Foot and Mouth Disease

Fiebre Aftosa

Last Updated: July 2025

Importance

Foot and mouth disease (FMD) is a highly contagious viral disease that mainly affects cloven-hooved livestock and wildlife. Although adult animals generally recover, the morbidity rate can be very high in naïve populations, and some animals experience significant pain and distress. Sequelae such as decreased milk yield, permanent hoof damage and chronic mastitis are possible, and high mortality rates can sometimes be seen in young animals or certain wildlife species. Foot and mouth disease was once found worldwide, but it has been eradicated from some regions including all of North America and western Europe. Where it is endemic, this disease causes production losses and is also a major constraint to the international livestock trade. Unless strict precautions are followed, the virus can be readily re-introduced into disease-free regions via animals or animal products. It may then spread rapidly, particularly if livestock densities are high or detection is delayed. Outbreaks can severely disrupt livestock production, result in embargoes by trade partners, and require significant resources to control. Direct and indirect economic losses equivalent to several billion U.S. dollars are not uncommon. Since the 1990s, a number of outbreaks have occurred in FMD-free countries. Some, such as the 2001 outbreak in the U.K. were devastating due to the large numbers of animals culled in control efforts.

Etiology

Foot and mouth disease virus (FMDV) is a member of the genus *Aphthovirus* in the family Picornaviridae. It has seven major serotypes: O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1. Some serotypes are more variable than others, but collectively, they contain more than 60 strains. While most strains affect all susceptible host species, some (e.g., the pig-adapted O Cathay strain) have a more restricted host range. Immunity to one FMDV serotype does not protect an animal from other serotypes. Protection from other strains within a serotype varies with their antigenic similarity.

Serotype O viruses have caused many of the recent outbreaks and incursions into FMD-free countries; however, any serotype can cause an outbreak, and multiple serotypes co-circulate in many endemic regions. Serotype C has not been isolated since 2004 and may have disappeared. Nevertheless, this remains to be conclusively demonstrated, as some limited serological evidence suggests that it (or reintroduced serotype C vaccine strains) might still occur in parts of Africa, and its previous emergence was after 9 years of inapparent circulation in the Amazon region of South America.

Species Affected

FMDV mainly affects members of the order Artiodactyla (cloven-hooved mammals). Most species in this order are thought to be susceptible to some degree, though the frequency and severity of the illness can vary. Cattle, pigs, sheep, goats, water buffalo, yaks and mithun (*Bos frontalis*) are often affected by this virus. Some camelids are also susceptible, and outbreaks have been seen in farmed cervids such as reindeer (*Rangifer tarandus*), deer and elk (*Cervus elaphus nelsoni*). Cattle are important maintenance hosts in most areas, but a few viruses are mainly adapted to pigs, and some isolates might circulate in water buffalo. It is uncertain whether small ruminants can maintain FMDV for long periods if cattle are absent.

This virus has also been reported in a number of artiodactylid wildlife including African buffalo (*Syncerus caffer*), bison (*Bison* spp.), chamois (*Rupicapra rupicapra*), giraffes (*Giraffa camelopardalis*); cervids such as moose (*Alces alces*), elk and deer, various wild suids (e.g., wild boar, warthogs, African bush pigs) and a number of antelope species, such as wildebeest (*Connochaetes gnou*) blackbuck (*Antilopa cervicapra*), kudu (*Tragelaphus strepsicornis*), impala (*Aepyceros melampus*), pronghorn, (*Antilocapra americana*) and oryx (*Oryx* spp.). While most wildlife are infected when FMDV viruses spill over from livestock and do not seem to maintain these viruses themselves, African buffalo are important reservoir hosts for the SAT serotypes.

Some other mammals including dogs, hedgehogs (both *Erinaceus europaeus* and *Atelerix prurei*), bears, armadillos and kangaroos, as well as nutrias (*Myocastor coypus*),

The Center for Food Security & Public Health

Institute for International Cooperation in Animal Biologics

Iowa State University College of Veterinary Medicine

World Organisation for Animal Health Founded as OIE

USDA

© 2001-2025

capybaras (*Hydrochaerus hydrochaeris*) and other rodents are also known to be susceptible to FMDV, though reports of clinical cases are rare. Several cases have been seen in captive Asian elephants (*Elephas maximus*); however, there are few descriptions of infections in African elephants *(Loxodonta africana*), and the latter species is not considered susceptible under natural conditions in southern Africa. Experimental infections have been reported in cats and wild rabbits, but to date, there are no reports of naturally acquired cases in these species. Currently, there is no evidence that non-artiodactylid mammals are significant in the epidemiology of foot and mouth disease. Some old reports suggested that European hedgehogs (*Erinaceus europaeus*) might acquire this virus from cattle and transmit it back, but more recent studies found no evidence that hedgehogs have helped to propagate this virus in the last 60 years. Rodents and dogs are likewise not thought to be epidemiologically important hosts, except as mechanical vectors.

Zoonotic potential

FMDV can occasionally infect people, but clinical cases seem to be rare and any symptoms are usually mild.

Geographic Distribution

Foot and mouth disease is endemic in parts of Asia, Africa, the Middle East, and South America, with significant progress reported toward eradication in South America. While serotypes O and A are widely distributed, SAT viruses occur mainly in Africa, with periodic incursions into the Middle East, and Asia 1 is currently found only in Asia.

Transmission

FMDV can be found in all secretions and excretions from acutely infected animals including expired air, saliva, milk, urine, feces and semen, as well as the fluid from FMD-associated vesicles, and amniotic fluid and aborted fetuses. Peak virus production usually occurs around the time the vesicles rupture and most clinical signs appear; however, some animals can excrete FMDV for up to four days before the onset of clinical signs, and those that develop subclinical infections[1] can also shed the virus.

FMDV mainly enters the body by inhalation or ingestion, but it can also infect an animal through other mucous membranes or skin abrasions. Most animals are thought to become infected via direct or close contact, but local aerosol transmission can also occur within a farm. Cattle are particularly susceptible to aerosolized viruses, while pigs require much higher doses to be infected by this route. Long distance airborne transmission may occasionally occur under favorable climatic conditions, with a few reports of FMDV spreading up to a hundred miles or more over water. Transmission over land is thought to be more limited, and rarely more than 10 km (6 miles). Because pigs produce large amounts of aerosolized virus, the presence of large herds of infected swine may increase the risk of airborne spread. Transplacental transmission has been documented in sheep and cattle; however, one study indicated that FMDV is probably not transmitted by embryo transfer of washed bovine embryos.

Mechanical transmission by fomites and living (e.g., animal or human) vectors can also cause outbreaks; however, there is limited information on the survival of FMDV in the environment, with much of this information collected more than 50 years ago. Most studies suggest that, at temperatures of 20°C (68°F) or above, FMDV remains viable for a few days to a week or two, and occasionally up to a month, on (or in) various fomites, vegetation, soil or water, with some studies indicating that it may persist for long as 2-6 months at cold or freezing temperatures. The presence of organic material promotes longer survival. There are also a few outliers that remain to be confirmed, such as an old study that found this virus in dry hay at 22°C (72°F) after 20 weeks of storage. How long animals can be infected from contaminated environments is still unclear. While FMDV has been reported to remain viable in stalls for 2 weeks, calves did not become infected in a recent study when they were placed in a room (18-20°C) that had contained infected animals 24 hours earlier, although viable virus was recovered from various sites in the room, with a half life ranging from 3 to 7 days, and calves moved into the room without a waiting period became infected.

FMDV can sometimes persist for prolonged periods in meat, milk and other animal products when the pH remains above 6.0. It is inactivated by acidification of muscles during rigor mortis, but because the pH does not drop to this extent in some tissues, such as bone marrow and lymph nodes, it may survive longer when these tissues are also present. One recent study found that, while infectious virus could not be recovered from the muscles of euthanized pigs after 7 days of carcass storage at 4°C (39°F), it was isolated from the epithelium of vesicles on these carcasses for at least 11 weeks. Older studies reported that, in animal tissues kept at 1-7°C (34-45°F), FMDV remained viable for 1-3 days in muscle, 7 months (but less than 12 months) in bone marrow, up to 4 months in lymph nodes, about 2 months in blood, 8-42 days in various internal organs (e.g., spleen, lung, stomach, kidney) and 8 days in dried hides. This virus is also reported to survive for 4-14 days in salt-cured bacon, tongue or sausages, 46-89 days in ham and a year in salt-cured hides.

