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# FOOT AND MOUTH DISEASE: A LOOK FROM THE WILD SIDE

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ABSTRACT: We review the literature and discuss control options regarding foot and mouth disease (FMD) in wildlife around the world. There are more than 100 species of wild, feral, laboratory, or domesticated animals that have been infected naturally or experimentally with FMD virus. Apart from the African buffalo (*Syncerus caffer*) in sub-Saharan Africa, wildlife has not been demonstrated to play a significant role in the maintenance of FMD. More often, wildlife are passively infected when outbreaks of FMD occur in domestic livestock, and, in some wild ungulates, infection results in severe disease. Efforts to control FMD in wildlife may not be successful when the disease is endemic in livestock and may cause more harm to wildlife, human livelihoods, and domestic animals. Currently in sub-Saharan Africa, the complete eradication of FMD on a subcontinental scale in the near term is not possible, given the presence of FMD-infected African buffalo and the existence of weak veterinary infrastructures in some FMD-endemic countries. Therefore efforts to control the disease should be aimed at improved vaccines and improved use of vaccines, improved livestock management practices, and utilization of programs that can help in disease control such as the FMD Progressive Control Program and regulatory frameworks that facilitate trade such zonation, compartmentalization, and commodity-based trade. Though not meeting the definition of wildlife used in this review, feral domestic animals warrant a special concern with regard to FMD control.

*Key words:* Experimental infections, FMD, FMD control, foot and mouth disease, natural infections, wildlife.

## INTRODUCTION

Despite numerous review articles on foot and mouth disease (FMD) in wildlife (Schaftenaar, 2002; Thomson et al., 2003; Pinto, 2004; Arzt et al., 2011a), important, science-backed information is lacking (Roeder, 2009). A key to understanding the epidemiologic role of wildlife in the maintenance of FMD infections at population levels is the understanding of the cycles of infection or persistence of infection for the species that are susceptible. Unfortunately, beyond the valuable experimental infection work performed in some wildlife species, much of the literature on the subject fails to distinguish between evidence of infection and the ability to effectively maintain infections at population levels that could result in persistence or frequent transmission to other species. Thomson et al. (2003) and Roeder (2009) have discussed this issue. As a result, erroneous statements and conclusions regarding the role of wildlife in the maintenance or transmission of FMD are widespread. Adding to confusion regarding FMD in wildlife is the lack of consistency in terminology used in many publications to indicate wildlife, wild animals, feral animals, zoo animals, species, etc. Herein, we use the World Organization of Animal Health (OIE) definitions for wild and feral animals:

- Wild animals are those animals that do not live under human supervision or control and do not have their phenotype selected by humans.
- Captive wild animals are those animals that live under human supervision or control but their phenotype is not selected by humans.
- Feral animals are those animals that do not live under human supervision or control but their phenotype is (or has been) selected by humans

We provide details on evidence of infection based on available published literature (Table 1) and discuss opportunities and challenges for control and management of FMD. Although Table 1 lists more than 100 species from the available literature on FMD, this does not imply that all of these species can become infected, effectively transmit the virus to other species, or play a role in the epidemiology of FMD. For example, intradermal or intradermolingual inoculation was used in many experimental infections. Many species including birds and fish can be become infected via this method, but that does not imply that they would become infected if exposed via aerosols or that they could transmit the virus. Although experimental infections can yield important information, there are limitations in many of these experiments, and results may not be relevant to the course of natural disease. In addition, there were several early reports of natural infections that were diagnosed based on clinical signs alone and were never laboratory confirmed. Because clinical signs of FMD can look like other diseases, it is possible that these cases were incorrectly diagnosed.

## CLINICAL DISEASE AND TRANSMISSION

The clinical presentation of FMD in wildlife has been reviewed (Thomson et al., 2003; Arzt et al., 2011a). In general, the symptoms in wildlife are similar to those in domestic animals, although the pathogenesis of FMD virus (FMDV) in many susceptible wildlife species has not been studied extensively. There is clear variation in the susceptibility to FMDV based on the host species and viral serotype involved. Among susceptible species, there may be differences in severity of infection based on the amount of virus at inoculation, the serotype involved, the species affected, and the individual animal's immunity (Alexandersen et al., 2003). There is a wide range of clinical symptoms ranging from subclinical, unapparent infection as is seen typically in African buffalo (*Syncerus caffer*; Vosloo et al., 2007) to acutely fatal infection with extensive pathology to the pancreas, as occurs in mountain gazelles (*Gazella gazella*; Shimshony et al., 1986; Perl et al., 1989). The virus is epitheliotrophic, and typical lesions are vesicles that rupture and leave erosions or ulcerations and result in lameness or difficulty eating (Alexandersen et al., 2003). Lesions often occur in the oral cavity (tongue, dental pad) and coronary bands in bovids and interdigital locations in suids and cervids. Lesions also occur on the snout or on the knees in warthogs (*Phacochoerus africana*; Hedger et al., 1972). Animals generally recover in 7–14 days from the acute infection; however, a carrier state may persist in some species. In addition, a chronic postviremia syndrome has been described that can include secondary skin infections, hoof malformations, decreased milk production, and heat intolerance (Arzt et al., 2011a,b).

Infection occurs generally through aerosolization of virus. Although transmission from abrasions of mucus membranes can occur, this requires 10,000 times more virus to cause an infection. Aerosol spread is frequently implicated in wildlife, but the exact transmission is uncertain as it is difficult to determine the contribution of other potential methods of transmission (e.g., fomites or waterborne; Arzt et al., 2011a). Virus has been isolated in milk, semen, urine, and feces. Replication occurs rapidly, and many species infected experimentally demonstrate virus in the respiratory tract 24 hr after infection and in epithelial cells of lesions after 72 hr. Incubation is 2–14 days depending on the infective dose and route of transmission. In domestic swine, infection usually occurs after being fed FMD-contaminated swill or direct contact with FMD-infected animals or fomites. Swine are less susceptible to spread via aerosols than cattle; however, they excrete the largest amount of aerosolized virus (Alexandersen et al., 2003; Arzt et al., 2011a).

