

RIFT VALLEY FEVER

AETIOLOGY

Classification of the causative agent

Rift Valley fever (RVF) virus is a negative-sense, single-stranded RNA virus of the family Bunyaviridae within the genus *Phlebovirus*. Only one serotype is recognised but strains exist of variable virulence.

Resistance to physical and chemical action

Temperature:	Virus recoverable from serum after several months at 4°C or 120 minutes at 56°C.
pH:	Resistant in alkaline environments but inactivated at pH <6.8.
Chemicals/Disinfectants:	Inactivated by lipid solvents (i.e. ether, chloroform, sodium deoxycholate), low concentrations of formalin and by strong solutions of sodium or calcium hypochlorite (residual chlorine should exceed 5000 ppm).
Survival:	Survives in freeze dried form and aerosols at 23°C and 50–85% humidity. Virus maintained in the eggs of certain arthropod vectors during inter-epidemic periods. Can survive contact with 0.5% phenol at 4°C for 6 months

EPIDEMIOLOGY

RVF is a vector-borne disease of sheep, cattle and goats; susceptibility of different breeds to RVF varies considerably. The disease usually presents in an epizootic form over large areas of a country following heavy rains and sustained flooding, and is characterised by high rates of abortion and neonatal mortality, primarily in sheep, goats and cattle.

Hosts

- Cattle, sheep, goats, dromedaries, several rodents
- Wild ruminants, buffaloes, antelopes, wildebeest, etc.
- Humans are very susceptible (major zoonosis)
- African monkeys and domestic carnivores present a transitory viraemia

Transmission

- RVF virus regularly circulates in endemic areas between wild ruminants and haematophagous mosquitoes; disease is usually inapparent
- Certain *Aedes* species act as reservoirs for RVF virus during inter-epidemic periods and increased precipitation in dry areas leads to an explosive hatching of mosquito eggs; many of which harbour RVF virus
- Precipitation cycles of 5–25 years produce RVF-immuno-naïve animal populations and when combined with introduction of virus, explosive outbreaks of the disease occur
 - satellite imaging has been used to confirm historic importance of precipitation in RVF outbreaks and in forecasting high-risk areas for future outbreaks
- Infected *Aedes* feed preferentially on domestic ruminants which act as an amplifier of RVF
 - broad vector range of mosquitoes (*Aedes*, *Anopheles*, *Culex*, *Eretmapodites*, *Mansonia*, etc.) coupled with increased circulating virus leads to expansion of disease
 - extrinsic incubation also occurs in vectors.
- Sylvatic cycle and inter-epidemic maintenance also occurs in some areas
- Direct contamination: occurs in humans when handling infected animals and meat
- Mechanical transmission by various vectors has been demonstrated in laboratory studies

Sources of virus

- For animals: wild fauna and vectors
- For humans: nasal discharge, blood, vaginal secretions after abortion in animals, mosquitoes, and infected meat; also by aerosols and possibly consumption of raw milk

Occurrence

RVF is endemic in tropical regions of eastern and southern Africa. Epizootic outbreaks in Africa among peri-endemic countries have been associated with above average rain fall and climatic conditions favourable for competent vectors. Important outbreaks of RVF have been recorded in Egypt (1977–78 and 1993), Mauritania (1987), Madagascar (1990–91), Kenya and Somalia (1997). RVF was recognised for the first time outside of the African continent in 2000 with outbreaks reported in Saudi Arabia and Yemen. The disease has not established itself in the Arabian Peninsula but sero-positive animals have been detected. RVF activity was recorded from 2002 to 2004 at various locations in Senegal, Mauritania and Gambia. Madagascar and Swaziland have most recently reported RVF in 2008 and later that year also South Africa.

DIAGNOSIS

Incubation period varies from 1 to 6 days; 12–36 hours in lambs. For the purposes of the Terrestrial Code, the infective period for RVF is considered 30 days.

Clinical diagnosis

Severity of clinical disease varies by species: lambs, kids, puppies, kittens, mice and hamsters are considered “extremely susceptible” with mortalities of 70–100%; sheep and calves are categorised as “highly susceptible” with mortality rates between 20–70%; in the “moderately susceptible” category are cattle, goats, African buffalo, domestic buffalo, Asian monkeys and humans with mortalities less than 10%; and camels, equids, pigs, dogs, cats, African monkeys, baboons, rabbits, and guinea pigs are considered “resistant” with infection being inapparent. Birds, reptiles and amphibians are not susceptible to RVF. Signs of the disease tend to be non-specific; however, the presentation of numerous abortions and mortalities among young animals, together with influenza-like disease in humans, is indicative.

Cattle

- Calves (highly susceptible)
 - fever (40–41°C)
 - inappetence
 - weakness and depression
 - bloody or fetid diarrhoea
 - more icterus than in lambs
- Adults (moderately susceptible):
 - often inapparent infection but some acute disease
 - fever lasting 24–96 hours
 - dry and/or dull coat
 - lachrymation, nasal discharge and excessive salivation
 - anorexia
 - weakness
 - bloody/fetid diarrhoea
 - fall in milk yield
 - abortion rate may reach 85% in the herd

Sheep

- Newborn lambs or under 2 week of age (extremely susceptible):
 - biphasic fever (40–42°C); fever subsides just prior to death

- anorexia; in part due to disinclination to move
- weakness, listless
- abdominal pain
- rapid, abdominal respiration prior to death
- death within 24–36 hours
- Lambs over 2 weeks of age (highly susceptible) and adult sheep
 - peracute disease: sudden death with no appreciable signs
 - acute disease more often in adult sheep
 - fever (41–42°C) lasting 24–96 hours
 - anorexia
 - weakness, listlessness and depression
 - increased respiratory rate
 - vomiting
 - bloody/fetid diarrhoea
 - mucopurulent nasal discharge
 - icterus may be evident in a few animals
 - in pregnant ewes, 'Abortion storms' with a rates approaching 100%

Goat

- Similar to adult sheep (see above)

Lesions

- Focal or generalised hepatic necrosis (white necrotic foci of about 1 mm in diameter)
- Congestion, enlargement, and discoloration of liver with subcapsular haemorrhages
- Brown-yellowish colour of liver in aborted fetuses
- Widespread cutaneous haemorrhages, petechial to ecchymotic haemorrhages on parietal and visceral serosal membranes
- Enlargement, oedema, haemorrhages and necrosis of lymph nodes
- Congestion and cortical haemorrhages of kidneys and gallbladder
- Haemorrhagic enteritis
- Icterus (low percentage except in calves)

The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated October 2009.