Humans as vectors for FMDV

People can act as mechanical vectors for FMDV by carrying the virus on clothing or skin. The virus might also be carried for a short time in the nasal passages, although several studies suggest prolonged carriage is unlikely. In one

[1] Occasionally referred to as a 'neoteric' infection

© 2001-2025

early study, nasal carriage was reported for up to 28 hours but less than 48 hours after contact with infected animals. In two subsequent reports, people did not transmit serotype O viruses to pigs or sheep when personal hygiene and biosecurity protocols were followed, and no virus could be detected in nasal secretions 12 hours after contact with infected animals. Another study found FMDV nucleic acids (serotypes O or Asia 1) in only one person tested 16-22 hours after exposure to infected animals, and live virus could not be isolated from this sample. One caveat is that, because factors such as sub-optimal facility sanitation or poor compliance with personal hygiene and biosecurity protocols could also influence transmission to animals, these studies might not apply directly to the situation in the field. Anecdotally, there are reports that some outbreaks at a zoo in Europe may have been associated with personnel from a nearby vaccine production facility taking their lunch breaks at the zoo; however, this occurred at a time when laboratory biosecurity protocols were less stringent than they are currently.

Carriers

Some animals clear FMDV completely after an infection, while others, which are called carriers, can remain persistently infected for a time. Virus carriage can occur whether or not an animal was symptomatic, and vaccinated animals can also become carriers. The formal definition of an FMDV carrier is an animal in which the virus can be found for more than 28 days after it became infected. In a recent experiment, calves either completely cleared the virus or became a carrier within 21 days if they were unvaccinated, and by 10 days if vaccinated. Whether transplacentally infected animals might also become carriers is unclear. In one study, live virus could not be found in healthy calves infected *in utero*, though viral RNA was detected for up to 8 months after birth.

In carrier cattle, live FMDV only persists in the nasopharyngeal mucosa. It can be harbored in both the nasopharyngeal mucosa and tonsils of some other species, such as African buffalo and sheep. The viruses in carriers appear to be cell-associated, and can be found in esophageal-pharyngeal fluid collected with a probang cup, in a process that scrapes cells as well as fluid from the nasopharyngeal region. Live virus is not usually found at any other site or in other secretions or excretions (e.g., oral or nasal swabs), though one study reported virus isolation from the nasal fluid of some experimentally infected water buffalo for up to 70 days.

The epidemiological significance of livestock FMDV carriers is still uncertain and controversial. To date, only African buffalo carrying SAT viruses have been demonstrated to transmit FMDV to animals in close contact. Some authors have speculated that sexual transmission might have been involved in these incidents, as FMDV can be found in semen and all successful experiments included both bulls and cows. If livestock carriers ever transmit the virus, this appears to be rare. Although there are a few old anecdotal reports of apparent transmission from carrier cattle in the field, recent field studies that monitored recovered herds have found no evidence for any infections, including in calves born after these outbreaks. Almost all laboratory experiments also failed to detect transmission from carrier cattle to susceptible contacts, though esophageal-pharyngeal fluid is infectious if it is injected directly into an animal (or deposited into the nasopharynx, at least when virus titers in carriers are at their peak, 30 days after inoculation). A single older study did document seroconversion in a few pigs housed with carrier cattle; however, some sources question whether these pigs might have been exposed accidentally from other experiments being conducted in the facility.

How long an animal remains a carrier varies with the species and the individual. Most cattle carry FMDV for 6 months or less, with declining titers over this period, but some individuals can remain persistently infected for up to 3.5 years. The virus has been found by virus isolation or PCR for up to 12 months in sheep (though most seem to be carriers for only 1 to 5 months), up to 4 months in goats, for a year in water buffalo, and up to 8 months in yaks (*Bos grunniens*). Individual African buffalo can be carriers for at least 5 years, and FMDV persisted in one herd of African buffalo for at least 24 years. Some deer have been reported to carry FMDV for up to 2.5 months. Pigs and camelids are not thought to become carriers; however, only limited research has been done in camelids.

Disinfection

Disinfectants reported to be effective against FMDV include sodium hydroxide, sodium carbonate, some acids (e.g., acetic acid, citric acid), sodium hypochlorite and accelerated hydrogen peroxide, as well as various commercial multi-component disinfectants such as Virkon-S.™ The disinfectant concentration and time needed can differ with the surface type, presence of organic matter and other factors. Heat treatment of 67-68°C (153-154°F) for less than 10 minutes was reported to destroy viruses in fecal slurry, as well as spiked meat slurry (pH 6.6) used in making pet food, while viruses in homogenized bovine tongue epithelium from an FMDV-infected animal or an FMDV-spiked slurry and meal mix adjusted to pH 6.7 were destroyed within a minute at 79°C (174°F). FMDV is also inactivated at pH below 6.0 or above 9.0.

Incubation Period

Reported incubation periods range from 1-2 days to about 2 weeks for most species, and possibly as long as 3 weeks in some water buffalo, with most animals developing clinical signs several days (e.g., 3-5 days) after exposure to infected contacts.

Clinical Signs

Infections with FMDV range from subclinical to severe. While there is some variability in the clinical signs, foot and mouth disease is usually an acute febrile illness characterized by vesicles (blisters) on the feet, in and around the mouth, on the mammary gland, and occasionally at other sites such as the vulva, prepuce, or pressure points on the legs. The typical pattern of affected sites varies with the species. Vesicles

© 2001-2025

usually rupture rapidly, becoming erosions and ulcers, and the resulting pain and discomfort can lead to depression, anorexia, excessive salivation, lameness and reluctance to move or rise. In severe cases, the hooves, footpads or even the horns may be sloughed. Lesions on the coronary band may also cause growth arrest lines on the hoof. Reproductive losses have been seen most often in small ruminants, but can also occur in other species.

In livestock, deaths are uncommon except in young animals, which may die from myocarditis or starvation, for instance when they are unable to nurse. However, deaths of all ages have been reported during some severe outbreaks in wildlife. Most adult livestock recover within 2-3 weeks, though secondary infections may slow recovery. Possible complications include temporary or permanent decreases in milk production, hoof malformations, chronic lameness or mastitis and loss of condition/ reduced productivity. Whether subclinical myocarditis can occur in adult livestock does not seem to have been investigated.

Cattle

While mild illnesses and subclinical infections are possible in cattle, particularly in endemic regions, naive animals are often significantly affected by FMD. Clinical signs are particularly severe in the highly productive breeds found in developed countries. Cattle often develop vesicles, erosions and ulcers at multiple sites including the tongue, dental pad, gums, soft palate, nostrils and/or muzzle, as well as the coronary band and interdigital space of the hooves. Lesions at the base of the horn have also been reported, and may result in separation of the horn in severe cases. Oronasal lesions often cause reluctance to eat, profuse salivation and nasal discharge, which is initially mucoid but becomes mucopurulent. Affected animals sometimes lose condition rapidly and may have gradual or sudden decreases in milk production. In some cases, milk may not be produced again until the next lactation, or milk yield may be lower indefinitely. Young calves sometimes die of heart failure without developing any vesicles.

While reproductive losses do not seem to occur as often in cattle as sheep, widespread abortions and neonatal deaths from myocarditis were reported in two Indian herds that had been regularly vaccinated for FMD and experienced an outbreak. However, abortions were seen in both clinically affected and asymptomatic cows in this outbreak, and it could not be confirmed that FMDV was the sole cause. In areas where cattle are intensively vaccinated, the entry of FMD into a herd can sometimes cause swelling of the tongue and severe clinical signs that resemble an allergic disease.

