

Rinderpest

Cattle Plague

Last Updated: August 2008

Minor updates: January 2016

Note: The World Organization for Animal Health (OIE) declared rinderpest eradicated worldwide, as of May 2011. However, samples of rinderpest virus are still believed to be present in laboratories.

Importance

Rinderpest is an acute, highly contagious, viral disease of cattle, domesticated buffalo and some species of wildlife. The classical form of rinderpest is one of the most lethal diseases of cattle, and can have a catastrophic effect on naïve herds. At one time, epidemics of rinderpest occurred regularly in Eurasia. In 1889, cattle shipped from India carried the rinderpest virus to Africa, causing an epidemic that established the virus on the continent. Initially, approximately 90% of the cattle in sub-Saharan Africa and many sheep and goats died. Wild buffalo, giraffe and wildebeest populations were decimated. The loss of plow animals, herds and hunting resulted in mass starvation, killing a third of the human population in Ethiopia and two-thirds of the Maasai people of Tanzania. The reduction in the number of grazing animals also allowed thickets to form in grasslands. These thickets provided breeding grounds for tsetse flies, resulting in an outbreak of sleeping sickness in humans. Some consider this epidemic to have been the most catastrophic natural disaster ever to affect Africa.

Although the rinderpest virus was eradicated from Europe early in the 20th century, epidemics continued to occur in sub-Saharan Africa and many parts of Asia. In areas where it persisted, rinderpest became the main constraint to livestock production. Several eradication campaigns were conducted after World War II. One international project, started in the 1960s, eradicated or controlled the virus in much of Africa; however, in the 1970s, the termination of vaccination campaigns and surveillance efforts allowed the disease to emerge from two remaining pockets of infection and recolonize large areas. A similar event happened in Asia in the 1980s. In 1992, the Food and Agriculture Organization (FAO) of the United Nations began the Global Rinderpest Eradication Programme, with the goal of complete eradication by the year 2010. As of 2011, rinderpest was declared eradicated. Rinderpest is the first worldwide eradication of an animal pathogen; only one other virus, human smallpox, has ever been completely eliminated from nature.

Etiology

Rinderpest results from infection by rinderpest virus, a member of the genus *Morbillivirus* of the family Paramyxoviridae. There is just one serotype of this virus, but three genetically distinct lineages – lineage 1, lineage 2, and lineage 3 – have been identified. In the past, these lineages were found in different geographic areas. Rinderpest viruses can undergo changes in virulence, and some recent strains caused only mild disease in cattle despite severe losses in wildlife. Such strains retain the capacity to become highly virulent again in domesticated animals.

Species Affected

Most cloven-hooved animals (order Artiodactyla) are susceptible to rinderpest virus to some degree. Cattle, water buffalo, yaks, African buffalo, giraffes, warthogs and Tragelaphinae (spiral-horned antelope) are particularly susceptible to disease. Wildebeest and East African zebu are moderately susceptible, and gazelles, sheep and goats are mildly susceptible. Asian breeds of pigs appear to be more susceptible than African or European breeds. Rinderpest is rare in camelids.

Cattle are the most important maintenance hosts for rinderpest virus. Sheep and goats are relatively unimportant in the epidemiology of this disease. Among wildlife, African buffalo seem to be the most important hosts. Although there is some disagreement on the length of time the virus can persist in this population, some studies suggest that it disappears after approximately three years. In at least one environment where African buffalo were infected, the virus did not spread to other susceptible species in the area, including wildebeest. Currently, it is believed that wildlife populations cannot maintain the rinderpest virus indefinitely.



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Zoonotic potential

Rinderpest virus has not been reported to infect humans

Geographic Distribution

Rinderpest was eradicated from Europe early in the 20th century. Lineage 1 has been reported only in Africa and the Middle East, and was last seen in 2001. Lineage 3 (the “Asian lineage”) was found in Russia, Turkey, and parts of Asia and the Middle East. This lineage has not been seen since 2000. Lineage 2 was once reported from many parts of Africa, but it has now been eradicated from the region.

