MORBILLIVIRUSES

(OTHER TAXONOMIC GROUPS)

AETIOLOGY

Classification of the causative agent

Morbilliviruses belong to the family *Paramyxoviridae*, and are enveloped, negative-sense single-stranded RNA viruses. Members of the genus *Morbillivirus* are able to infect a wide range of hosts to cause varied types of disease, many of which are severe.

Rinderpest virus (RPV, also known as “cattle plague”) and peste des petits ruminants virus (PPR, also known as “ovine rinderpest” and “goat plague”) are members of *Morbillivirus* known for their high virulence and mortality rates. These viruses are extremely transmissible and are considered significant risks to both agricultural industries and small communities that rely on susceptible species for food.

It is believed that RPV is the archetype morbillivirus and gave rise to the others, including distemper viruses and measles. Thanks to vaccination programmes, movement restrictions, and increased surveillance, rinderpest became the second viral disease to have been successfully eradicated (after smallpox). The OIE declared rinderpest eradicated worldwide in May 2011. However, this does not guarantee the absence of isolated, undetected geographic foci of viral persistence or the absence of viable samples in laboratories. With the help of the Food and Agriculture Organisation of the United Nations (FAO), the OIE is endeavouring to eradicate PPR by the year 2030.

For the purpose of voluntary reporting on non OIE-notifiable disease in wildlife, “infection with morbillivirus” refers to infection in **species other than domestic cattle, goats, and sheep**. Information on **domestic cattle, goats, and sheep** must be submitted through the mandatory reports for OIE-notifiable diseases.

Resistance to physical and chemical action

Temperature:	Steam cleaning is effective; survival can be prolonged by chilling or freezing tissues.
pH:	PPR is stable at a pH between 5.8-10.0. Rinderpest is stable at a pH between 4.0-10.0.
Chemicals/Disinfectants:	Susceptible to a 1:30 bleach dilution, potassium peroxymonosulfate, accelerated hydrogen peroxide, and aldehydes with contact times over 10 minutes. RPV is also susceptible to lipid solvents, and PPR is inactivated by alcohol.
Survival:	Environmental survival depends on ambient temperatures; colder temperatures prolong viability. PPR does not persist long outside of a host. RPV is extremely sensitive to UV radiation and dry conditions.

EPIDEMIOLOGY

Hosts

Rinderpest

- Order Artiodactyla (even-toed ungulates)
    - Wild and domestic yak and cattle (*Bos* spp.)
    - African buffaloes, Asian water buffaloes (*Syncerus caffer*l, *Bubalus bubalis*)
    - Giraffes (*Giraffa camelopardalis*)
    - Eland (*Taurotragus oryx*)
    - Kudu (*Tragelaphus* spp.)
    - Wildebeest (*Connochaetes* spp.)
    - Waterbuck (*Kobus* spp.)
    - Gazelles (*Gazella* spp.)
    - Bison (*Bison bonasus*)
    - Hippopotamuses (*Hippopotamus amphibius*)
    - Swine, especially bush pigs (*Potamochoerus porcus*) and warthogs (*Phacochoerus africanus*)
        - Wild pigs (*Sus scrofa*)
        - Asian pigs are believed to be more susceptible and epidemiologically significant than African and European pigs
    - Many antelope species
- Cervidae (cervids)
- Sheep and goats (*Ovis* spp., *Capra* spp.)
- Camelids (rarely)

Peste des petits ruminants

- Wild and domestic sheep and goats (*Ovis* spp., *Capra* spp.)
    - Laristan sheep (*Ovis orientalis laristanica*)
    - Ibex species (*Capra ibex*, *C. aegagus*)
    - Afghan markhor goat (*C. falconeri*)
    - Barbary sheep (*Ammotragus lervia*)
- Bovidae, including antelope
    - Gemsbok (Oryx *gazella*)
    - Dorcas gazelle (*Gazella dorcas*)
    - Nilgai (*Boselaphus tragocemalus*)
    - Gazelles (*Gazella* spp.)
    - Bushbuck *Tragelaphus scriptus*)
    - Impala (*Aepyceros melampus*)
    - Saiga antelope (*Saiga tatarica*, critically endangered)
- Camelids (rarely)
- White-tailed deer (*Odocoileus virginianus*) have been infected experimentally
- Cattle and pigs are susceptible to infection but do not shed virus