TABLE 1. Species documented with either natural (Nat) or experimental (Exp) infection with various serotypes of foot and mouth disease virus with indication of proven transmission to other animals or species, observation of clinical signs of infection, and test methods used (antibody or virus isolation). Dashes indicate no data available.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Agouti (*Dasyprocta agouti*) | Conspecific | Exp (A, C) | Y | Y | Y | N | Federer, 1969 |
| Alpaca (*Vicugna pacos*) | | Nat (A) | Mild | N | N | N | Mauro and Guererro, 1971, cited in Wernery and Kaaden, 2004 |
| Armadillo, Big Hairy (*Chaetophracius villosus*) | Conspecific, swine | Exp (A, C, O) | Y | Y | Y | — | Campion, 1950 |
| Armadillo, Nine-Banded (*Dasypus novemcinctus*) | Conspecific | Exp (A) | Y | Y | Y | — | Wilder et al., 1974 |
| Babirusa (*Babyrousa babyrussa*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| Bandicoot, Long-nosed (*Perameles nasuta*) | | Exp (SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Bat, Vampire (*Desmodus rotundus*) | | Exp (A, O) | Y | — | Y | — | Lord et al., 1986 |
| Bear, Brown (*Ursus arctos*) | | Nat | Y | — | — | — | Neugebauer, 1976 |
| Bear, Grizzly (*Ursus arctos horribilis*) | | Nat | Y | — | — | — | Grosso, 1957 |
| Bear, Tibetan/Asiatic Black (*Ursus thibetanus*) | | Nat | Y | — | — | — | Neugebauer, 1976 |
| Bison, European (*Bison bonasus*) | | Nat | Y | — | — | — | Folmer, 1957, cited in Schaftenaar, 2002 |
| Bison, North American (*Bison bison*) | Conspecific, cattle | Nat | Y | — | — | — | Urbain et al., 1938 |
| | | Exp (O) | Y | Y | Y | N | Rhyan et al., 2008 |
| Black buck (*Antilope cervicapra*) | | Nat (O) | Severe | — | Y | — | Kar et al., 1983; Hegde et al., 2011 |
| Brown marsupial mouse (*Antechinus stuartii*) | | Exp (A, SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Buffalo, African (*Syncerus caffer*) | Conspecific, cattle, impala, kudu | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| | | Nat (SAT-1) | Mild | N | Y | Y | Hedger et al., 1972 |
| | | Nat (SAT-1) | Y | Y | Y | Y | Vosloo et al., 2007 |
| | | Exp (SAT-2) | N | N | Y | Y | Hedger et al., 1972 |
| | | Exp (SAT-1, 2) | Y | Y | Y | Y | Gainaru et al., 1986; Ferris et al., 1989 |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Buffalo, Water (*Bubalus bubalis*) | Conspecific, cattle | Nat (A) | N | Y | Y | — | Samara and Pinto, 1983 |
| | | Exp (O) | Y | Y | Y | Y | Moussa et al., 1979; Maroudam et al., 2008 |
| | | Exp (Asia-1) | Y | Y | Y | Y | Maddur et al., 2009 |
| Bushbuck (*Tragelaphus scriptus*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Bush pig (*Potamochoerus porcus*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| | | Exp (SAT-2) | Severe | Y | Y | N | Hedger et al., 1972 |
| Camel, Bactrian (*Camelus bactrianus*) | No transmission | Nat (O) | Y | — | N | N | Review in Wernery and Kaaden, 2004 |
| | | Exp (A) | Y | Y | Y | N | Larska et al., 2009 |
| Camel, Dromedary (*Camelus dromedaries*) | Cattle (possibly) | Nat (A, O, SAT-1, 2) | Mild/N | Y | Y | N | Review in Wernery and Kaaden, 2004 |
| | | Nat (O) | — | Y | — | — | Yousef et al., 2012 |
| | | Exp (O) | N | Y | Y | N | Review in Wernery and Kaaden, 2004 |
| | | Exp (A) | N | N | N | N | Larska et al., 2009 |
| Capybara (*Hydrochoerus hydrochaeris*) | Conspecific, cattle | Exp (O) | Y | Y | Y | — | Gomes and Rosenberg, 1984 |
| Cattle (*Bos primigenius*) | Many | Nat/Exp | Y | Y | Y | Y | Alexandersen et al., 2003; Arzt et al., 2011b |
| Caucsasian tur/wild goat (*Capra aegagrus*) | | Nat | Y | — | — | — | Heymann, 1964, cited in Schaftenaar, 2002 |
| Chamois (*Rupicapra rupicapra*) | | Nat | Y | — | — | — | Stroh, 1939 |
| Chinchilla (*Chinchilla lanigera*) | | Exp (A) | Y | Y | Y | — | Dellers, 1963 |
| Coypu/nutria (*Myocaster coypus*) | | Exp (A, C, O) | Y | Y | Y | — | Capel-Edwards, 1967 |
| Deer, Brown brocket (*Mazama gouzoubira*) | | Nat | — | — | — | — | Pinto, 2004 |
| Deer, Columbian (*Odocoileus virginianus leucurus*) | | Nat | Y | — | — | — | Grosso, 1957 |
| Deer, Eld's (*Rucervus eldii*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| Deer, Fallow (*Dama dama*) | Conspecific, Red deer | Nat | Y | — | — | — | Urbain et al., 1938 |
| | | Exp (C, O) | Mild | Y | Y | Y | Forman and Gibbs, 1974; Forman et al., 1974 |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Deer, Marsh (*Blastocerus dichotomus*) | | Nat (A, C, O) | — | Y | — | — | Araujo et al., 2010 |
| | | Exp | — | — | — | — | Pinto, 2004 |
| Deer, Mule (*Odocoileus hermionus*) | | Nat | Severe | — | — | — | Keane 1927 in McVicar et al., 1974 |
| | | Exp (O) | Y | — | — | — | Dunbar et al., 2009 |
| Muntjac/Barking Deer (*Muntiacus muntjak*) | Conspecific, Sika deer, sheep | Nat | Y | Y | N | — | Barman et al., 1999 |
| | | Exp (C) | Severe | Y | Y | N | Gibbs et al., 1975 |
| Deer, Red (*Cervus elaphus*) | Conspecific, Fallow deer | Nat | Y | — | — | — | Thomson et al., 2003 |
| | | Exp (C, O) | Mild | Y | Y | Y | Forman and Gibbs, 1974; Forman et al., 1974 |
| Deer, Red brocket (*Mazama americana*) | | Exp | — | — | — | — | Stroh, 1933, cited in Federer, 1969 |
| Deer, Roe (*Capreolus capreolus*) | Conspecific, Fallow deer, Red deer | Nat | Y | — | — | — | Thomson et al., 2003 |
| | | Nat (O) | — | Y | — | — | EFSA, 2012 |
| | | Exp (C, O) | Severe | Y | Y | N | Forman and Gibbs, 1974; Forman et al., 1974 |
| Deer, Sambar (*Rusa unicolor*) | | Nat (O) | Y | — | Y | — | Kar et al., 1983; Barman et al., 1999 |
| Deer, Sika (*Cervus nippon*) | Conspecific, Muntjac, sheep | Exp (C) | Mild | Y | Y | Y | Gibbs et al., 1975 |
| Deer, Spotted/Axis (*Axis axis*) | | Nat (O) | Y | — | Y | — | Kar et al., 1983 |
| | | Nat (O) | — | Y | — | — | Bhat and Manickam, 1997 |
| Deer, White-tailed (*Odocoileus virginianus*) | Cattle | Exp (O) | Y | — | Y | Y | McVicar et al., 1974 |
| | | Exp (O) | Y | Y | Y | N | Moniwa et al., 2012 |
| Dorcas gazelle (*Gazella dorcas*) | | — | Y | — | — | — | Arzt et al., 2011a |
| Duiker (*Sylvicapra grimmia*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Echidna (*Tachyglossus aculeatus*) | | Exp (A, O, SAT-1) | Mild | Y | Y | — | Snowdon, 1968 |
| Eland (*Taurotragus oryx*) | Conspecific | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| | | Nat (O, SAT-2) | — | Y | — | — | Anderson, 1981 |
| | | Exp (SAT-1) | Mild | Y | Y | N | Ferris et al., 1989 |
| Elephant, African (*Loxodonta africana*) | No transmission | Nat (A) | Y | — | — | — | Piragino, 1970, cited in Schaftenaar, 2002 |
| | | Nat (SAT-1) | N | N | N | N | Hedger et al., 1972 |
| | | Exp (SAT-2) | N | N | N | N | Hedger et al., 1972 |
| | | Exp (SAT-2) | Y | Y | Y | N | Howell et al., 1973 |
| | | Exp (SAT-1) | N | N | N | N | Bengis et al., 1984 |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Elephant, Asian (*Elephas maximus*) | Conspecific | Nat (O) | Y | — | Y | — | Pyakural et al., 1976; Barman et al., 1999 |
| | | Nat (O) | — | Y | — | — | Bhat and Manickam, 1997 |
| | | Nat (A) | — | Y | — | — | Hedge et al., 2010 |
| Elk (*Cervus elaphus nelsonii*) | Conspecific | Nat | Y | — | — | — | Magnusson, 1939, cited in Thomson et al., 2003 |
| | | Exp (O) | Mild | Y | Y | N | Rhyan et al., 2008 |
| Gaur/Indian Bison (*Bos gaurus*) | | Nat (Asia-1) | Severe | Y | Y | — | Verma and Sarma, 1997 |
| | | Nat (A, O) | Y | — | Y | — | Barman et al., 1999 |
| Gemsbok (*Oryx oryx gazelle*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Gerbil (unspecified) | | Exp | Y | — | — | — | Capel-Edwards, 1971b |