Cattle that recover from FMD have occasionally been reported to develop an unusual sequela called heat-intolerance syndrome (HIS, also ‘hairy panters’). This poorly understood syndrome is characterized by abnormal hair growth with failure of normal seasonal shedding, pronounced panting with elevated body temperature and pulse rate during hot weather, and failure to thrive. Some affected animals are reported to have low body weight, severely reduced milk production and reproductive disturbances. Animals with HIS do not appear to recover. The pathogenesis of this syndrome is not known, and a definitive link with FMD has not been established, but endocrine disturbances were suspected by some investigators.

Water buffalo

FMD in water buffalo resembles the illness in cattle, with both mouth and foot lesions; however, the clinical signs are reported to be milder in most outbreaks, the mouth lesions may be relatively small, with scant fluid, and the lesions often heal more rapidly. Nevertheless, there is at least one report of a farm where buffalo calves were more severely affected than cattle calves of a similar age. In one study, foot lesions were more likely to occur on the bulb of the heel than in the interdigital space.

Suids

In pigs, the most severe lesions usually occur on the feet, and the first signs of FMD may be lameness and blanching of the skin around the coronary bands. Vesicles then develop on the coronary band and heel, and in the interdigital space. In some cases, the lesions may become so painful that pigs crawl rather than walk. The horns of the digits are sometimes sloughed. Mouth lesions are usually small and relatively mild in swine, and drooling is rare. Vesicles are also found sometimes on the snout or udder, as well as on the hock or elbows if the pigs are housed on rough concrete floors. Nonspecific signs of illness can include a decreased appetite, lethargy and huddling, but fever is sometimes transient, low or absent. Young pigs up to 14 weeks of age may die suddenly from heart failure; piglets younger than 2 months are particularly susceptible.

Lesions may be milder or less apparent in feral pigs and wild boar than domestic pigs, in part due to their thicker skin and long, coarse hair. However, the pattern of the lesions is overall similar.

Sheep and goats

Foot and mouth disease is mostly a mild illness in sheep and goats, and a significant number of animals can be subclinically infected or develop lesions only at one site. In many cases, the clinical signs are limited to fever and mild to severe lameness of one or more legs. However, foot lesions can sometimes be missed, as they may be hidden by secondary infections or lesions from other causes, which are common in sheep. Any lesions in the mouth generally appear as shallow erosions. Vesicles may also be noted sometimes on the teats, and rarely on the vulva or prepuce, and may be accompanied by a drop in milk production or reluctance to mate, respectively.

In some outbreaks, significant numbers of ewes abort or give birth to stillborn lambs. Abortions can occur at any stage of gestation. Young lambs and kids sometimes die from emaciation or heart failure (vesicles may be absent), with signs such as fever, tachycardia, marked abdominal respiration and collapse. There have been reports of incidents where large numbers of lambs with myocarditis fall down dead when stressed.

© 2001-2025

Camelids

Experimentally infected llamas and alpacas are mostly reported to have mild clinical signs or develop subclinical infections, though severe cases have been reported. Mild signs were seen in alpacas during one FMD outbreak in Peru, but the virus could not be isolated and these cases are unconfirmed.

Two experimentally infected Bactrian camels developed moderate to severe clinical signs, with hindleg lesions that included swelling and exudation of the footpad, but no oral lesions. However, mouth lesions and salivation, as well as severe footpad lesions and skin sloughing at the carpal and tarsal joints, knee pads and chest were reported in Bactrian camels during outbreaks in the former U.S.S.R. Detachment of the soles of the feet was noted in several reports.

Other species

The clinical signs in artiodactylid wildlife resemble cases in domestic livestock. In some cases, lesions have been noted to affect sites where there is frequent mechanical trauma, such as the feet and snout of suids or the carpal joints of warthogs. Loss of the horns has been seen in some ungulates. Infections with SAT-type viruses in African buffalo are often subclinical, though small mouth and/or foot lesions have been reported. In contrast severe outbreaks have been seen in free-living mountain gazelles (*Gazella gazella*), impala and saiga antelope (*Saiga tatarica*), and in some captive wildlife (see Weaver et al., 2013 for a detailed review). Similarly to livestock, young animals can die suddenly of myocarditis, Myocarditis and/or heart failure has also been noted in some severely affected adult wildlife.

Clinical cases in species other than ungulates seem to be rare. Captive bears developed vesicles on the footpads, as well as nasal and oral lesions. During one outbreak at a rehabilitation facility, some other bears developed nonspecific signs alone and one of these animals died, though whether FMD caused any of these cases was not investigated. Weakness and lethargy were reported in fatal illnesses in 5 FMDV-infected puppies during outbreaks in Iran. At least some of the puppies had cardiac lesions thought to have caused or contributed to the deaths. Historical cases of possible FMD in dogs, as well as old reports of experimental inoculation, included reports of cardiac abnormalities and/ or vesicles, including small vesicles at the inoculation site in some experimentally infected dogs. Some experimentally infected dogs died.

Post Mortem Lesions

Click to view images

Typical FMD lesions are transient, single or multiple fluid-filled vesicles or bullae, which may initially present as small pale areas or vesicles and become red, eroded areas or ulcers once they rupture. Erosions may be covered with a gray fibrinous coating, and a demarcation line of newly developing epithelium may be noted. Loss of vesicular fluid through the epidermis can lead to the development of “dry” lesions, which appear necrotic rather than vesicular. In domestic animals, dry lesions are particularly common in the oral cavity of pigs. The location and prominence of FMD lesions can differ with the species (see ‘Clinical Signs’); however, possible sites of lesions include the oral cavity and snout/ muzzle; the heel, coronary band and feet; the teats or udder; pressure points on the legs; the ruminal pillars (in ruminants and cervids); and the prepuce or vulva. Coronitis may be seen on the hooves, and the hooves or claws may be sloughed in severe cases.

Myocarditis has been reported mainly in some young livestock and certain wildlife. Some of these animals may have few or no gross cardiac lesions, particularly if they die quickly. In others, cardiac degeneration and necrosis can cause irregular gray or yellow areas, which may include streaking, in the myocardium. The latter lesions are sometimes called “tiger heart” lesions. Petechiae and ecchymoses may also be noted on the heart and pericardium, and there may be fluid in the pericardium and thoracic and abdominal cavities. Microscopic evidence of pancreatitis, with or without gross lesions, has been documented in several species, including mountain gazelles and other antelope, cervids and even some cattle. There are also occasional reports of other internal lesions, such as abomasitis and/or enteritis with congestion and/or edema of the mucosa, signs of septicemia or evidence of secondary infections such as bronchopneumonia, and myositis of the skeletal muscles in young animals.

Infected fetuses from experimentally infected sheep had nonspecific gross lesions including petechial hemorrhages in the skin, subcutaneous edema, ascites with blood-tinged peritoneal fluid and epicardial petechiae. Vesicles were not found, and the placenta did not appear to be affected. Some infected fetuses had no gross lesions. In one study, infected fetuses were generally autolyzed.

Diagnostic Tests

In clinical cases, FMDV, its antigens and nucleic acids can be found in the fluid and epithelial tissue of vesicles, as well as nasal and oral secretions, esophageal-pharyngeal fluid, blood and milk, and in some internal organs such as the heart. In some experiments, live virus could also be recovered from other internal organs for a few days to about a week, and nucleic acids may persist longer. Guidelines from the World Organization for Animal Health (WOAH, formerly OIE) recommend collecting epithelium from unruptured or freshly ruptured vesicles, vesicle fluid, or in cases with no vesicles, blood (serum) and esophageal–pharyngeal fluid samples, taken by probang cup from ruminants or as throat swabs from pigs. Carrier animals are identified by collecting esophageal-pharyngeal fluid with a probang cup; however, one study found that sampling of the palatine tonsils with a nylon brush might be useful for identifying African buffalo carriers. Repeated sampling of suspected carriers may be necessary, as the amount of virus is often low, and fluctuates.