Transmission

Transmission of rinderpest virus usually occurs through direct or close indirect contact with infected animals. Small amounts of virus can be found in nasal and ocular secretions, saliva, milk, urine and feces beginning 1-2 days before the onset of fever. Blood and all tissues are also infectious before the clinical signs appear. Large amounts of rinderpest virus can be found in the animal’s secretions and excretions (including nasal and ocular discharges, saliva, feces, milk, semen, vaginal discharges and urine), as well as expired air, during the first week of clinical signs, but virus shedding decreases as specific antibodies develop and the animal recovers. Pigs can be infected if they ingest contaminated meat, and infected pigs can transmit the virus to cattle. Rinderpest virus can remain viable for at least a week in meat kept at 4°C. Aerosol transmission is insignificant in the epidemiology of the disease, and is typically only seen over short distances in confined spaces. However, some sources suggest that rinderpest virus may occasionally be transmitted up to 100m or more at night, when the humidity is very high. Infected animals do not become carriers; the virus maintains itself by passing from animal to animal in a large, susceptible population. Vertical transmission does not occur.

Although fomites can spread rinderpest virus, this virus is readily inactivated by sunlight and drying, and fomite-mediated transmission is relatively unimportant. Rinderpest virus can remain viable on unshaded pastures for 6 hours or on shaded pastures for 18-48 hours. Bare enclosures usually lose their infectivity within 48 hours and contaminated buildings within 96 hours. Because rinderpest virus is inactivated quickly by autolysis and putrefaction, this virus is destroyed within 24 hours in carcasses; however, freezing or chilling of the carcass in some climates could slow these processes and allow the virus to survive longer.

Disinfection

Rinderpest virus can be killed by most common disinfectants including phenol, cresol, sodium hydroxide (2% for 24 hours) and lipid solvents. The FAO recommends that premises, equipment and clothing be cleaned, then decontaminated with oxidizing agents such as sodium or calcium hypochlorite, or alkalis such as sodium hydroxide or sodium carbonate. Feces and effluents should be treated with sodium carbonate, before they are burned or buried.

Pasteurization or heat treatment can inactivate the virus in milk.

Incubation Period

The incubation period for rinderpest ranges from 3 to 15 days; 4 to 5 days is typical. The virulence and dose of virus and the route of exposure affect the incubation period. Mild forms of the disease can have an incubation period between one and two weeks. The World Organization for Animal Health (OIE) has established a maximum incubation period of 21 days for zoosanitary measures.

Clinical Signs

Rinderpest infections can vary in severity depending on the virulence of the strain and resistance of the infected animal. A peracute form, characterized primarily by high fever and sudden death, is mainly seen in young and newborn animals. In the acute (classical) form in cattle, a prodromal period of fever, depression, decreased appetite, decreased milk yield, congestion of mucous membranes, and serous ocular and nasal discharges is followed in approximately 2-5 days by the development of necrotic oral lesions. Necrotic epithelium can be found on the lips, tongue, gums, buccal mucosa, soft and hard palates. These lesions begin as pinpoints but enlarge rapidly to form gray plaques or a thick, yellow pseudomembrane. They slough to form shallow, nonhemorrhagic erosions. The muzzle eventually dries and develops cracks, and the animal becomes anorexic and develops mucopurulent ocular and nasal discharges. The breath is fetid. Necrotic lesions may also be found on the nares, vulva, vagina and preputial sheath. Diarrhea usually starts a few days after the onset of oral necrosis; it is typically profuse and watery at the onset, but may contain mucus, blood and shreds of epithelium in the later stages. Severe abdominal pain, thirst and tenesmus often accompany the diarrhea, and animals may die from dehydration. Dyspnea may be seen, and a maculopapular rash has been described on sparsely haired areas such as the groin and axillae. Mortality varies with the strain. Convalescence can be prolonged and may be accompanied by secondary infections. Pregnant cows often abort during this period.

In endemic areas, cattle may also develop mild subacute disease or atypical forms of rinderpest. Lineage 2 viruses can appear in cattle as mild, short-lived fever with slight congestion of the mucous membranes. Small, focal areas of raised, pale epithelial necrosis may be seen on the lower gum, and a few eroded cheek papillae can occur in some animals; these lesions are transient. A slight, serous ocular or nasal discharge may also be seen; this discharge does not usually become mucopurulent. Most animals are not noticeably depressed, and can continue to graze and behave normally. Lineage 2 infections can be difficult to recognize in cattle; however, these viruses can cause severe disease if they spread to susceptible wildlife such as Asian buffalo, giraffe, eland, and lesser kudu.