Transmission

Rinderpest

- Close contact (direct or indirect) with infected animals
- Swine can become infected by ingesting contaminated meat
- Inhalation of infectious droplets is limited and only possible under specific conditions
- Introduction into naïve areas is typically by movement of infected individuals

Peste des petits ruminants

- Close contact (direct or indirect) with infected animals
- Inhalation of infectious aerosols
- Contaminated water and food sources
- Fomites including contaminated environmental spaces
- Virus is excreted before clinical signs are detectable

Sources

Rinderpest

- Nasal and ocular secretions; saliva
    - Expired air also contains high concentrations of virions
- Urine and faeces
- Semen and vaginal discharge; milk
- Blood and all tissues are infectious before clinical signs appear

Peste des petits ruminants

- Respiratory and ocular secretions
- All secretions and excretions, including faeces, of infected animals

Occurrence

There are three lineages of RPV, all of which have been eradicated. Lineage 1 has only been detected in Africa and the Middle East, lineage 2 was found across Africa, and lineage 3 was found across Asia, including Russia and Turkey, and the Middle East. Europe became free of RPV in the early 20th century. Historically, the virus was maintained by cycles of infection between domestic cattle and African buffalo (*Syncerus caffer*), most often during periods of drought. Many speculated buffalo and other wild animals were able to carry the virus across great distances. However, it is now known that wildlife does not persistently harbour the virus, which suggests that introduction of an infected domestic animal is culpable for most outbreaks in naïve cattle herds.

There are 4 lineages of PPR, all of which belong to the same (and only) serogroup. Lineages 1 and 2 are predominantly located in West Africa, 3 spans across East Africa, the Middle East, and southern India, and 4 ranges from the Middle East to the Tibet Autonomous Region of China. The virus appears to be more prevalent in humid regions. Disease in wildlife appears to not be significant in West Africa for reasons that are not completely understood. PPR is believed to be a significant risk to Asian wildlife, especially in geographic areas near mountain ranges. However, the Himalayas are preventing the disease from spreading north and are therefore protecting a number of endangered bovid species in central Asia.

Animals that have recovered from RPV or PPR infection develop lifelong immunity to reinfection. Small ruminants vaccinated for or infected with RPV develop cross-protection against PPR.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information System - Wild (WAHIS-Wild) Interface [http://www.oie.int/wahis_2/public/wahidwild.php/Index].

DIAGNOSIS

The incubation period for RPV is 1-2 days (some sources report 4-5 days with a broader range of 3-15 days to include outliers), after which follows a prodromal phase and subsequent increasing severity of clinical signs. Young animals are more susceptible to disease. Once enzootic in a population, RPV infections become much milder and are typically only seen in neonates and yearlings; if all ages are affected, it is considered an outbreak in a naïve population.

The reported duration of PPR's incubation period varies by source within the range of 1-6 days. The severity of disease and mortality rate are variable by species and breed; sheep are considerably more resistant to serious illness, and goat breeds have differential morbidity rates. Young animals are more susceptible to disease.

Clinical diagnosis

Animals infected with RPV become markedly depressed, febrile, and dehydrated. Ocular and nasal discharge ranging from serous to mucoid to mucopurulent is apparent; the discharge may darkly stain an animal's face. Some animals have been observed with increased corneal opacification and ulceration. Diarrhoea is common and may be haemorrhagic and projectile in nature. Animals may cough. Buffalo have been documented drooling and frothing at the mouth as well as developing behavioural changes such as anorexia and increased aggression. Most notable are the necrotic erosions and ulcers that develop inside the nares and throughout the oral mucosa, including the hard and soft palate. These lesions can contribute to the development of foul-smelling breath. Buffalo between 7-18 months develop severe erythaema and maculopapular rashes on the axilla and perineum, parakeratosis, skin flaking, and desquamation on their flanks and back. Buffalo (any age) may also develop raised skin nodules ≤1cm in diameter. Several affected herds of buffalo showed signs of abortion with retained placentas. Peracute death is possible.