Clinical cases are often diagnosed with reverse transcription polymerase chain reaction (RT-PCR) assays to identify FMDV nucleic acids. Some of these tests can distinguish the viral serotype. Reverse transcription loop-

© 2001-2025

mediated isothermal amplification (RT-LAMP) tests have also been published. Viral antigens are usually detected with ELISAs, but other commercial tests, such as lateral flow assays, may also be available in some countries. Some of the newer lateral flow tests are capable of identifying the viral serotype. Live viruses can be isolated in primary bovine thyroid cells, primary pig, calf or lamb kidney cells, or certain cell lines such as BHK-21 or IBRS-2 cells. Isolated viruses are generally identified as FMDV with ELISAs or RT-PCR, and nucleotide sequence analysis can be used for identifying the viral strain. FMDV can also be recovered from clinical samples in unweaned mice, though animal inoculation is generally discouraged unless there is no good alternative.

Serological tests can be used in surveillance and diagnosis, as well as to certify animals for export, monitor immunity from vaccination, or match the field strain to a vaccine effective against that strain. Test cutoff values differ with the purpose of the test. There are two general types of serological tests for FMD. One group of assays, which includes ELISAs and virus neutralization tests, detects antibodies to the viral structural (e.g., capsid) proteins. These tests are serotype specific; thus, the test chosen must match the serotype(s) of the suspected isolates. They can only be used to diagnose FMD in unvaccinated animals, as vaccination also generates antibodies to these proteins.

The second group of serological tests (e.g., some ELISAs and the enzyme-linked immuno-electrotransfer blot) detects antibodies to the nonstructural proteins (NSPs) of FMDV, which are expressed only during virus replication. These tests are not serotype specific and can be used in both vaccinated and unvaccinated animals. However, they are less sensitive than the assays for structural proteins and may not detect cases with limited virus replication, such as some vaccinated animals that become infected. Due to these limitations, NSP tests are generally employed only as herd tests. Tests to detect IgA in oral fluids are also being investigated as a way to distinguish infected from vaccinated animals, or as a method of identifying carriers, which seem to maintain persistently elevated levels of FMDV-specific IgA.

Treatment

There is no specific treatment for foot and mouth disease, other than supportive care. One recent development is the use of topical anesthetic wound dressings, which appear to be promising. Antiviral agents (e.g., favipiravir derivatives) are under investigation.

Control

Disease reporting

Veterinarians who encounter or suspect foot and mouth disease should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately of any suspected vesicular disease, as it could be FMD.

Prevention

In FMD-free countries, import regulations help prevent this virus from being introduced in infected animals or contaminated foodstuffs fed to animals. Waste food (swill) fed to swine is a particular concern. Heat treatment can kill FMDV in swill; however, some nations have banned feeding swill to pigs due to the risk that this and other viruses may not be completely inactivated, for instance if parts of the swill do not reach the target temperature. Protocols for the inactivation of FMDV in various animal products such as milk products, meat, hides and wool have been published by WOAH.

FMD outbreaks in virus-free regions are usually controlled with standard stamping out measures (quarantines, movement restrictions, euthanasia of affected and exposed animals, and cleaning and disinfection). Good biosecurity measures should be practiced on uninfected farms to prevent entry of the virus. People who have been exposed to FMDV may be asked to avoid contact with susceptible animals for a period of time, in addition to decontaminating clothing and other fomites. In some countries, vaccination may be used as an adjunct measure to help reduce the spread of FMDV or protect specific animals (e.g. those in zoos) during an outbreak. Vaccines are also used in endemic regions to protect animals from illness. FMD vaccines are only effective against the serotype(s) contained in the vaccine. For adequate protection, the vaccine strains must also be well matched with the field strain. Raw tissues from animals that may be infected should not be fed to dogs, cats or other carnivores, as they may become infected or ill.

In southern Africa, transmission from wild African buffalo has been controlled by separating wildlife reserves from domestic livestock with fences, and by vaccinating livestock. However, wildlife fencing may not be practical in some areas, and there are also some disadvantages to its use. Other species of wildlife do not seem to act as reservoirs for FMDV, but they may become infected and carry the virus to uninfected farms during an outbreak. The effects of FMD itself are also a concern in some wildlife species. Vaccination of livestock was reported to decrease outbreaks in some severely affected populations, such as saiga antelope.

Morbidity and Mortality

Morbidity from foot and mouth disease varies with the animal's species, breed and pre-existing immunity, as well as the dose of virus and other factors. The morbidity rate can approach 100% in naive cattle or swine herds, but some FMD viruses can disappear from a sheep flock after infecting a relatively low percentage of the animals. When there is only a single serotype in a region, the virus may cause only mild clinical signs in many cases, with illnesses seen mainly in young animals as they lose their protection from maternal antibodies. Where more than one virus circulates, there may be periodic outbreaks, due to the lack of protection between serotypes and the limited cross-protection between some strains.

© 2001-2025

In livestock, the overall case fatality rate is approximately 1-5% for most FMDV strains, and the mortality rate is often < 1% in adult animals. Reported mortality rates in lambs range from 5% to 94%, and have been reported to reach 80% in some groups of calves and up to 100% in suckling piglets, with lower rates in older piglets. Abortions have been mainly reported in sheep. The average abortion rate in this species was 3-10% during outbreaks in Germany in the 1920s; though there were occasional reports of rates up to 100%. What percentage of FMDV-infected animals become carriers, with or without vaccination, is still uncertain. Estimates vary widely, with experimental and field studies reporting carrier rates that range from less than 5% to more than 50% under different conditions.

Most infections in wildlife are similar to those in domestic animals, but some species or populations can be more severely affected. Approximately 2000 mountain gazelles, representing at least half of the population on one wildlife reserve, died from FMD during an outbreak in Israel. During a second outbreak, an estimated 10-15% of the population was affected, and the case fatality rate was greater than 50%. Likewise, the case fatality rate was as high as 75% in experimentally infected saiga antelope, and some outbreaks in this species resulted in the death of an estimated 10% of the wild population. FMDV does not seem to persist long term in wildlife other than African buffalo.

Reports of foot and mouth disease in carnivores are apparently limited to a few putative cases in dogs described from the late 1800s to early 1900s; experimental infections in dogs and/or cats published between 1920 and 1950; and a recent description of 5 fatal illnesses in puppies, which had been fed infected tissues from lamb carcasses in two separate incidents in Iran. A review from the 1970s described no recent infections in dogs or cats, and noted that, except for acting as mechanical vectors, transmission from these species to livestock appeared to be very rare.

Public Health

Foot and mouth disease is not considered to be a public health problem, as clinical cases seem to be rare and their consequences mild. In the past, many people who worked with FMDV in vaccine laboratories or other sites developed antibodies to this virus; however, one laboratory reported only 2 clinical cases in more than 50 years, while a large FMD vaccine manufacturer documented 3 cases among its workers. Some sources have speculated that only those who are exposed to extremely large amounts of virus or have a predisposing condition develop any symptoms.

A summary of more than 40 laboratory-confirmed cases described between 1921 and 1969 found that these illnesses were usually mild, short-lived and self-limited, and were characterized by vesicular lesions and flu-like symptoms. Broken skin was a recognized route of entry for some cases, with the initial lesions developing at the inoculation site. There is also a report that three veterinarians deliberately infected themselves in 1934, by drinking virus-contaminated, unpasteurized milk for three days. One review from 1970 suggested that infections may have been more common in children than adults. Person-to-person transmission has never been reported; however, vesicles from affected people do contain virus.

[Note: Foot and mouth disease is not related to hand, foot and mouth disease, a condition seen only in humans.]

Internet Resources

FAO Emergency Prevention System for Animal Health (EMPRES). Foot and mouth disease

The European Commission for the Control of Foot-and-Mouth Disease

The Merck Veterinary Manual

U.K. Department for Environment, Food and Rural Affairs. Foot and Mouth Disease.

United States Animal Health Association. Foreign Animal Diseases

U.S. Department of Agriculture (USDA). Foot and Mouth Disease.