| Giraffe (*Giraffa camelopardalis*) | No transmission | Nat | Y | — | — | — | Bengis, 1984, cited in Thomson et al., 2003 |
| | | Exp (SAT-1, 2) | Y | Y | Y | N | Vosloo et al., 2011 |
| Goat (*Capra aegagrus hircus*) | Many | Nat/Exp | Y | Y | Y | Y | Alexandersen et al., 2003; Arzt et al., 2011a |
| Grant's gazelle (*Gazella granti*) | | Nat (A, O) | — | Y | — | — | Anderson, 1981 |
| Grysbuck (*Raphicerus sharpei*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Guanaco (*Lama guanicoe*) | | Exp | — | — | — | — | Pinto, 2004 |
| Guinea pig (*Cavia porcellus*) | | Exp | Y | Y | Y | — | Capel-Edwards, 1971a |
| Hamster, Syrian/Golden (*Mesocricetus auratus*) | | Exp | Y | — | Y | — | Capel-Edwards, 1971a |
| Hartebeest (*Alcelaphus buselaphus*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Ayebazibwe et al., 2010a |
| Hedgehog, East African (*Atelerix prurei hindu*) | | Exp | Y | Y | Y | — | Macaulay, 1963 |
| Hedgehog, European (*Erinaceus europaeus*) | Guinea pig, cattle | Nat (O) | Y | N | Y | — | McLauchlan and Henderson, 1947 |
| | | Exp (A) | Y | | Y | — | Wolf, 1939 |
| Human (*Homo sapiens*) | | Nat (A, C, O) | Y | Y | Y | — | Niedbalski et al., 2006 |
| Hyrax, Eastern tree (*Dendrohyrax arboreus*) | | Exp | Y | Y | Y | — | Macaulay, 1963 in Hedger, 1981 |
| Ibex (*Capra sp.*) | | Nat | Y | — | — | — | Hediger, 1940 |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Impala (*Aepyceros melampus*) | Conspecific, cattle | Nat (SAT-1) | Y | Y | Y | N | Hedger et al., 1972 |
| | | Nat (SAT-1, 2, 3) | Y | Y | — | — | Vosloo et al., 2009 |
| | | Exp (SAT-2) | Y | Y | Y | N | Hedger et al., 1972 |
| | | Exp (O) | N | Y | Y | N | Anderson et al., 1975 |
| | | Exp (SAT-2) | N | Y | Y | N | Anderson et al., 1975 |
| | | Exp (SAT-1, 2, 3) | Y | Y | Y | N | Bengis et al., 1994 |
| Kangaroo, Eastern Grey (*Macropus giganteus*) | | Nat (O) | Y | — | Y | — | Bhattacharya et al., 2003 |
| | | Exp (A, SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Kangaroo, Red (*Megaleia rufa*) | Cattle | Exp (A, Asia-1, O, SAT-1) | Mild | Y | Y | — | Snowdon, 1968 |
| Kangaroo, Tree (*Dendrolagus matschiei*) | | Exp (A, SAT-1) | Mild | Y | Y | — | Snowdon, 1968 |
| Kouprey (*Bos sauveli*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| Kudu, Greater (*Tragelaphus strepsiceros*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| | | Nat (SAT-1) | Y | Y | Y | N | Hedger et al., 1972 |
| | | Nat (SAT-1) | Y | Y | N | — | Letshwenyo et al., 2006 |
| | | Exp (SAT-2) | Y | Y | Y | Y | Hedger et al., 1972 |
| Kudu, Lesser (*Tragelaphus imberbis*) | | Nat | — | Y | — | — | Anonymous, 1966 |
| Llama (*Lama glama*) | No transmission | Nat | — | Y | — | — | Marin et al., 2008 |
| | | Exp (A, C, O) | Mild | Y | Y | N | Fondevila et al., 1995 |
| Mink (*Mustela vison*) | | Exp (A, O) | N | Y (A) | Y (A) | — | Sahu and Dardiri, 1979 |
| Mithun/Gayal (*Bos frontalis*) | | Nat (A, Asia-1, O) | Severe | — | Y | — | Rajkhowa et al., 2003 |
| | | Nat (O) | Severe | — | Y | — | Hegde et al., 2011 |
| Mole, European/Common (*Talpa europaea*) | | Exp (A, C, O) | Severe | Y | Y | — | Capel-Edwards, 1971b |
| Mole rat, East African (*Tachyoryctes splendens*) | | Exp | Y | Y | Y | — | Macaulay, 1963 |
| Mongolian gazelle (*Procapra gutturosa*) | | Nat (Asia-1, C, O,SAT) | — | Y | — | — | Nyamsuren et al., 2006 |
| | | Nat(O) | — | Y | — | — | Bolortsetseg et al., 2012 |
| Moose/Eurasian elk (*Alces alces*) | | Nat | Y | — | — | — | Thomson et al., 2003 |
| Mouflon (*Ovis musimon*) | | Nat | Y | — | — | — | Folmer, 1957, cited in Schaftenaar, 2002 |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Mountain gazelle (*Gazella gazella*) | Conspecific | Nat (O) | Severe | — | Y | — | Shimshony et al., 1986 |
| | | Exp (O) | Severe | — | — | — | Perl et al., 1989 |
| Mouse (*Mus musculus*) | | Exp | Y | — | — | — | Capel-Edwards, 1971a |
| Nilgai (*Boselaphus tragocamelus*) | | Nat (O) | Severe | — | Y | — | Sujatha and Srilatha, 2007 |
| Nyala antelope (*Tragelaphus angasi*) | | Nat | Y | — | — | — | Thomson et al., 2003 |
| Oryx, Arabian (*Oryx leucoryx*) | | Nat (O) | Severe | — | Y | — | Ostrowski and Anajariyah, 2002 in Frolich et al., 2005 |
| | | Nat | — | Y | — | — | Frolich et al., 2005 |
| Peccary, Collared/Javelina (*Pecari tajacu*) | Conspecific | Nat | Y | — | — | — | Urbain et al., 1938 |
| | | Exp (O) | Y | Y | Y | — | Dardiri et al., 1969 |
| Peccary, White-lipped (*Tayassu pecari*) | | Nat | — | — | — | — | Pinto, 2004 |
| Porcupine, African (*Hystrix galeata*) | | Exp (O) | Mild | — | Y | — | Haq, 1951 |
| Potoroo (*Potorous tridactylus*) | | Exp (A, SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Possum, Brush tail (*Trichosurus vulpecula*) | | Exp (A, SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Pronghorn antelope (*Antilocapra americana*) | | Exp | Severe | — | — | — | Dunbar et al., 2009; Arzt et al., 2011a |
| Pudu, Southern (*Pudu pudu*) | | Nat | Y | — | — | — | Lindau, 1964, cited in Schaftenaar, 2002 |
| Rabbit (*Oryctolagus cuniculus*) | Conspecific | Exp | Y | Y | Y | — | Gins and Fortner, 1926, cited in Capel-Edwards, 1971a |
| | | Exp (A, Asia-1) | N | Y | Y | — | Snowdon, 1968 |
| Rat, African grass (*Arvicanthis niloticus*) | | Exp | Y | Y | Y | — | Macaulay, 1963 |
| Rat, Brown (*Rattus norvegicus*) | Conspecific | Exp (O) | Y | Y | Y | Y | Capel-Edwards, 1970 |
| Rat, Water (*Hydromys chrysogaster*) | | Exp (A, SAT-1) | Mild | Y | Y | — | Snowdon, 1968 |
| Reedbuck (*Redunca arundinum*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Reindeer (*Rangifer tarandus*) | | Nat | Severe | — | — | — | Ogryzkov, 1968 |
| | | Exp | Y | — | — | — | Kvitkin, 1959 |
| Roan antelope (*Hippotragus equinus*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Sable antelope (*Hippotragus niger*) | Conspecific | Nat | Y | — | — | — | Cotchin, 1947, cited in Macaulay, 1963 |
| | | Nat(SAT-1,2,3) | — | Y | — | — | Condy et al., 1969 |
| | | Exp (SAT-1) | Y | Y | Y | N | Ferris et al., 1989 |
| Saiga antelope (*Saiga tatarica*) | | Nat (A) | Severe | — | Y | — | Kindyakov et al., 1972 |
| Sheep (*Ovis aries*) | Many | Nat/Exp | Y | Y | Y | Y | Alexandersen et al., 2003; Arzt et al., 2011a |
| Springbok (*Antidorcas marsupialis*) | | Nat (SAT-1,2,3) | — | Y | N | — | Falconer and Child, 1975 |
| Squirrel, Grey (*Sciurus carolinensis*) | Conspecific | Exp (A) | Y | Y | Y | — | Capel-Edwards, 1971b |
| Squirrel, Indian (*Funambulus pennanti*) | No transmission | Exp (A, Asia-1, C, O) | Severe | — | Y | — | Tewari et al., 1976 |
| Steenbok (*Rhaphicerus campestris*) | | Nat | Y | — | — | — | Lindau, 1964, cited in Schaftenaar, 2002 |
| Swine (*Sus scrofa domesticus*) | Many | Nat/Exp | Y | Y | Y | N | Alexandersen et al., 2003; Arzt et al., 2011a |
| Tapir, Asian (*Tapirus indicus*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| Tapir, South American (*Tapirus terrestris*) | | Nat | Y | — | — | — | Urbain et al., 1938 |
| Thomson's gazelle (*Eudorcas thomsonii*) | | Nat (A, C, O) | — | Y | — | — | Anderson, 1981 |
| Topi (*Damaliscus korrigum*) | | Nat (O) | — | Y | — | — | Anderson, 1981 |
| Tsessebe (*Damaliscus lunatus*) | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Vicugna (*Vicugna vicugna*) | | Exp | — | — | — | — | Mancini, 1952, cited in Federer, 1969 |
| Vole, European water (*Arvicola amphibius amphibius*) | Conspecific | Exp (A, C) | Y | Y | Y | — | Capel-Edwards, 1971b |
| Vole, Field/Short-tailed (*Microtus agrestis*) | No transmission | Exp | Y | — | Y | — | Edwards, 1934, cited in Capel-Edwards, 1971a |