Animals infected with PPR experience a sudden increase of body temperature and become unthrifty, anorectic, and mentally dull. Mucous membranes (including the conjunctiva) become congested, and the muzzle becomes dry. Animals develop a serous nasal discharge that later becomes mucopurulent; this causes the animal's breath to develop an unpleasant odour. Similar to RPV, PPR causes necrotic plaques on mucous membranes and within the nasal cavity. Most commonly, stomatitis involves the lower lip and mandibular gingiva, but the dental pad, palate, buccal and lingual mucosa, and oral papillae can be involved in more serious cases. Severe catarrhal conjunctivitis may cause matting of the eyelids. Animals may develop a cough and dyspnoea due to bronchopneumonia. Abortions may occur. Diarrhoea is common and is often haemorrhagic. Animals often succumb to emaciation, dehydration, and hypothermia by 5-10 days, but peracute death may occur before clinical signs are observed.

Lesions

Rinderpest

- Emaciation
- Corneal opacification and ulceration; sunken eyes
- Hypopyon, keratitis, uveitis, iridocyclitis, cataracts
- Stomatitis
    - Necrotic erosions/ulcers of the buccal and lingual mucosa, inside the nares, on the lips and gingiva, and on the hard and soft palates
- Dry, cracked muzzle
- Dermatitis
    - Erythaema and maculopapular rashes on the axilla and perineum, hyper- and parakeratosis, skin flaking, desquamation on flanks and back (young buffalo)
        - Squamous metaplasia of sebaceous glands
    - Raised nodules ≤1cm in diameter that resemble lumpy-skin disease or papillomatosis (buffalo of any age)
    - Necrosis of epithelial cells
- Lymphadenopathy
    - White necrotic foci in Peyer's patches +/- haemorrhage
- Necrotising erosions, congestion, and/or hemorrhage throughout the gastrointestinal tract
    - "Zebra-striped" or "tiger-striped" appearance of the rectal mucosa
- Distended gallbladder +/- haemorrhage and congestion in the mucosa
- Necrosis of urogenital mucosa
- Lesions in the lungs are rare; congestion is most common
- Mild periportal hepatitis
- Necrosis of renal tubules, salivary gland, bile duct, pancreas (kudu)
- Syncytia formation +/- inclusion bodies

Peste des petits ruminants

- Emaciation
- Catarrhal conjunctivitis +/- matting of the eyelids
- Stomatitis

    - Necrotic plaques and erosions on the lower lip and mandibular gingiva, buccal mucosa and commissures, ventral surface of the tongue
    - The hard palate and pharynx may be involved in severe cases
    - Erosions are shallow and range from red at their base to pinkish white at their margins where they are sharply bounded and demarcated by healthy epithelial tissue (unless foci have enlarged to coalesce).
- Vulvar and vaginal congestion and erosion
- Abomasal ulcers and sharply demarcated red erosions that ooze blood
- Haemorrhagic streaks and erosions in the duodenum and terminal ileum (uncommon)
- Sloughing of lymphoid tissue
    - Oedematous and friable lymph nodes, rarely enlarged or haemorrhagic
    - Necrosis of Peyer's patches
    - Congested and firm spleen
    - Lymphoid depletion
- Congestion of mucosal folds around the ileocaecal valve and rectum
    - "Zebra-striped" appearance of the mucosa
- Petechial haemorrhage and congestion of the turbinates, larynx, trachea
- Bronchopneumonia
- Rarely, hepatocellular necrosis and glomerulonephritis
- Hydropic degeneration of stratum granulosum cells
- Multinucleated giant cells +/- inclusion bodies

Differential diagnoses

- Bovine herpesvirus-2
- Bovine viral diarrhoea
- *Theileria* spp. infection
- Malignant catarrhal fever
- Bovine papular stomatitis
- Bluetongue virus
- Foot-and-mouth disease
- Contagious caprine pleuropneumonia
- Heartwater disease (*Ehrlichia ruminantium*)
- Orf (contagious pustular dermatitis, contagious ecthyma)
- Vesicular stomatitis virus
- Swine vesicular disease
- Nairobi sheep disease
- Coccidiosis
- Nematodiasis
- Bacterial enteritis (*Salmonella* spp., *Escherichia coli*)
- Bacterial pneumonia
- Mineral poisoning

Laboratory diagnosis

Samples

For isolation of agent

- Lymph node biopsy
    - Place in phosphate-buffered saline or cell culture medium
    - Avoid glycerol, a common component of viral transport medium, because it inactivates RPV.
- Tissues to collect postmortem:
    - Lymph and haemal nodes
    - Spleen
    - Lung
    - Affected sections of the alimentary tract

    - Place on ice and send immediately to the testing facility. If not possible, freezing at -20°C is acceptable.