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. 2025. *Foot and Mouth Disease*. Retrieved from http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

References

Abdela N. Sero-prevalence, risk factors and distribution of foot and mouth disease in Ethiopia. Acta Trop. 2017;169:125-32.

Acha PN, Szyfres B [Pan American Health Organization (PAHO)]. Zoonoses and communicable diseases common to man and animals. Volume 2. Chlamydioses, rickettsioses, and viroses. 3rd ed. Washington DC: PAHO; 2003. Scientific and Technical Publication No. 580. Foot and mouth disease; p.133-45.

Alexandersen, S., Quan, M., Murphy, C., Knight, J., Zhang, Z. Studies of quantitative parameters of virus excretion and transmission in pigs and cattle experimentally infected with foot-and-mouth disease virus. J Comp Pathol. 2003;129:268-82.

 © 2001-2025

Alexandersen S, Wernery U, Nagy P, Frederiksen T, Normann P. Dromedaries (*Camelus dromedarius*) are of low susceptibility to inoculation with foot-and-mouth disease virus serotype O. J Comp Pathol. 2008;139(4):187-93.

Alexandersen S, Zhang Z, Donaldson AI. Aspects of the persistence of foot and mouth disease virus in animals --the carrier problem. Microbes Infect. 2002;4:1099-110.

Alexandrov T, Stefanov D, Kamenov P, Miteva A, Khomenko S, Sumption K, Meyer-Gerbaulet H, Depner K. Surveillance of foot-and-mouth disease (FMD) in susceptible wildlife and domestic ungulates in southeast of Bulgaria following a FMD case in wild boar. Vet Microbiol. 2013;166(1-2):84-90.

Amass SF, Mason PW, Pacheco JM, Miller CA, Ramirez A, Clark LK, Ragland D, Schneider JL, Kenyon SJ. Procedures for preventing transmission of foot and mouth disease virus (O/TAW/97) by people. Vet Microbiol. 2004;103:143-9.

Amass SF, Pacheco JM, Mason PW, Schneider JL, Alvarez RM, Clark LK, Ragland D. Procedures for preventing the transmission of foot-and-mouth disease virus to pigs and sheep by personnel in contact with infected pigs. Vet Rec. 2003;153(5):137-40.

Anderson EC, Doughty WJ, Anderson J. The role of sheep and goats in the epizootiology of foot-and-mouth disease in Kenya. J Hyg (Lond). 1976;76(3): 395-402.

Arzt J, Baxt B, Grubman MJ, Jackson T, Juleff N, Rhyan J, Rieder E, Waters R, Rodriguez LL. The pathogenesis of foot-and-mouth disease II: viral pathways in swine, small ruminants, and wildlife; myotropism, chronic syndromes, and molecular virus-host interactions. Transbound Emerg Dis. 2011;58(4):305-26.

Arzt J, Belsham GJ, Lohse L, Bøtner A, Stenfeldt C. Transmission of foot-and-mouth disease from persistently infected carrier cattle to naive cattle via transfer of oropharyngeal fluid. mSphere. 2018;3(5):e00365-18.

Ayebazibwe C, Mwiine FN, Tjørnehøj K, Balinda SN, Muwanika VB, Ademun Okurut AR, Belsham GJ, Normann P, Siegismund HR, Alexandersen S. The role of African buffalos (*Syncerus caffer*) in the maintenance of foot-and-mouth disease in Uganda. BMC Vet Res. 2010;6:54.

Barnett P, Garland AJ, Kitching RP, Schermbrucker CG. Aspects of emergency vaccination against foot-and-mouth disease. Comp Immunol Microbiol Infect Dis. 2002;25(5-6):345-64.

Barros JJ, Malirat V, Rebello MA, Costa EV, Bergmann IE. Genetic variation of foot-and-mouth disease virus isolates recovered from persistently infected water buffalo (*Bubalus bubalis*). Vet Microbiol. 2007;120(1-2):50-62.

Bartley LM, Donnelly CA, Anderson RM. Review of foot and mouth disease virus survival in animal excretions and on fomites. Vet Rec. 2002;151:667-9.

Bauer K. Foot- and-mouth disease as zoonosis. Arch Virol Suppl. 1997;13:95-7.

Berkowitz A, Waner T, King R, Yadin H, Perle S. Description of the pathology of a gazelle that died during a major outbreak of foot-and-mouth disease in Israel. J S Afr Vet Assoc. 2010;81(1):62-4.

Bertram MR, Vu LT, Pauszek SJ, Brito BP, Hartwig EJ, et al. Lack of transmission of foot-and-mouth disease virus from persistently infected cattle to naïve cattle under field conditions in Vietnam. Front Vet Sci. 2018;5:174.

Bessler AL, Nayee S, Garabed R, Krug P, Obrycki J, Rodriguez L. Surviving the summer: foot-and-mouth disease virus survival in U.S. regional soil types at high ambient temperatures. Front Vet Sci. 2024;11:1429760.

Bhattacharya S, Banerjee R, Ghosh R, Biswas A, Chatterjee A. Identification of foot and mouth disease from a captive kangaroo in a zoological garden in India. Vet Rec. 2003;153:504-5.

Biswal JK, Nardo AD, Taylor G, Paton DJ, Parida S. Development and validation of a mucosal antibody (IgA) test to identify persistent infection with foot-and-mouth disease virus. Viruses. 2021;13(5):814.

Body MHH, Al-Senaidi NYA, Al-Subhi AHA, Al-Maawali MG, Ahmed MS, Hussain MH. Foot and mouth disease virus serological study of dromedary camels in Oman. Rev Sci Tech. 2019;38(3):731-6.

Borah B, Deka P, Sharma K, Baro S, Hazarika AK, Das C, Garam GB, Boro P, Ltu K. Isolation, identification and retrospective study of foot-and-mouth disease virus from affected mithun (*Bos frontalis*) in north-eastern India. Transbound Emerg Dis. 2018;65(1):e63-9.

Breithaupt A, Depner K, Haas B, Alexandrov T, Polihronova L, Georgiev G, Meyer-Gerbaulet H, Beer M. Experimental infection of wild boar and domestic pigs with a foot and mouth disease virus strain detected in the southeast of Bulgaria in December of 2010. Vet Microbiol. 2012;159(1-2):33-9.

Bronsvoort BMD, Handel IG, Nfon CK, Sørensen KJ, Malirat V, Bergmann I, Tanya VN, Morgan KL. Redefining the "carrier" state for foot-and-mouth disease from the dynamics of virus persistence in endemically affected cattle populations. Sci Rep. 2016;6:29059.

Bronsvoort BM, Parida S, Handel I, McFarland S, Fleming L, Hamblin P, Kock R. Serological survey for foot-and-mouth disease virus in wildlife in eastern Africa and estimation of test parameters of a nonstructural protein enzyme-linked immunosorbent assay for buffalo. Clin Vaccine Immunol. 2008;15(6):1003-11.

Brown E, Nelson N, Gubbins S, Colenutt C. Airborne transmission of foot-and-mouth disease virus: A review of past and present perspectives. Viruses. 2022;14(5):1009.

Buckle K, Bueno R, McFadden A, van Andel M, Spence R, et al. Detection of foot-and-mouth disease virus in the absence of clinical disease in cattle and buffalo in South East Asia. Front Vet Sci. 2021;8:691308.

Burrows R. The persistence of foot-and mouth disease virus in sheep. J Hyg (Lond). 1968;66(4):633-40.

Capel-Edwards M. Foot-and-mouth disease in the brown rat. J Comp Pathol 1970;80:543-8.

Chang H, Ma Y, Lin T, Cong G, Du J, Ma J. Foot-and-mouth disease virus carrier status in *Bos grunniens* yaks. Virol J. 2013;10:81.

Clavijo A, Wright P, Kitching P. Developments in diagnostic techniques for differentiating infection from vaccination in foot-and-mouth disease.Vet J. 2004;167(1):9-22.