TABLE 1. Continued.

| Species | Confirmed transmission to species | Exposure and serotype | Clinical signs | Antibody positive | Virus positive | Carrier | Reference |
|---|---|---|---|---|---|---|---|
| Wallaby, Bennett's (*Wallabia rufrogrisea frutica*) | | Exp (A, SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Warthog (*Phacochoerus aethiopicus*) | Conspecific | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| | | Nat (SAT-1) | N | N | N | — | Hedger et al., 1972 |
| | | Exp (SAT-2) | Severe | Y | Y | N | Hedger et al., 1972 |
| Waterbuck (*Kobus ellipsiprymnus*) | | Nat | Y | — | — | — | Rossiter and Albertyn, 1947 |
| | | Nat (SAT-1, 2, 3) | — | Y | — | — | Condy et al., 1969 |
| Watussi/Wild buffalo (*Bos taurus*) | | Nat | Y | — | — | — | Lindau, 1964, cited in Schaftenaar, 2002 |
| | | Nat (A) | Y | — | Y | — | Barman et al., 1999 |
| Wildebeest, Black (*Connochaetes gnou*) | | Nat (O) | Y | — | Y | — | Walker, 1934 |
| | | Nat | Y | — | — | — | Urbain et al., 1938 |
| Wildebeest, Common (*Connochaetes taurinus*) | | Nat (A, SAT-1, 2) | — | Y | — | — | Anderson, 1981 |
| | | Nat (O, SAT-1, 2) | Y | — | Y | — | Swai et al., 2009 |
| | | Nat (SAT-1) | N | N | Y | Y | Hedger et al., 1972 |
| | | Exp (SAT-2) | N | N | N | N | Hedger et al., 1972 |
| | | Exp (O) | Mild | Y | Y | N | Anderson et al., 1975 |
| Wild boar (*Sus scrofa*) | Conspecific, swine | Nat | Y | — | — | — | Urbain et al., 1938 |
| | | Nat (O) | Y | Y | — | — | EFSA, 2012 |
| | | Exp (A) | Y | Y | Y | Y | Mohammed et al., 2011 |
| | | Exp (O) | Mild | Y | Y | N | Breithaupt et al., 2012, cited in EFSA, 2012 |
| Wombat (*Vanitatus hirsutas*) | | Exp (A, O, SAT-1) | N | Y | Y | — | Snowdon, 1968 |
| Yak, Domestic (*Bos grunniens/Poephagus grunniens*) | | Nat (O) | Y | — | Y | — | Prasad et al., 1978 |
| Yak, Wild (*Bos mutus*) | | Nat (O) | Y | — | Y | — | Barman et al., 1999 |

## THE SEROTYPES

All seven serotypes (O, A, C, Asia-1, SAT-1, SAT-2, and SAT-3) of FMDV have been found in wildlife. Within each serotype there are regional differences in the virus called topotypes. Topotypes can be sometimes used to determine the origin of the strain involved in an outbreak. Within the SAT viruses, there are at least eight topotypes within SAT-1, 14 in SAT-2, and six within SAT-3 (Vosloo et al., 2005). Apart from the SAT-type viruses found in African buffalo populations, the other serotypes are all endemic to livestock, and there is no evidence for any wildlife reservoir of other FMDV serotypes.

SAT-type FMD viruses have evolved with African buffalo populations. Young buffalo typically become infected with SAT-1 first, then SAT-2, and finally SAT-3, which indicates the differences in the adaptation of spread among the three serotypes (Vosloo et al., 2007). SAT-1 is more commonly found circulating in buffalo herds (Condy and Hedger, 1974). Buffalo are able to transmit to other susceptible wildlife (Dawe et al., 1994a), and SAT-2 appears to be the more common serotype transmitted to cattle and other wildlife species such as impala (*Aepyceros melampus*; Vosloo et al., 2007, 2009, 2011). There is also evidence for a changing pattern of infection with SAT viruses in impala. Previously SAT-3 was dominant from 1954 to 1968, while SAT-1 was isolated more from 1969 to 1982 (Bengis et al., 1994).

Foot and mouth disease virus appears to behave differently according to serotype and host factors. The exact mechanism of pathogenesis for each serotype in each possible host species has not been completely defined (Arzt et al., 2011a). Animals can be impacted by more than one serotype of FMD (Woodbury, 1995). There is also evidence that the serotypes may have varying degrees of pathogenicity among species and among individuals (Alexandersen et al., 2003). This may account for differences in the response of wildlife to different strains. Although this issue is critical and of concern in the epidemiology of FMD, it is beyond the scope of this review to evaluate and compare the pathogenicity of strains within and among species. Table 1 includes the serotypes, when available, of natural and experimental infections in a variety of species.

## THE CONTROVERSIAL CARRIER STATE

The importance of carriers or persistently infected animals is controversial. A carrier is defined as an animal with an unapparent infection and where virus can be isolated from the oropharynx beyond 28 days postinfection (dpi). Carrier states have been well studied for domestic animals. Cattle can carry FMDV for 3.5 yr after infection and goats and sheep up to 9 mo after an infection (Salt, 1993). Water buffalo (*Bubalus bubalis*) may carry virus for 1–2 yr (Moussa et al., 1979; Barros et al., 2007). In general, domestic pigs (*Sus scrofa*) do not appear to carry FMDV; however, Mohamed et al. (2011) isolated virus from feral pigs 33–35 dpi.

African buffalo may carry FMDV for >5 years, and virus may persist in a herd for 24 yr or longer (Condy et al., 1985). Typically, FMDV is transmitted only from acutely infected animals, and it appears difficult under experimental conditions for persistently infected animals or carriers to transmit virus to susceptible individuals (Condy and Hedger, 1974; Anderson et al., 1979; Bengis et al., 1986; Gainaru et al., 1986). Experimental studies have shown that the virus obtained from carrier animals can be less virulent in some susceptible animals but also has the ability to revert and regain virulence factors depending on the species (Salt, 1993). In experimental studies of carriers, the levels of virus that have been obtained from oropharyngeal fluid were 500 times lower than what is seen during acute infection (Woodbury, 1995;

Grubman and Baxt, 2004). Transmission from carriers may be more likely when the ratio of carrier animals to susceptible animals increases (Woodbury, 1995). Nonetheless, transmission has been demonstrated between African buffalo and from African buffalo to cattle under natural and experimental conditions (Hedger and Condy, 1985; Dawe et al., 1994a, b). Because FMDV may be isolated from semen (Bastos et al., 1999), sexual contact may be the source of transmission from persistently infected animals to susceptible cattle; there may also be a change in the virus or in the host's susceptibility (Dawe et al., 1994a; Woodbury, 1995).

Other species are capable of persistent infection, although not every susceptible wild species has been examined. Greater kudu (*Tragelaphus strepsiceros)* become carriers for up to 160 days (Hedger et al., 1972). Eland (*Taurotragus oryx*) can carry FMDV for 32 days, wildebeest (*Connochaetes taurinus*) for up to 45 days (Hedger et al., 1972), and sable (*Hippotragus niger*) remain viremic up to 28 days (Ferris et al., 1989). Fallow deer (*Dama dama*), sika deer (*Cervus nippon*), and white-tailed deer (*Odocoileus virginianus*) can become carriers (Forman et al., 1974; McVicar et al., 1974; Gibbs et al., 1975). With the exception of African buffalo where carrier transmission to other buffalo and cattle has been demonstrated, transmission by persistently infected livestock or wildlife to susceptible individuals has not been proven despite decades of research.