Serological tests

- Whole uncoagulated and coagulated blood, serum
    - Place on ice and expedite transportation to the testing facility
    - Do not freeze uncoagulated blood.
- Nasal, oral, and ocular swabs
    - Swabs should be placed in phosphate-buffered or normal saline (+/- RNAse) solution to prevent dehydration.

Procedures

Identification of the agent

- Virus isolation via cell culture
    - Definitive for PPR, but is labour intensive
- Reverse-transcriptase polymerase chain reaction (RT-PCR)
- Immunofluorescence
- Immunohistochemistry

Serological tests

- The current gold standard for PPR is the virus neutralization test (VNT). It is not commonly used except in exceptional circumstances, as it is costly and impractical in most national diagnostic laboratories. Consequently, there is a need to review alternative gold standards for this disease.
- Agar gel immunodiffusion (AGID)
- Antibody-capture enzyme-linked immunoassay (ELISA) on paired sera
    - Concessions are typically made for wildlife where obtaining paired sera is difficult; an adequately high titre in an animal from a population with consistent clinical signs is sufficient.
    - PPR antibodies cross react with RPV.
- None of the commercially available diagnostic tests for PPR serology are validated for wildlife species, and there are unanswered questions regarding their sensitivity and specificity with wildlife serum samples. New serological test methodologies have been published such as Luciferase Immunoprecipitation System (LIPS) and Pseudotype Virus Neutralization Assay (PVNA) that could be useful in this context, including potentially as the gold standard to replace the current PPR VNT. Clear guidelines and standards for application of PPR diagnostics tests in wildlife species will need to be established using true positive and negative sera and across species.

PREVENTION AND CONTROL

There are currently few tools with which to effectively manage PPR in free-ranging wildlife populations. Vaccination of free-ranging wildlife at numbers sufficient to establish herd immunity is neither practical nor feasible. If the virus is detected in wildlife in your jurisdiction, contacting international experts and discussing options is advised.

Currently, the best control efforts involve strict management of the interface between wildlife and livestock populations. The ultimate goal is to prevent spillover of disease into wildlife and spill-back where wildlife populations are sufficient to distribute the virus. The FAO/OIE eradication campaign aims to control and eliminate PPR in domestic animals, especially in areas where wildlife species abundance and richness is high, and prevent interactions between livestock and free-ranging wildlife.

Sanitary prophylaxis

- Contaminated areas in captive facilities should be cleaned and disinfected with appropriate solvents.

- Prevent contact between susceptible wild and domestic species by utilising proper biosecurity protocols, namely adequate fencing for livestock.
- Because these viruses are short-lived in the environment, movement restrictions that incorporate testing and quarantines of susceptible domestic species can be effective measures in preventing introduction into a naïve area.
- Burn or bury the carcasses of livestock in areas where RPV and PPR are a concern.

Medical prophylaxis

- All animals introduced to a captive facility should be held in quarantine for at least three weeks; this also applies to pre-shipping quarantines.
- Animals that develop clinical signs in captivity should be group-housed in quarantine until 1 month after recovery of the last clinical case.
- Multiple RPV and PPR vaccines are available for domestic animals.
    - National vaccination programmes should take virus epidemiology and transmission risks into consideration.
    - Attenuated RPV vaccines (or infection with RPV) utilised in small ruminants are cross-protective against PPR and have been successfully utilised in susceptible wild species.
    - There is little information about domestic sheep/goat PPR vaccines in wild animals.
    - Depending on the vaccine type used, there may be complications in effectively interpreting serologic assays performed on a vaccinated animal.
    - There is evidence that vaccinating domestic herds for PPR may help ameliorate outbreaks in susceptible wild species in the same region.
    - Vaccination of suids and non-domestic captive ruminants is recommended.

POTENTIAL IMPACTS OF DISEASE AGENT BEYOND CLINICAL ILLNESS

Risks to public health

- PPR does not infect people, and there have not been reports of RPV infection in humans.

Risks to agriculture

- RPV and PPR have the potential to cause mass morbidity and mortality in livestock species; outbreaks have significant implications for agricultural industries and small communities that rely on livestock for food.

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Science Department (scientific.dept@oie.int). Last updated 2019. Written by Marie Bucko and Samantha Gieger with assistance from the USGS National Wildlife Health Center.