Colenutt C, Brown E, Nelson N, Paton DJ, Eblé P, Dekker A, Gonzales JL, Gubbins S. Quantifying the transmission of foot-and-mouth disease virus in cattle via a contaminated environment. mBio. 2020;11(4):e00381-20.

 © 2001-2025

Colenutt C, Gonzales JL, Paton DJ, Gloster J, Nelson N, Sanders C. Aerosol transmission of foot-and-mouth disease virus Asia-1 under experimental conditions. Vet Microbiol. 2016;189:39-45.

Cortey M, Ferretti L, Pérez-Martín E, Zhang F, de Klerk-Lorist LM, Scott K, Freimanis G, Seago J, Ribeca P, van Schalkwyk L, Juleff ND, Maree FF, Charleston B. Persistent infection of African buffalo (*Syncerus caffer*) with foot-and-mouth disease virus: limited viral evolution and no evidence of antibody neutralization escape. J Virol. 2019;93(15):e00563-19.

Cottral GE. Persistence of foot-and-mouth disease virus in animals, their products and the environment. Bull Off Int Epizoot. 1969;71:549-68.

Cox SJ, Barnett PV. Experimental evaluation of foot-and-mouth disease vaccines for emergency use in ruminants and pigs: a review.Vet Res. 2009;40(3):13.

Dhollander S, Belsham GJ, Lange M, Willgert K, Alexandrov T, Chondrokouki E, Depner K, Khomenko S, Özyörük F, Salman M, Thulke HH, Bøtner A. Assessing the potential spread and maintenance of foot-and-mouth disease virus infection in wild ungulates: general principles and application to a specific scenario in Thrace. Transbound Emerg Dis. 2016;63(2):165-74.

Doel TR. FMD vaccines. Virus Res. 2003;91:81-99.

Donaldson A. The role of sheep in the epidemiology of foot-and-mouth disease and proposals for control and eradication in animal populations with a high density of sheep. In: European Commission for the Control of Foot and Mouth Disease of the Food and Agriculture Organization of the United Nations. Bulgaria: Borovets; 2000. p. 107-16.

Donaldson AI, Alexandersen S. Predicting the spread of foot and mouth disease by airborne virus. Rev Sci Tech. 2002;21(3):569-75.

Elnekave E, King R, van Maanen K, Shilo H, Gelman B, Storm N, Klement E. Seroprevalence of foot-and-mouth disease in susceptible wildlife in Israel. Front Vet Sci. 2016;3:32.

European Food Safety Authority (EFSA) Panel on Animal Health and Welfare (AHAW). Scientific opinion on foot-and-mouth disease in Thrace. EFSA J. 2012;10:2635.

Foglia EA, Mioulet V, Cavalera S, Baguisi J, Turgut Sİ, et al. Validation of two multiplex lateral flow devices for the rapid detection and typing of foot-and-mouth disease viruses. Res Vet Sci. 2025;185:105558.

Gloster J, Champion HJ, Mansley LM, Romero P, Brough T, Ramirez A. The 2001 epidemic of foot-and-mouth disease in the United Kingdom: epidemiological and meteorological case studies. Vet Rec. 2005;156(25):793-803.

Grubman MJ, Baxt B. Foot and mouth disease. Clin Microbiol Rev. 2004;17:465-93.

Gubbins S, Forster J, Clive S, Schley D, Zuber S, Schaaff J, Corley D. Thermal inactivation of foot and mouth disease virus in extruded pet food. Rev Sci Tech. 2016;35(3):965-72.

Gulbahar MY, Davis WC, Guvenc T, Yarim M, Parlak U, Kabak YB. Myocarditis associated with foot-and-mouth disease virus type O in lambs.Vet Pathol. 2007;44(5):589-99.

Gurhan SI, Gurhan B, Osturkmen A, Aynagoz G, Candas A, Kizil S. Establishment of the prevalence of persistently infected cattle and sheep in Anatolia with FMDV. Etlik Veteriner Mikrobioyologii Dergisi 1993;7:52-9.

Hyslop NS. The epizootiology and epidemiology of foot and mouth disease. Adv Vet Sci Comp Med. 1970;14:261-307.

Jamal SM, Belsham GJ. Foot-and-mouth disease: past, present and future. Vet Res. 2013;44:116.

Juleff N, Windsor M, Reid E, Seago J, Zhang Z, Monaghan P, Morrison IW, Charleston B. Foot-and-mouth disease virus persists in the light zone of germinal centres. PLoS One. 2008;3:e3434.

Karniely S, Hamed F, Gelman B, King R, Storm N, Eyngor E, Even Tov B. First isolation of foot and mouth disease virus from wild boars in the Middle East. Transbound Emerg Dis. 2020;67(4):1725-9.

Kitching RP. Clinical variation in foot and mouth disease: cattle. Rev Sci Tech. 2002;21:499-504.

Kitching RP. Global epidemiology and prospects for control of foot-and-mouth disease. Curr Top Microbiol Immunol. 2005;288:133-48.

Kitching RP. Identification of foot and mouth disease virus carrier and subclinically infected animals and differentiation from vaccinated animals. Rev Sci Tech. 2002;21(3):531-8.

Kitching RP, Alexandersen S. Clinical variation in foot and mouth disease: pigs. Rev Sci Tech. 2002;21:513-8.

Kitching RP, Hughes GJ. Clinical variation in foot and mouth disease: sheep and goats. Rev Sci Tech. 2002;21:505-12.

Kitching RP, Thrusfield MV, Taylor NM. Use and abuse of mathematical models: an illustration from the 2001 foot and mouth disease epidemic in the United Kingdom. Rev Sci Tech. 2006;25(1):293-311.

Kittelberger R, Nfon C, Swekla K, Zhang Z, Hole K, et al. Foot-and-mouth disease in red deer - experimental infection and test methods performance. Transbound Emerg Dis. 2017;64(1):213-25.

Knight-Jones TJ, Robinson L, Charleston B, Rodriguez LL, Gay CG, Sumption KJ, Vosloo W. Global foot-and-mouth disease research update and gap analysis: 4 - diagnostics. Transbound Emerg Dis. 2016;63 Suppl 1:42-8.

Krug PW, Larson CR, Eslami AC, Rodriguez LL. Disinfection of foot-and-mouth disease and African swine fever viruses with citric acid and sodium hypochlorite on birch wood carriers. Vet Microbiol. 2012;156(1-2):96-101.

Krug PW, Lee LJ, Eslami AC, Larson CR, Rodriguez L. Chemical disinfection of high-consequence transboundary animal disease viruses on nonporous surfaces. Biologicals. 2011;39(4):231-5.

Larska M, Wernery U, Kinne J, Schuster R, Alexandersen G, Alexandersen S. Differences in the susceptibility of dromedary and Bactrian camels to foot-and-mouth disease virus. Epidemiol Infect. 2009;137(4):549-54.

Lendzele SS, Mavoungou JF, Burinyuy KA, Armel KA, Dickmu SJ, Young JR, Thomson PC, Windsor PA. Efficacy and application of a novel topical anaesthetic wound formulation for treating cattle with foot-and-mouth disease: A field trial in Cameroon. Transbound Emerg Dis. 2021;68(4):2531-42.

Letshwenyo M, Mapitse N, Hyera JM. Foot and mouth disease in a kudu (*Tragelaphus strepsiceros*) in Botswana. Vet Rec. 2006;159:252-3.

Li F, Li Y, Ma J, Wu R, Zou X, Liu Y, Zhao Q, Zhu Y. Molecular evolution, diversity, and adaptation of foot-and-mouth disease virus serotype O in Asia. Front Microbiol. 2023;14:1147652.

 © 2001-2025

Lignereux L, Chaber AL, Saegerman C, Heath L, Knowles NJ, Wadsworth J, Mioulet V, King DP. Foot-and-mouth disease outbreaks in captive scimitar-horned oryx (*Oryx dammah*). Transbound Emerg Dis. 2020;67(4):1716-24.