## GLOBAL STATUS OF FMD IN WILDLIFE

### Africa

Foot and mouth disease in sub-Saharan African wildlife has been studied since the early 20th century. Both natural and experimental infections have been demonstrated in many species. Antibodies to FMDV or clinical disease have been found in numerous species including the African buffalo, impala, eland, waterbuck (*Kobus ellipsiprymnus*), sable, greater kudu, lesser kudu (*Tragelaphus imberbis*), warthog, bush pig (*Potamochoerus porcus*), topi (*Damaliscus korrigum*), wildebeest (*C. taurinus and Connachaetes gnou*), duiker (*Sylvicapra grimmia*), bushbuck (*Tragelaphus scriptus*), African elephant (*Loxodonta africana*), giraffe (*Giraffa camelopardalis*), grysbuck (*Raphicerus sharpei*), reedbuck (*Redunca arundinum*), roan antelope (*Hippotragus equinus*), tsessebe (*Damaliscus lunatus*), gemsbok (*Oryx gazelle*), dorcas gazelle (*Gazella dorcas*), Grant's gazelle (*Gazella granti*), hartebeest (*Alcelaphus buselaphus*), nyala antelope (*Tragelaphus angasi*), springbok (*Antidorcas marsupialis*), steenbok (*Rhaphicerus campestris*), and Thomson's gazelle (*Eudorcas thomsonii*) (Walker, 1934; Urbain et al., 1938; Rossiter and Albertyn, 1947; Condy et al., 1969; Hedger et al., 1972; Howell et al., 1973; Falconer and Child, 1975; Anderson, 1981; Schaftenaar, 2002; Thomson et al., 2003; Swai et al., 2009; Ayebazibwe et al., 2010a; Arzt et al., 2011a; Vosloo et al., 2011).

Bush pigs and warthogs develop severe clinical disease after experimental infection but do not excrete virus as heavily as domestic pigs (Hedger et al., 1972). African elephants exhibited severe clinical disease in one experimental infection (Howell et al., 1973); however, the examination of thousands of culled elephants with exposure to FMD yielded no evidence of FMD in natural populations (Bengis et al., 1984). Clinical disease is rare in African buffalo, although an outbreak occurred while a group of animals were held in captivity (Vosloo et al., 2007). Severe outbreaks in impala have been reported in the Kruger National Park (KNP) for decades (Vosloo et al., 2009). Natural and experimental infections have occurred in giraffe, although they cannot transmit virus to other giraffe (Vosloo et al., 2011). Other African wildlife that have been infected experimentally include the impala, eland, sable antelope, greater kudu, common wildebeest, East African hedgehog (*Atelerix prurei hindu*), hyrax

(*Dendrohyrax arboreus*), East African mole rat (*Tachyoryctes splendens*), porcupine (*Hystrix galeata*), and grass rat (*Arvicanthis niloticus*) (Haq, 1951; Macaulay, 1963; Hedger et al., 1972; Anderson et al., 1975; Ferris et al., 1989).

Despite all the wildlife species that have been infected with FMDV, only African buffalo and impala (at least in southern Africa) have been implicated in the transmission of FMDV to cattle, particularly the SAT-type FMD viruses (Vosloo et al., 2002; 2009). African buffalo are a known reservoir for SAT-type FMDV (Condy and Hedger, 1974). Genetic and epidemiologic analysis has shown that buffalo have caused outbreaks in cattle surrounding the KNP (Vosloo et al., 2002, 2005). Young buffalo are acutely infected at 3–8 mo of age when their maternal antibodies wane. Once infected, they excrete virus in large amounts, and it is believed that this is the method of transmission to other species (Gainaru et al., 1986; Bastos et al., 2000). African buffalo are an important reservoir of FMDV in other parts of sub-Saharan Africa as well. In Uganda, 80% of buffalo sampled had antibodies to SAT-1, 2, 3, and possibly C and O (Ayebazibwe et al., 2010b). A survey of wildlife in Kenya, Tanzania, Ethiopia, and Chad found a majority of buffalo screened to have antibodies to at least one SAT serotype, while at most 11 (<2%) of other samples of nonbuffalo species were positive (Broonsvoort et al., 2008). In Ethiopia, there was a positive association between cattle that had FMD and contact history with wildlife. Cattle herds with the greatest antibody prevalence had the greatest number of contacts with wild animals and were located closer to wildlife sanctuaries where there were large populations of African buffalo (Molla et al., 2010).

Antelope species may propagate FMD. Impala, particularly in the KNP and possibly elsewhere in sub-Saharan Africa, have been associated with outbreaks in cattle (Vosloo et al., 2009). Eighty to 90 percent of infections in impala occur during the dry season from June through November (Bengis et al., 1994). This is the same time in which buffalo calves are losing maternal antibodies and becoming infected with FMDV (Bastos et al., 2000). Since impala have not been shown to become long-term carriers, it is suspected that impala are an intermediary host species and become acutely infected and spread the virus to cattle outside the KNP by jumping over fences (Vosloo et al., 2009). Kudu may play a role in a similar transmission pathway in other parts of sub-Saharan Africa (Letshwenyo et al., 2006; Vosloo et al., 2007). In Zimbabwe, an outbreak in cattle outside a privately owned conservancy implicated impala or kudu in the transmission (Hargreaves et al., 2004). In Tanzania, an outbreak of FMD occurred in the Serengeti impacting the wildebeest population and may have spread to cattle in the surrounding area (Vosloo et al., 2005).

Despite the significant role of African buffalo in the epidemiology of FMD, livestock and human movement remain significant causes of outbreaks. Past outbreaks that were blamed on African buffalo may have been caused by carrier cattle. In West and Northeast Africa where there is much less wildlife, SAT-1 and SAT-2 are maintained between epidemics in domestic animals (Vosloo et al., 2002). In Tanzania, FMD outbreaks from 2001 to 2006 appeared to be a result of human activity (Picado et al., 2011). The same was found in Uganda where human and livestock movements were the predominant cause of FMD outbreaks (Ayebazibwe et al., 2010b).

#### Eurasia/Central Asia

Outbreaks of FMD occur regularly in the countries of Central Asia. There are several important wildlife species that are impacted by FMD. Mongolian gazelles (*Procapra gutturosa*) from the Eastern Steppe of Mongolia have been infected by FMDV; however, several studies indicate that it is the continued circulation of FMDV in the domestic livestock of the region that results in the virus entering the

susceptible gazelle population (Nyamsuren et al., 2006; Bolortsetseg et al., 2012). There is no evidence for the persistence of the virus in the gazelle population between outbreaks (Thomson, 2011), and actions such as culling of Mongolian gazelles and fencing will not impact the disease in livestock (Bolortsetseg et al., 2012). It is also suspected, based on clinical signs, that Bactrian camels (*Camelus bactrianus*) in Mongolia and parts of Russia have been infected with FMDV during outbreaks in livestock (Wernery and Kaaden, 2004), as they have been shown to be susceptible to experimental infection (Larska et al., 2009). Almost all existing Bactrian camels are of the "domestic" type, maintained as domestic animals and physically distinct from wild Bactrian camels. Wild Bactrian camels are extremely rare and limited in geographic range (Roeder, 2009).

In Kazakhstan, saiga antelope (*Saiga tatarica*) are susceptible to FMDV and suffer from more severe disease than what is seen in domestic ruminants (Kindyakov et al., 1972). Saiga populations have declined dramatically due to excessive hunting in the 1990s. Mortality due to FMD can be high (as much as 75% in experimentally infected animals), and past outbreaks have resulted in a loss of 10% of a population. A decrease in outbreaks of FMD in saiga occurred when surrounding cattle were vaccinated. The direction of transmission of FMDV has been from livestock to saiga; however, during seasons of limited pasture, saiga may transmit to livestock through direct contact at shared pasture (Morgan et al., 2006). Saiga antelope also undertake seasonal migrations, providing opportunity to overlap with domestic ungulate ranges and can be found in Uzbekistan and Turkmenistan (Roeder, 2009).

#### Middle East

Foot-and-mouth disease has occurred in captive populations of Arabian oryx (*Oryx leucoryx*; OIE definition: captive wild animal) in Bahrain and the United Arab Emirates with high mortality, and antibody has been found in at least two captive individuals in Saudi Arabia. However, FMDV has not been detected in wild Arabian oryx populations (OIE definition: wild animal) in Saudi Arabia (Frolich et al., 2005). Antibodies to all seven serotypes of FMDV have been found in dromedary camels (*Camelus dromedaries*), a domestic animal (Yousef et al., 2012). They are believed to be mostly resistant to clinical FMD, and virus isolation is difficult. Natural and experimental infections have occurred in dromedaries; however, they are not believed to play a significant role in transmission to livestock (Wernery and Kaaden, 2004). Outbreaks in mountain gazelles in Israel have resulted in about 10% to 15% of the population becoming acutely infected with 50% mortality (Shimshony et al., 1986). Experimental infections in this species have confirmed the severity of the disease (Perl et al., 1989). Other reports of FMD in nondomestic animals include clinical disease in a captive Caucasian tur (*Capra aegagrus*) and mouflon (*Ovis musimon*; OIE definition: captive wild animal) (Schaftenaar, 2002).