Rinderpest is usually milder in sheep and goats than cattle, and some infections are subclinical. The clinical signs

may include fever and anorexia, with diarrhea in some animals. Severe cases with necrotic stomatitis, oculonasal discharge, conjunctivitis, pneumonia and diarrhea (similar to classical disease in cattle) may also be seen.

Peracute disease, with fever and sudden death, can be seen in Asiatic breeds of pigs. These animals can also have acute disease characterized by the sudden onset of fever, depression, inappetence, shivering, vomiting and epistaxis. Mucosal necrosis and erosions can develop, and diarrhea with rapid dehydration and emaciation may be seen. Some pigs may die. Subclinical infections have been reported in European pigs.

In susceptible wildlife, the clinical signs can include fever, nasal discharge, erosive stomatitis, gastroenteritis, and death; however, the signs can vary with the species. In buffalo, rinderpest generally resembles the disease in cattle, but lymphadenopathy, plaque-like keratinized skin lesions and keratoconjunctivitis might also be seen. Similar signs can be seen in lesser kudu, and severe keratoconjunctivitis often causes blindness, but diarrhea is uncommon in this species.

Post Mortem Lesions [Click to view images](#)

In the classical form of rinderpest, the carcass is often dehydrated and emaciated, and shows evidence of diarrhea and mucopurulent nasal discharges. The eyes may be sunken. Depending on the stage of the disease and strain of the virus, congestion, pinhead or larger gray necrotic foci, or extensive necrosis and erosions may be seen in the oral cavity. Necrotic areas are sharply demarcated from healthy mucosa. In some cases, the necrotic lesions extend to the soft palate, pharynx and upper esophagus. Necrotic plaques are occasionally found on the pillars of the rumen, but other areas of the rumen and reticulum are usually unaffected. Occasionally, erosions and hemorrhages may be seen in the omasum. Severe congestion, petechiation and edema may be found in the abomasum, particularly in the pyloric region. White necrotic foci may be seen in Peyer's patches; necrosis, erosions and sloughing can be seen in the adjacent areas. The small intestine is otherwise unaffected. In the large intestine, blood and blood clots may be found in the lumen, and edema, erosions and congestion may be seen in the walls, particularly in the upper colon. The ileocecal valve, cecal tonsil and crests of the longitudinal folds of the cecal, colonic and rectal mucosae can be greatly congested in animals that die acutely, and may be darkened in more chronic cases, a lesion known as 'tiger striping' or 'zebra striping'. (Tiger striping can also occur in other diarrheas, and is probably caused by tenesmus.) The lymph nodes are usually enlarged and edematous, and the spleen may be slightly larger than normal. Petechiae and ecchymoses may be found in the gall bladder, and emphysema, congestion and secondary bronchopneumonia are sometimes present in the lungs.

Morbidity and Mortality

Rinderpest is highly contagious in species such as cattle; the classical form of the disease can affect all exposed animals within a short time. In endemic areas where animals had developed immunity from exposure or vaccination, rinderpest was often a disease of young animals. Maternal antibodies to rinderpest can persist for 6-11 months, and young animals may become ill after maternal immunity wanes but before they are vaccinated. During epidemics in naïve populations, the virus usually infects most susceptible animals. Rinderpest epidemics can affect different species at different rates. In some outbreaks, only a single species may be affected; in others, multiple concurrent epidemics can occur in different species at different rates. Outbreaks of rinderpest are self-limiting unless the virus can be passed from animal to animal in a large susceptible population. Animals that survive are immune for life.

Morbidity and mortality vary with the strain of the virus, and the susceptibility and immunity of the animal. In endemic areas, the morbidity rate was low and the clinical signs were frequently mild. However, in naïve animals, some strains can cause morbidity and mortality rates up to 100%. During the early stage of the outbreak, the mortality rate is often 10–20%, but it rises as more animals are exposed. Repeated epidemics that occurred in Eurasia before the virus was eradicated typically killed 30% of an affected herd. Lineage 2 strains can be mild and cause no mortality or significant morbidity in cattle, but these strains can cause severe disease with high morbidity and mortality rates in susceptible wildlife. For this reason, severe outbreaks in wildlife can be a sign that rinderpest viruses are being maintained in cattle populations.

Diagnostic Tests

Laboratory tests

Rinderpest virus can be isolated in B95a, a marmoset lymphoblastoid cell line, or other cell lines.