Longjam N, Deb R, Sarmah AK, Tayo T, Awachat VB, Saxena VK. A brief review on diagnosis of foot-and-mouth disease of livestock: conventional to molecular tools. Vet Med Int. 2011;2011:905768.

Ludi AB, Baker H, Sanki R, De Jong RMF, Maryan J, Walker M, King DP, Gubbins S, Limon G, Officer K. Epidemiological investigation of foot-and-mouth disease outbreaks in a Vietnamese bear rescue centre. Front Vet Sci. 2024;11:1389029.

Maddur MS, Kishore S, Gopalakrishna S, Singh N, Suryanarayana VV, Gajendragad MR. Immune response and viral persistence in Indian buffaloes (*Bubalus bubalis*) infected with foot-and-mouth disease virus serotype Asia 1. Clin Vaccine Immunol. 2009;16(12):1832-6.

Maree F, de Klerk-Lorist LM, Gubbins S, Zhang F, Seago J, Pérez-Martín E, Reid L, Scott K, van Schalkwyk L, Bengis R, Charleston B, Juleff N. Differential persistence of foot-and-mouth disease virus in African buffalo is related to virus virulence. J Virol. 2016;90(10):5132-40.

Maroudam V, Nagendrakumar SB, Madhanmohan M, Santhakumar P, Thiagarajan D, Srinivasan VA. Experimental transmission of foot-and-mouth disease among Indian buffalo (*Bubalus bubalis*) and from buffalo to cattle. Comp Pathol. 2008;139(2-3):81-5.

Mebus CA, Singh EL. Embryo transfer as a means of controlling the transmission of viral infections. XIII. Failure to transmit foot-and-mouth disease virus through the transfer of embryos from viremic donors. Theriogenology. 1991;35(2):435-41.

Metwally S, Bkear N, Badr Y, Elshafey B, Alhag SK, Al-Shuraym LA, Batiha G, Fakhry B, Hamada R. A newly emerging serotype A strain in foot-and-mouth disease virus with higher severity and mortality in buffalo than in cattle calves in North Egypt. Vet Sci. 2023;10(8):488.

Mezencio JMS, Babcock GD, Kramer E, Brown F. Evidence for the persistence of foot-and-mouth disease virus in pigs. Vet J. 1999;157:213-7.

Mielke SR, Garabed R. Environmental persistence of foot-and-mouth disease virus applied to endemic regions. Transbound Emerg Dis. 2020;67(2):543-54.

Mohamed F, Swafford S, Petrowski H, Bracht A, Schmit B, et al. Foot-and-mouth disease in feral swine: susceptibility and transmission. Transbound Emerg Dis. 2011;58(4):358-71.

Mohebbi MR, Barani SM, Mahravani H. An uncommon clinical form of foot-and-mouth disease in beef cattle presented with cornual skin lesions. Iran J Vet Res. 2017l;18(4):291-3.

Morris RS, Sanson RL, Stern MW, Stevenson M, Wilesmith JW. Decision-support tools for foot and mouth disease control. Rev Sci Tech. 2002;21(3):557-67.

Musser JM. A practitioner's primer on foot and mouth disease. J Am Vet Med Assoc. 2004;224:1261-8.

Nishi T, Fukai K, Masujin K, Kawaguchi R, Ikezawa M, Yamada M, Nakajima N, Komeno T, Furuta Y, Sugihara H, Kurosaki C, Sakamoto K, Morioka K. Administration of the antiviral agent T-1105 fully protects pigs from foot-and-mouth disease infection. Antiviral Res. 2022;208:105425.

Officer K, Lan NT, Wicker L, Hoa NT, Weegenaar A, Robinson J, Ryoji Y, Loukopoulos P. Foot-and-mouth disease in Asiatic black bears (*Ursus thibetanus*). Vet Diagn Invest. 2014;26(5):705-13.

Onodera T, Sakudo A, Sugiura K, Haritani M, Furusaki K, Kirisawa R. Antiviral agents and disinfectants for foot‐and‐mouth disease (review). Biomed Rep. 2023;19(3):57.

Orsel K, Roest HI, Elzinga-Bril EM, van Hemert-Kluitenberg F, Dekker A. Detection of foot-and-mouth disease virus in infected pigs by RT-PCR four weeks after challenge. Vet Rec. 2008;162(23):753-4.

Parida S, Fleming L, Oh Y, Mahapatra M, Hamblin P, Gloster J, Doel C, Gubbins S, Paton DJ. Reduction of foot-and-mouth disease (FMD) virus load in nasal excretions, saliva and exhaled air of vaccinated pigs following direct contact challenge. Vaccine. 2007;25(45):7806-17.

Parthiban AB, Mahapatra M, Gubbins S, Parida S. Virus excretion from foot-and-mouth disease virus carrier cattle and their potential role in causing new outbreaks. PLoS One. 2015;10(6):e0128815.

Paton DJ, Di Nardo A, Knowles NJ, Wadsworth J, Pituco EM, et al. The history of foot-and-mouth disease virus serotype C: the first known extinct serotype? Virus Evol. 2021;7(1):veab009.

Poonsuk K, Giménez-Lirola L, Zimmerman JJ. A review of foot-and-mouth disease virus (FMDV) testing in livestock with an emphasis on the use of alternative diagnostic specimens. Anim Health Res Rev. 2018;19(2):100-12.

Prempeh H, Smith R, Müller B. Foot and mouth disease: the human consequences. The health consequences are slight, the economic ones huge. BMJ. 2001;322(7286):565-6.

Ranjan R, Biswal JK, Subramaniam S, Dash BB, Singh KP, Arzt J, Rodriguez LL, Pattnaik B. Evidence of subclinical foot-and-mouth disease virus infection in young calves born from clinically recovered cow under natural condition. Trop Anim Health Prod. 2018;50(5):1167-70.

Ranjan R, Biswal JK, Subramaniam S, Singh KP, Stenfeldt C, Rodriguez LL, Pattnaik B, Arzt J. Foot-and-mouth disease virus-associated abortion and vertical transmission following acute infection in cattle under natural conditions. PLoS One. 2016;l11:e0167163.

Rhyan J, Deng M, Wang H, Ward G, Gidlewski T, McCollum M, Metwally S, McKenna T, Wainwright S, Ramirez A, Mebus C, Salman M. Foot-and-mouth disease in North American bison (*Bison bison*) and elk (*Cervus elaphus nelsoni)*: susceptibility, intra- and interspecies transmission, clinical signs, and lesions. J Wildl Dis. 2008;44(2):269-79.

Rhyan J, McCollum M, Gidlewski T, Shalev M, Ward G, Donahue B, Arzt J, Stenfeldt C, Mohamed F, Nol P, Deng M, Metwally S, McKenna T, Salman M. Foot-and-mouth disease in a small sample of experimentally infected pronghorn (*Antilocapra americana*). J Wildl Dis. 2016;52(4):862-73.

Rhyan J, McCollum M, Gidlewski T, Shalev M, Ward G, Donahue B, Arzt J, Stenfeldt C, Mohamed F, Nol P, Deng M, Metwally S, Salman M. Foot-and-mouth disease in experimentally infected mule deer (*Odocoileus hemionus*). J Wildl Dis. 2020;56(1):93-104.

 © 2001-2025

Rivera AM, Sanchez-Vazquez MJ, Pituco EM, Buzanovsky LP, Martini M, Cosivi O. Advances in the eradication of foot-and-mouth disease in South America: 2011-2020. Front Vet Sci. 2023;9:1024071.

Royal Society. Infectious diseases in livestock: summary and main recommendations (B. Follett, Chair). Policy document 19/02. The Royal Society, London; 2002. 8 p. Available at: http://reports.royalsoc.ac.uk/idl_sum.pdf.* Accessed 2009.

Rweyemamu M, Roeder P, Mackay D, Sumption K, Brownlie J, Leforban Y, Valarcher JF, Knowles NJ, Saraiva V. Epidemiological patterns of foot-and-mouth disease worldwide. Transbound Emerg Dis. 2008;55(1):57-72.