#### Southeast and South Asia

Domestic water buffalo are common throughout Southeast Asia and are susceptible to FMD. In India, water buffalo are frequently kept with both cattle and sheep (Maddur et al., 2009). Maroudam et al. (2008) demonstrated that water buffalo can transmit FMDV to cattle and to each other. Clinical signs in water buffalo tend to be more covert than in cattle. Water buffalo appear to have a longer incubation period and are infective prior to exhibiting any lesions. Water buffalo can become acutely infected or become a carrier of FMDV (Maddur et al., 2009; Maroudam et al., 2008). Persistence of infection was found >1 yr after exposure (Barros et al., 2007).

Eurasian wild boar, feral swine, and feral water buffalo (OIE definitions: wild animal, feral animal, and feral animal, respectively) are often found in proximity to livestock and could play an important role in the epidemiology of FMD. In Sri Lanka, FMD outbreaks frequently occur in areas in close proximity to national parks where there are significant populations of feral water buffalo and wild boar (Roeder, 2009).

Most of the reports of FMD in Southeast Asia in wildlife are from India. Evidence of the initiation of an outbreak of FMD from nearby livestock was found in almost every case. Severe FMD has been reported in mithun (*Bos frontalis*), semidomesticated yak (*Bos grunniens*), and gaur (*Bos gaurus*) (Prasad et al., 1978; Verma and Sarma, 1997; Rajkhowa et al., 2003). Frequently, migratory herds of domestic cattle come into contact with these species and transmission occurs. Asian elephants (*Elephus maximus*) suffer moderately severe disease, particularly in younger animals (Pyakural et al., 1976), but serosurveys from captive groups found antibodies to FMD despite no history of clinical disease (Bhat and Manickam, 1997; Hedge et al., 2010).

There are several reports of infection of Asian wildlife in captive zoo animals including Eld's deer (*Rucervus eldii*), sambar deer (*Rusa unicolor*), spotted deer (*Axis axis*), and barking deer (*Muntiacus muntjak*) (Urbain et al., 1938; Barman et al., 1999). Severe disease occurred in captive nilgai (*Boselaphus tragocamelus*) and captive black buck (*Antilope cervicapra*) (Kar et al., 1983; Sujatha and Srilatha, 2007; Hegde et al., 2011). Suspected FMD outbreaks in European zoos resulted in several Asian species becoming infected including Asian tapirs (*Tapirus indicus*), babirusa (*Babyrousa babyrussa*), kouprey (*Bos sauveli*), and an Asian black bear (*Ursus thibetanus*) (Urbain et al., 1938; Neugebauer, 1976; Schaftenaar, 2002). Experimental infections have been conducted in Indian squirrels (*Funambulus pennanti*), and while resulting disease was severe, infected squirrels could not transmit virus to other squirrels (Tewari et al., 1976).

#### Australia

We found only one published experimental study conducted on the susceptibility of various Australian fauna (Snowdon, 1968). Clinical disease was unapparent for most infected animals, and only the red kangaroo (*Megaleia rufa*), tree kangaroo (*Dendrolagus matschiei*), water rat (*Hydromys chrysogaster*), and echidna exhibited mild clinical symptoms. The Bennett's wallaby (*Wallabia rufrogrisea frutica*), brown marsupial mouse (*Antechinus stuartii*), long-nosed bandicoot (*Perameles nasuta*), potoroo (*Potorous tridactylus*), brush tail possum (*Trichosurus vulpecula*), and wombat (*Vanitatus hirsutas*) did not show clinical disease. The Eastern grey kangaroo (*Macropus giganteus*) also did not exhibit clinical signs with an experimental infection of serotype A and SAT-1; however, during a natural outbreak in India of serotype O from cattle, one captive grey kangaroo was severely affected (Bhattacharya et al., 2003).

#### Europe

Despite experimental infections of several European cervids (Forman and Gibbs, 1974; Gibbs et al., 1975), reports of natural infection in captive animals at European zoos (Schaftenaar, 2002), and the recent report of infected roe deer (*Capreolus capreolus*) and wild boar in Bulgaria, there is no evidence for the maintenance of FMDV in wildlife in Europe (EFSA, 2012). Experimental infections have been demonstrated in red (*Cervus elaphus*), fallow, roe, sika, and muntjac deer with severe infections demonstrated in muntjac and roe deer (Forman et al., 1974; Gibbs et al., 1975). These species did not play a role in the epidemiology of recent outbreaks in the United Kingdom and the Netherlands (Moniwa et al., 2012), and transmission of FMDV to the abundant local deer and

wild boar populations (OIE definition: wild animals) did not occur (Elbers et al., 2003; Highfield et al., 2010). Additional experimental infections were conducted in European moles (*Talpa europaea*), water voles (*Arvicola amphibius amphibius*), and field voles (*Microtus agrestis*), which resulted in high mortality (Capel-Edwards, 1971a, b). Other natural infections that are suspected to have been FMD based on clinical signs only include infections in a chamois (*Rupicapra rupicapra*), captive ibex (*Capra* sp.) fallow deer, European bison (*Bison bonasus*), red deer, and roe deer (Urbain et al., 1938; Stroh, 1939; Hediger, 1940; Schaftenaar, 2002). Severe natural and experimental infections have been reported in reindeer (*Rangifer tarandus*) (Kvitkin, 1959; Ogryzkov, 1964). Experimental and natural infections have also been demonstrated in the European hedgehog (*Erinaceus europaeus*). There was suspicion that European hedgehogs were involved in outbreaks in livestock; however, there has been no further evidence of their role in FMD in Europe over the last 50 yr (Wolf, 1939; McLauchlan and Henderson, 1947).

Serologic surveys of cervids in Germany and of European bison in Poland failed to detect antibodies to FMDV (Kita and Anusz, 1991; Mouchantat et al., 2005; Frolich et al., 2006), although natural infections in captive zoo animals have been reported (Schaftenaar, 2002). A survey during and after the 2011 outbreak of FMD Serotype O in livestock in Bulgaria found a low antibody prevalence and clustered distribution of positive roe deer and wild boar (OIE definition: wild animals) indicating that FMD failed to become established in the wild populations. This suggests that European wildlife populations are not able to maintain FMDV in the absence of FMD infection in livestock (EFSA, 2012).

#### North America

There have been no confirmed outbreaks of FMD in wildlife in North America; however, there was one notable outbreak that is generally attributed to FMDV. An outbreak of FMD occurred in cattle in California in 1924, and there was suspicion that FMDV may have spilled over to the mule deer (*Odocoileus hemionus*) population that shared pastures. More than 22,000 mule deer were culled in an effort to prevent the spread of possible FMD. About 10% of the culled deer exhibited lesions that were believed to be FMD; however, no laboratory testing was conducted (Keane, 1927). Retrospectively, it is now recognized that the lesions found in deer may have been due to infection with epizootic hemorrhagic disease. Due to the lack of FMD in North America, most FMD research in the region has utilized either experimental infections or mathematical modeling. It has been proposed that high density populations of wildlife such as white-tailed deer and feral pigs, especially in areas where they receive supplemental feed and are hunted, may present a risk to commercial livestock industries if the virus were to be introduced into the United States (Ward et al., 2007). The mathematical model created by Ward et al. (2007) found that wild deer and feral pigs have the potential to amplify disease spread and form a possible reservoir of FMDV infection in Texas.

Experimental data indicate that there are many North American species that are susceptible to FMD and are capable of transmitting the disease to cattle. North American bison (*Bison bison*) and elk (*Cervus elaphus nelsonii*) have been infected with FMDV experimentally. The pathogenesis of FMD in bison is similar to cattle, while elk exhibit a mild disease that appears not to be transmissible to other elk or cattle (Rhyan et al., 2008). Mule deer and pronghorn antelope (*Antilocapra americana*) have been experimentally infected (Dunbar et al., 2009). White-tailed deer exhibited similar clinical signs of FMD as cattle and are capable of transmitting virus to cattle (McVicar et al., 1974;

Moniwa et al., 2012). McVicar et al. (1974) isolated virus up to 11 wk after infection in white tailed deer; however, Moniwa et al. (2012) did not find evidence of persistent infection. Other North American species that have been infected experimentally include the nine-banded armadillo (*Dasypus novemcinctus*), mink (*Mustela vison*), collared peccary (*Pecari tajacu*), and grey squirrel (*Sciurus carolinensis*) (Dardiri et al., 1969; Capel-Edwards, 1971a; Wilder et al., 1974; Sahu and Dardiri, 1979). Natural infections at non-North American zoos (OIE definition: captive wild animals) have been suspected in brown bears (*Ursus arctos*) and grizzly bears (*Ursus arctos horribilis*) and American bison (Urbain et al., 1938; Neugebauer, 1976).

