with proper quarantine and testing. If an outbreak occurs, the area should be quarantined, infected and exposed animals slaughtered and buried or burned, and

ring vaccination considered.

Experimentally it has been shown that RPV will not be transmitted by bovine embryo transfer if the embryos have been processed by the technique recommended by the International Embryo Transfer Society and the OIE.

High-risk countries (those trading with, or geographically close to, infected countries) can protect themselves by having all susceptible animals vaccinated before they enter the country or vaccinating the national herd, or both. If an outbreak occurs, the area should be quarantined and ring vaccinated.

Endemic countries should vaccinate the national herd. Owing to the uncertainty of vaccine potency, the recommendation is to vaccinate annually for at least 4 years, followed by annual vaccination of calves. Foci of infection should be quarantined and stamped out. Wildlife, sheep, and goats should be monitored serologically. Serological monitoring of sheep and goats could be complicated by using RP vaccine to protect against peste des petits ruminants.

### Public Health [top](#)

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