Rinderpest can also be confirmed by demonstrating viral antigens or RNA in clinical samples. Rinderpest antigens can be detected with various assays including agar gel immunodiffusion (AGID) tests, counterimmunoelectrophoresis or the immunocapture enzyme-linked immuno-sorbent assay (ELISA). The AGID test can be useful under field conditions, but it does not differentiate between rinderpest and peste des petits ruminants. The immunocapture ELISA can be used for definitive diagnosis and the differentiation of rinderpest from peste des petits ruminants. Antigens can be identified in tissues by immuno-peroxidase or immunofluorescence staining. Reverse-transcription polymerase chain reaction (RT-PCR) assays can be used to identify the virus, distinguish the three viral lineages, or differentiate rinderpest virus from peste des petits ruminants virus.

Serological tests include the competitive ELISA and virus neutralization. These tests can be used for surveillance, but cannot distinguish infected from vaccinated animals. An

indirect ELISA method has also been developed. [Note: This description of diagnostic tests was last updated in 2008, and it may not include assays developed after this time. Most laboratories in endemic areas are no longer likely to maintain rinderpest reagents for diagnostic testing.]

Samples to collect

Viremia can be seen a day or two before the fever begins, and can continue for 1-2 days after the fever begins to wane. Samples for virus isolation and antigen or RNA detection should ideally be collected when a high fever and oral lesions are present, but before the onset of diarrhea – the period when viral titers are highest. Blood (in heparin or EDTA) is the preferred sample for virus isolation in live animals. Whenever possible, samples should be submitted from more than one animal. Serum, swabs of lacrimal fluid, necrotic tissues from oral lesions, and aspiration biopsies of superficial lymph nodes should also be collected. At necropsy, samples should be taken from the spleen, lymph nodes (prescapular or mesenteric) and tonsil. The ideal post-mortem lesions come from an animal that has been euthanized during the febrile stage. A second choice would be a moribund animal that has been euthanized. Samples for RT-PCR can be taken from the lymph nodes, tonsils or blood (peripheral blood lymphocytes). The spleen is less desirable due to its high blood content. An additional set of tissue samples should be collected for histopathology and immunohistochemistry. In addition to other tissues, it should include the base of the tongue, retropharyngeal lymph node and third eyelid. Samples for virus isolation should be kept cold on ice during transport, but should not be frozen.

Control

Disease reporting

2016: Rinderpest was declared eradicated from the world, as of May 2011. If any rinderpest infections are identified, it is of the utmost importance to report them to national authorities immediately.

Prevention

At one time, rinderpest was controlled by annual vaccination of all cattle and domesticated buffalo more than a year of age. Maternal antibodies to rinderpest can persist for 6-11 months.

Rinderpest is usually introduced into an area by infected animals. Outbreaks can be controlled with quarantines and movement controls, euthanasia of infected and exposed animals, decontamination of infected premises, and intensive focal vaccination. Although it is not a desirable option, quarantine and ring vaccination without slaughter can also eradicate the disease. Vaccination for one strain is protective against all strains of the virus. Vaccinated animals should be marked.

Rinderpest virus is inactivated rapidly in the environment, and decontamination is not difficult. This virus can remain viable on unshaded pastures for six hours or shaded pastured for 18-48 hours. Bare enclosures usually

lose their infectivity within 48 hours and contaminated buildings within 96 hours. The FAO recommends that the premises, equipment and clothing be cleaned, then decontaminated with oxidizing agents such as sodium or calcium hypochlorite, or alkalis such as sodium hydroxide or sodium carbonate. Feces and effluents should be treated with sodium carbonate, before they are burned or buried. Pasteurization or heat treatment can inactivate the virus in milk. During an outbreak, carcasses from infected or exposed animals should be burned or buried. However, because rinderpest virus is inactivated quickly by autolysis and putrefaction, this virus is usually destroyed within 24 hours in carcasses. Restocking should be delayed for at least 30 days after cleaning and disinfection.

Internet Resources

[Food and Agriculture Organization of the United Nations. Manual on the preparation of rinderpest contingency plans](#)

[United States Animal Health Association. Foreign Animal Diseases](#)

[World Organization for Animal Health \(WOAH\)](#)

[WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals](#)

[WOAH Terrestrial Animal Health Code](#)

Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet. Spickler, Anna Rovid. 2016. *Rinderpest*. Retrieved from <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php>.

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