#### Central and South America

There has been no evidence of transmission from wildlife to livestock in Central or South America and no history of outbreaks of disease despite the fact that many Central and South American wild animal species are susceptible to FMDV (Pinto, 2004). New World camelids can become infected with FMDV; however, they are not highly susceptible and appear to be unable to transmit the disease (Roeder, 2009). There was a suspicion of a mild infection in alpaca (*Vicugna pacos*), a domestic animal, during an outbreak in cattle; however, it was not confirmed (Wernery and Kaaden, 2004). Another study found a low prevalence of antibodies to FMDV in llamas (*Lama glama*), another domestic animal, in Argentina (Marin et al., 2008). Llamas were difficult to infect under experimental conditions, and when infected, they developed only very mild disease (Fondevila et al., 1995). Reports of susceptible species of wildlife in South America include experimental infections in vampire bats (*Desmodus rotundus*), capybaras (*Capybara capybara*), agoutis (*Dasyprocta agouti*), big hairy armadillos (*Chaetophracius villosus*), red brocket deer (*Mazama americana*), chinchillas (*Chinchilla lanigera*), coypu (*Myocaster coypus*), and brown brocket deer (*Mazama gouzoubira*) (Campion, 1950; Dellers, 1963; Capel-Edwards, 1967; Federer, 1969; Gomes and Rosenberg, 1984; Lord et al., 1986; Pinto, 2004). Capybaras and armadillos can transmit FMDV to cattle and swine under experimental conditions. Agoutis are capable of transmitting virus to each other. A captive southern pudu (*Pudu pudu*), South American tapirs (*Tapirus terrestris*), and a vicuna (*Vicugna vicugna*) were infected during outbreaks at European zoos (Urbain et al., 1938; Schaftenaar, 2002).

A recent study of marsh deer (*Blastocerus dichotomus*), an IUCN red listed vulnerable species in Brazil, found a low prevalence of antibody to FMDV serotype A, but no virus could be isolated from any individuals. Their role in the epidemiology of FMD in Brazil is unclear, and there does not appear to be evidence of transmission to livestock (Araujo et al., 2010). Other serologic surveys of South American wildlife have failed to find antibodies to FMDV. Free-ranging vicuna in Argentina and Bolivia (Marcopiddo et al., 2010; Beltrán-Saavedra et al., 2011), grey brocket deer (*Mazama gouazoubira*) in Bolivia (Deem et al., 2004), pampas deer (*Ozotoceros bezoarticus celer*) in Argentina (Uhart et al., 2003), pudus in Chile (Pizarro-Lucero et al., 2005), and guanacos (*Lama guanicoe*) in Argentina (Karesh et al., 1998) had no evidence of FMD (OIE definition: wild animals). Despite conjecture on the role of South American wildlife as a possible reservoir for FMDV, there is no evidence to date supporting that claim.

As mentioned previously, domestic water buffalo are also found throughout South America reared along with other livestock or in feral populations. Water buffalo in South America have been shown to transmit FMD to other livestock and have also been shown to carry the virus >1 yr (Samara and Pinto, 1983; Barros et al., 2007). This represents a more probable scenario for the transmis-

sion to other livestock species compared with more isolated wildlife populations. The extent of contact with feral buffalo populations that domestic livestock have and the amount of virus circulating in these populations has not been studied.

## OTHER EXPERIMENTAL INFECTIONS

Experimental infections with a variety of species have been attempted. The guinea pig was the laboratory animal model for studying FMD for many years (Capel-Edwards, 1971a). Other species exhibit clinical signs, and virus isolation is sometimes possible; however, their role in natural infections has been largely dismissed. Experimental infections have been reported in gerbils, hamsters, goldfish, jackdaws, cats, puppies, mice, rabbits, and rats (Capel-Edwards, 1971a). The brown rat (*Rattus norvegicus*) has been found experimentally to become a carrier of the virus for at least 19 wk (Capel-Edwards, 1970) although there has been no further implication in their role in the epidemiology of FMD.

## FMD IN HUMANS

Foot and mouth disease can very rarely be zoonotic. Most people can become mechanical carriers of the virus for up to 36 hr but do not become viremic. However, there are about 40 reported cases of humans becoming infected. These cases were in people with close contact with sick livestock. Clinical signs included fever, malaise, and oral blisters, which typically resolved in a week. Because human infection is rare, there is no significant public health concern, and FMD is not treated as a zoonotic disease (Nieldbalski et al., 2006).

## CONTROL AND MANAGEMENT OF FMD

### Feral animals

In many instances, government authorities consider feral animals similar to wildlife, and, in some cases, feral animals may serve similar epidemiologic roles as wild animals. However, feral animals in most of the world play a significantly different role from wild animals in FMD maintenance and transmission. In many cases, feral animals can be more easily managed than wild animals. For example, euthanasia or culling of feral animals such as feral goats (*Capra hircus*) or feral pigs is more supported by natural resource managers than culling of true wild animals.

Feral animals are more similar to domestic animals than most wild, native animals. They derive from domesticated genetic stock and so retain many of the physiologic and behavioral qualities of domesticated animals. They are also more likely to be in contact with domestic livestock and humans. Although there is little evidence for propagation of FMDV within feral populations (Roeder, 2009), there is a greater risk than from native wildlife. Feral animals in natural settings have not been studied extensively with regard to FMD; however, several studies suggest that feral swine carry the virus in their pharynx for at least 33–35 dpi (Mohamed et al., 2011; EFSA, 2012). An FMD outbreak in Israel was blamed on aerosol transmission from feral swine (Donaldson et al., 1987). Water buffalo are used extensively throughout Asia and South America and have clearly demonstrated the ability to carry FMDV and to infect cattle (Moussa et al., 1979; Barros et al., 2007). Therefore feral populations of this animal may present the most significant "wild" threat to domestic livestock.

### Stamping out/modified stamping out in wildlife

Mongolian gazelles, as mentioned previously, are not known to be a reservoir of FMDV but are passively infected when outbreaks occur in livestock (Nyamsuren et al., 2006; Thomson, 2011; Bolortsetseg et al., 2012). Modified stamping out, as was conducted in 2010 in Mongolia for Mongolian gazelles and livestock, involves culling of clinically diseased animals. This

method does not ensure a decrease in the spread of FMDV because not all infected animals demonstrate clinical illness and often FMD is infectious prior to the appearance of clinical signs. The controlled movement of people and livestock as well as vaccinating before and during outbreaks are more effective means of handling FMD outbreaks (Thomson, 2011).

#### Fencing

Veterinary cordon fences are commonly used to separate livestock from wildlife in southern Africa. Fences can be important for people who live adjacent to wildlife reserves as they may protect crops from damage and livestock from predation. Fencing is accepted by the OIE as a method of establishing FMD disease-free zones in southern Africa (Thomson et al., 2003; Jori et al., 2009) and can be effective. However, reliance on FMD exclusion provided by fences is problematic. There is a high cost of construction, maintenance, and patrolling of fences that may be cost prohibitive in some countries (Sutmoller et al., 2000). Fences are frequently subject to various environmental and human pressures, including flooding, breakage due to wildlife (particularly elephants), and damage from theft (Jori et al., 2009). The magnitude of fencing that exists in some parts of southern Africa (e.g., the 750-km fence surrounding parts of the KNP) makes it difficult for fences to be maintained (Jori et al., 2011). Compromised fences allow cattle to move into reserves and wildlife areas, and buffalo will exit a fence breakage after 48 hr of disrepair (Jori et al., 2011). Using a spatial model, Dion et al. (2011) found that the main risk to FMD transmission was interaction between livestock and buffalo that occurs when a fence break is left unrepaired for days or multiple fence breakages occur simultaneously in an area up to 6 km. When relying only on fencing to prevent FMD transmission, particularly over long distances, the vulnerability to FMD is heightened. Impala and other antelope species can easily jump a fence if not sufficiently high (Sutmoller, 2002).

Fences also may negatively impact wildlife and human populations. Fences interfere with normal migration patterns and in times of water scarcity may block wildlife from critical water sources. Fences also prevent genetic exchange among populations of various wild species, potentially resulting in inbreeding and loss of genetic diversity. Inadvertently fenced wildlife populations may remain small and capped, impacting their long-term survival (Hayward and Kerley, 2009), or they may exceed the carrying capacity of the land, resulting in overcrowding, malnutrition, increased infectious disease, and the need for added population control efforts.