Ryan E, Horsington J, Brownlie J, Zhang Z. Foot-and-mouth disease virus infection in fetal lambs: tissue tropism and cytokine response. J Comp Pathol. 2008;138(2-3):108-20.

Ryan E, Horsington J, Durand S, Brooks H, Alexandersen S, Brownlie J, Zhang Z. Foot-and-mouth disease virus infection in young lambs: pathogenesis and tissue tropism. Vet Microbiol. 2008;127(3-4):258-74.

Ryan E, Zhang Z, Brooks HW, Horsington J, Brownlie J. Foot-and-mouth disease virus crosses the placenta and causes death in fetal lambs.J Comp Pathol. 2007;136(4):256-65.

Salt JS. Persistent infections with foot-and-mouth disease virus. Top Trop Virol. 1998;1:77-128.

Sanchez-Vazquez MJ, Buzanovsky LP, Dos Santos AG, Allende RM, Cosivi O, Rivera AM. Investigating the temporal and spatial distribution of foot-and-mouth disease virus serotype C in the region of South America, 1968-2016. Transbound Emerg Dis. 2019;66(2):653-61.

Sangula AK, Siegismund HR, Belsham GJ, Balinda SN, Masembe C, Muwanika VB. Low diversity of foot-and-mouth disease serotype C virus in Kenya: evidence for probable vaccine strain re-introductions in the field. Epidemiol Infect. 2011;139(2):189-96.

Schaftenaar W. Use of vaccination against foot and mouth disease in zoo animals, endangered species and exceptionally valuable animals. Rev Sci Tech. 2002;21(3):613-23.

Sellers RF, Donaldson AI, Herniman KA. Inhalation, persistence and dispersal of foot-and-mouth disease virus by man. J Hyg (Lond). 1970;68(4):565-73.

Stenfeldt C, Arzt J. The carrier conundrum; A review of recent advances and persistent gaps regarding the carrier state of foot-and-mouth disease virus. Pathogens. 2020;9(3):167.

Stenfeldt C, Bertram MR, Smoliga GR, Hartwig EJ, Delgado AH, Arzt J. Duration of contagion of foot-and-mouth disease virus in infected live pigs and carcasses. Front Vet Sci. 2020;7:334.

Stenfeldt C, Diaz-San Segundo F, de Los Santos T, Rodriguez LL, Arzt J. The pathogenesis of foot-and-mouth disease in pigs. Front Vet Sci. 2016;3:41.

Stenfeldt C, Eschbaumer M, Rekant SI, Pacheco JM, Smoliga GR, Hartwig EJ, Rodriguez LL, Arzt J. The foot-and-mouth disease carrier state divergence in cattle. J Virol. 2016;90(14):6344-64.

Stenfeldt C, Lohse L, Belsham GJ. The comparative utility of oral swabs and probang samples for detection of foot-and-mouth disease virus infection in cattle and pigs. Vet Microbiol. 2013;162(2-4):330-7.

Stenfeldt C, Pacheco JM, Singanallur NB, Vosloo W, Rodriguez LL, Arzt J. Virulence beneath the fleece; a tale of foot-and-mouth disease virus pathogenesis in sheep. PLoS One. 2019;14(12):e0227061.

Suchowski M, Eschbaumer M, Teifke JP, Ulrich R. After nasopharyngeal infection, foot-and-mouth disease virus serotype A RNA is shed in bovine milk without associated mastitis. J Vet Diagn Invest. 2021;33(5):997-1001.

Sutmoller P, Casas OR. Unapparent foot and mouth disease infection (sub-clinical infections and carriers): implications for control. Rev Sci Tech. 2002;21(3):519-29.

Tekleghiorghis T, Moormann RJ, Weerdmeester K, Dekker A. Serological evidence indicates that foot-and-mouth disease virus serotype O, C and SAT1 are most dominant in Eritrea. Transbound Emerg Dis. 2014;61(6):e83-8.

Thomson GR, Vosloo W, Bastos AD. Foot and mouth disease in wildlife. Virus Res. 2003;91:145-61.

Torres A. Foot-and-mouth disease. In: Foreign animal diseases. Bota Raton, FL: United States Animal Health Association; 2008. p. 261-75.

United Kingdom. Department for Environment, Food and Rural Affairs (DEFRA). FMD: Commonly asked questions. DEFRA; 2007 Aug. Available at: http://www.defra.gov.uk/animalh/diseases/fmd/qanda/qanda-general.htm. *Accessed 5 Sept 2007.

United Kingdom. Department for Environment, Food and Rural Affairs (DEFRA). FMD disease emergency vaccination - question and answer brief. DEFRA:2007 Aug. Available at: http://www.defra.gov.uk/animalh/diseases/fmd/policy/vaccinationqanda.htm.* Accessed 5 Sept. 2007.

Verin B, Edwards J, Babu1 A, Di Nardo A, Grazioli S, Brocchi E, Paton D, Benigno C, Sumption K, Parida S. Detection of FMDV in carrier buffalo in South East Asia. In: Open session of the Standing Technical Committee of the European Commission for the Control of FMD. 2010 Sept 27 – Oct 1; Vienna, Austria. Available at: ftp://ext-ftp.fao.org/AG/Data/agah/EuFMD/Open%20Session%20ppts/Presentations.PDF/.* Accessed 26 Oct 2010.

Ward MP, Laffan SW, Highfield LD. Disease spread models in wild and feral animal populations: application of artificial life models. Rev Sci Tech. 2011;30(2):437-46.

Waters RA, Wadsworth J, Mioulet V, Shaw AE, Knowles NJ, Abdollahi D, Hassanzadeh R, Sumption K, King DP. Foot-and-mouth disease virus infection in the domestic dog (*Canis lupus familiaris*), Iran. BMC Vet Res. 2021;17(1):63.

Weaver GV, Domenech J, Thiermann AR, Karesh WB. Foot and mouth disease: a look from the wild side. J Wildl Dis. 2013;49(4):759-85.

Wernery U, Kinne J. Foot and mouth disease and similar virus infections in camelids: a review. Rev Sci Tech. 2012;31(3):907-18.

Wernery U, Nagy P, Amaral-Doel CM, Zhang Z, Alexandersen S. Lack of susceptibility of the dromedary camel (*Camelus dromedarius*) to foot-and-mouth disease virus serotype O. Vet Rec. 2006;158(6):201-3.

Wright CF, Gloster J, Mazelet L, Paton DJ, Ryan ED. Short-lived carriage of foot-and-mouth disease virus in human nasal cavities after exposure to infected animals. Vet Rec. 2010;167(24):928-31.

 © 2001-2025

World Organization for Animal Health [WOAH] . Manual of diagnostic tests and vaccines for terrestrial animals [online]. Paris: WOAH; 2022. Foot and mouth disease. Available at: https://www.woah.org/en/what-we-do/standards/codes-and-manuals/. Accessed 3 Jul 2025:

World Organization for Animal Health [OIE] . Terrestrial animal health code [online]. Paris: OIE; 2012. Foot and mouth disease. Available at: http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.8.6.htm.* Accessed 7 Apr 2014.

Yadin H, Chai D. Surveillance of FMD in wild animals in Israel. In: Research Group of the Standing Technical Committee, European Commission for the Control of Foot and Mouth Disease. 1994 Sept. 19-22; Vienna, Austria. Rome: FAO; 1995.

Yang M, Mudabuka B, Dueck C, Xu W, Masisi K, Fana EM, Mpofu C, Nfon C. Development of two rapid lateral flow test strips for detection of foot-and-mouth disease virus SAT 1 and SAT 3. J Virol Methods. 2021;291:113967.

Zhang Z, Bashiruddin JB. Quantitative analysis of foot-and-mouth disease virus RNA duration in tissues of experimentally infected pigs.Vet J. 2009;180(1):130-2.

Zhang Z, Waters R, Li Y. Pathogenesis of non-epithelial foot-and-mouth disease in neonatal animals. Vet Microbiol. 2021;254:108961.

* Link defunct

© 2001-2025