In parts of southern Africa, revenues from tourism now exceed the total revenues of agriculture, fisheries, and forestry combined (Alberston, 2010). Strategies selected for FMD control need to ensure that the costs and benefits of controlling the disease for livestock production and export revenues are evaluated in context with existing or potential costs and benefits from other land-use choices such as wildlife managed for meat, trophies, hides, or tourism. Because wildlife tourism and the benefits to local communities can have significant economic value, stakeholder engagement in decision making about fencing and intensified livestock production is warranted for the long-term economic development of many parts of sub-Saharan Africa. Because fencing may provide other benefits to communities, decisions about restructuring of existing fence lines and the placement of new fences should consider the needs of people, livestock, and wildlife.

#### Vaccination

Currently, there are no commercially available FMD vaccines approved for use in wildlife. Vaccination of livestock in locations where eradication of FMD is not feasible can provide effective control

of FMD in the prevention or mitigation of an outbreak (OIE, 2012a); however, current vaccination programs are not providing maximum protection. Vaccine programs should be adapted to improve efficacy through the incorporation of appropriate strains based on the virus strains or topotypes circulating in the region. Increasing the frequency of vaccination to every 4 mo may be necessary with the currently available vaccines. Because current vaccines are labile and must be kept refrigerated, proper vaccine handling is critical. Postvaccination monitoring is needed to determine if the current programs are effective (Article 8.5.48 of OIE Code; OIE, 2012b). This would all necessitate an increase in vaccine production from current levels and would require significant financial investment (Paton et al., 2009; OIE Collaborating Center for Training in Integrated Livestock and Wildlife Health Management, 2010a; Paton and Taylor, 2011).

Recent advances in vaccine technology show promise in addressing some of the issues with vaccination. Traditional manufacturing of FMD vaccine requires the production of virulent FMDV, which is then inactivated. Inadvertent release of virus from vaccine manufacturing is a potential consequence and has been the source of past outbreaks. A newly developed technique uses infectious cDNA technology and does not require the production of virulent virus. The resulting attenuated virus also supports differentiating infected from vaccinated animals with a companion enzyme-linked immunosorbent assay. The technique may also simplify production and result in improved immunogenicity (Uddowla et al., 2012). Antiviral therapeutics may also be effective. The inclusion of a synthetic double-stranded RNA viral mimic with an adenovirus gene for interferon-alpha (Adt-pIFN-a) into an FMD vaccine can provide protection against viremia within 24 hr of administration, which could be protective and economical in the face of an outbreak (Dias et al., 2012) ). These techniques are new but appear very promising in the implementation of a successful vaccination strategy.

## POLICY OPTIONS

A FMD Global Control Strategy has been prepared under the umbrella of the Food and Agriculture Organization of the United Nations (FAO) and OIE Global Framework for the progressive control of Transboundary Animal Diseases. This strategy was developed with input of experts and representatives from national, regional, and international organizations and from FMD scientific reference centers and various stakeholder communities, including wildlife and natural resource managers, the private sector, and donor communities (OIE, 2012c; OIE FAO, 2012). The plan describes tools, methods, and strategies developed and agreed upon, including references to new or newly revised tools such as the relevant articles of the OIE Terrestrial Animal Health Code (OIE, 2012b) and the Progressive Control Pathway for FMD (see following section) (FAO EUFMD OIE, 2011). Strong, in-country veterinary services are considered prerequisite for implementing FMD control strategies. Effective surveillance and laboratory diagnostic programs as well as traceability and appropriate authorizing legislation are among the indispensable tools identified in the Global Strategy.

The FMD Global Control Strategy allows for adaptation to specific wildlife situations. Reference is made to the importance of protecting wildlife and biodiversity as well as the costs and benefits of controlling FMD compared to other revenue sources such as wildlife tourism. Research is also strongly supported notably for the development of new vaccines.

### Progressive control pathway for FMD

The joint FAO OIE Progressive Control Pathway for FMD (PCP) has been developed to assist countries where FMD is still endemic to progressively reduce the

impact of FMD and eventually enter into an eradication strategy for livestock. The PCP has been adopted by FAO and OIE as a standardized tool to be used for monitoring of FMD country (and some regional) control program implementation. The PCP defines levels of FMD control status according to implemented activities and achievements and consists of six stages ranging from zero (continuous FMDV circulation with no reporting or control actions) to five (the country is ready to be officially recognized by the OIE as free without vaccination). This phased, progressive approach and the described activities for each level can be used as the basis to design control programs (FAO EUFMD OIE, 2011).

Currently the OIE recognizes only three official categories for countries with regards to FMD: 1) Countries not free from FMD (corresponding to PCP stages 0–3), 2) FMD-free countries or zones practicing vaccination (corresponding to PCP stage 4), and 3) FMD-free countries or zones where vaccination is not practiced (corresponding to PCP stage 5) (Paton et al., 2009). The PCP is not intended to be prescriptive; rather, it is outcome oriented and acknowledges that the most effective approach to achieve the key outcomes might vary among countries and regions. Recognizing the myriad of governmental responsibilities, the PCP allows countries to determine priorities and strategies for the level of FMD control they are willing or able to pursue. Given the range of roles that wildlife may play in the circulation or persistence of FMD, countries implementing strategies that are in line with the PCP approach should benefit from this opportunity and the framework for natural resource managers and wildlife disease experts to be included in country and regional FMD prevention and control efforts.

#### Commodity-based trading

Commodity-based trading involves the trade in animal products that are deemed to be safe and pose a minimal risk of transmission of disease based on meeting established processing criteria. These criteria or conditions under which international trade of fresh meat of cattle and water buffalo from countries or zones that are not free from FMD is allowed are described in the article 8.5.25 of the OIE Code (OIE, 2012b). For fresh meat products, other processes such as appropriate refrigerated aging and deboning (removal of bones and lymph nodes) from healthy cattle from individual herds free of FMD and coming from a part of the country where cattle are regularly vaccinated against FMD is acceptable. For other meat products, proper cooking ensures the destruction of FMDV, making it allowable in international trade (Article 8.5.34 of the OIE Code; OIE, 2012b). Commodity-based trading therefore enables countries and zones access to international markets independent of their FMD status and provides mechanisms for managing FMD in wildlife separately from domestic animals (Thomson, 2009).

## CONCLUSION

Although FMD has been confirmed, through both natural and experimental infections, in more than 100 species of wild, domesticated, and laboratory animals, it is almost exclusively a disease of livestock. Foot and mouth disease has been studied for decades around the world, and the emergence of a wildlife reservoir, besides the African buffalo, has not been discovered. Wildlife has been suspected as the origin of apparent spontaneous outbreaks of FMD, but no scientific evidence exists to support this. Even in the midst of outbreaks of FMD in livestock, wildlife has either failed to propagate the disease or failed to become infected with FMDV.

Sub-Saharan Africa is the exception because it has the unique condition of having a significant reservoir of FMDV in a wildlife species. Therefore the region may

never achieve disease freedom defined geographically—at least in the near term. Efforts should be directed at developing opportunities for Africa to participate in international trade and to improve its economic situation without requiring complete eradication of FMD. Approaches to be considered regarding international trade are regionalization (zoning), compartmentalization, and recommended risk-based commodity mitigation measures. The development of internal markets within Africa will also assist in economic development during further disease control improvements.

New approaches should be more creative considering paradigm shifts and integration of interests of the livestock sector with wildlife tourism and production, and cultural and social concerns of local populations in order to achieve sustainable benefits to the different sectors. Mixed land-use scenarios such as those envisioned for the Transfrontier Conservation Areas where there is added utilization of wildlife resources could be highly beneficial economically and politically (OIE Collaborating Center for Training in Integrated Livestock and Wildlife Health Management, 2010b).

The diversity of FMD viruses and their behavior make it problematic to generalize any one strategy for management or control. The method used to control FMD will ultimately be regionally specific. To date, the scientific evidence indicates that outside of the sub-Saharan Africa situation with SAT types of FMD adapted to African buffalo, effective control of FMD in domestic livestock will result in both the protection of livestock and wildlife without requiring direct management or interventional activities directed at wildlife. Control of feral domestic animals may be required in some situations. Eradication of the virus will not be feasible in some places and may not be needed everywhere. In most parts of the world, effective FMD prevention and control efforts in domestic animals will prevent infections in wildlife populations and serve to protect this natural resource. This broader view of animal health and human livelihoods and well-being provides an opportunity for animal health scientists to make a significant contribution to global good.

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