Journal of Current Veterinary Research



ISSN: 2636-4026

Journal homepage: http://www.jcvr.journals.ekb.eg

Animal health & Zoonotic disease

Epidemiological Patterns of Foot and Mouth Disease in Egypt and Other African Countries

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ABSTRACT

FMD is a severe and highly contagious disease of all cloven foot domestic and wild animals. There are seven immunologically distinct serotypes (O, A, C, Asia 1, SAT1-3) with several topotypes within each serotype. Hence, there is not complete crossprotection between different serotypes and topotypes of the same serotype. The epidemiology of FMD is more complicated in Africa than in any other parts of the world. This is due to six serotypes are circulating in Africa and there are considerable regional differences in the distribution and prevalence of various serotypes and topotypes. In Egypt, FMD is considered as a major transboundary disease that produces great restrictions on trade of animal and animal products.FMDis transmitted from diseased to susceptible animal by inhalation of exhaled contaminated air. Perhaps, some wild animals contribute to FMD transmission. The African buffalo, *Syncerus caffer*, was observed to be a true maintenance host for serotype SAT1-3 in West Africa. RT-PCR has been estimated at the world reference laboratory (WRL), Pirbright, UK for the routine diagnosis of FMDV using universal primers for all serotypes and serotype-specific primers. Thespeed and direction of the wind, ambient temperature and humidity, and animal movement are important Factors enhancing the rapid spread of the disease within the herds. For the effective control of FMD, outbreaks should be identified at an early stage and persistent infections must be detected. These can be occurred by regular vaccination and using rapid and specific diagnostic tools.

Keywords: Africa, Epidemiology, FMD, Syncerus caffer and WRL.

INTRODUCTION

Foot-and-mouth disease (FMD) is aglobal major significant cattle disease due to its vast distribution, transboundary nature, and serious economic implications (Knight-Jones and Rushton, 2013). The Foot and Mouth Disease Virus (FMDV), an aphthous virus, is incredibly destructive and has a big impact on animal health and production. The disease was first discovered fifteen centuries ago in Venice and now affects both domestic animals with cloven hooves (such as cattle, pigs, sheep, and goats) and wild populations of animals with cloven hooves all over the world (Alexandersen and Mowat, 2005; Rowlands, 2008). Since its detection, the virus has spread to several other countries. Up until the 1950s, the virus was known to have three serotypes; after that, four additional serotypes were noted for the virus (Jamal and Belsham, 2013).

FMDV has different serotypes and numerous sub-lineages or topotypes. In the field, it is still evolving, creating new strains that periodically cause an increase in the number of cases and raise the risk of the virus spreading to new areas. There is a global clustering of FMD viruses, and these have been split into seven virus pools. Each pool has its own topotypes or lineages, but each pool also contains many serotypes. Africa is covered by three pools, Asia and the Middle East by three pools, and the Americas by only 1 pool (Knowles and Samuel, 2003; Rweyemamu et al., 2008; Brito et al., 2017). Each pool can use more specialized vaccines relevant to the topotypes prevalent in that pool rather than dependence on the more readily available vaccines; as vaccination is employed as a significant tactic for control in endemic areas (Hammond et al., 2012).

1. <u>Etiology:</u>

The virus is a single-stranded, positive sense RNA aphthovirus that belongs to the family Picornaviridae (i.e. genome is approximately 8500 base). The virus has seven distinct serotypes that are known as serotypes O, A, C, Asia1, SAT1- 3 (Hussein et al., 2021; Brown et al., 2022).

The viral topographical presence in various nations and regions has been determined by the virus' topographical spread. In this respect, serotype A is divided into three topotypes: Africa, Asia, and Europe-South America (Euro-SA) (Bari et al., 2014). Based on nucleotide sequences and genetic studies, the serotype A virus diversified to 26 genotypic lineages in addition to having a genetic variation of about 24% between various international topotypes and more than 15% in viral protein 1 (VP1). When it came to genetic diversity for Euro-Asian serotypes in the 1970s, there were roughly 32 subtypes that could be distinguished (Grubman and Baxt, 2004; Bari et al., 2014). Additionally, the

Middle East and South Asian regions are where the Asian topotype is most prevalent, with identified lineages such as A15, A22, A-IRN99, A-Iran05, A-IRQ24, 46, A-TUR2006, etc. According to Knowles et al. (2009); Jamal et al. (2011), the A-Iran 05 lineage is associated with dominance in the West-Eurasian area. According to the virus capsid's sequence analysis, there are four distinct genotypes of the African serotype A (I, II, IV, and VII) (Bari et al., 2014).

FMDV's icosahedral structure can be distinguished thanks to the viral proteins (VPs)-organized capsid proteins (VP1-VP4). The VPs (1-4) are situated below the other proteins, while the VP1-3 are situated externally (Longjam et al., 2011). VP1-4, which are structural viral proteins, are encoded in the P1 region (Seago et al., Because of its two primary 2012). immunogenic regions at the C terminus (residues 200 - 213) and G-H loop (residues 141 - 160), the VP1 has 213 residues and has the capacity to affect the virus' antigenicity and immunogenicity (Grubman and Baxt, 2004; Belsham and Martinez-Salas, 2019). in addition to the serotypes differentiating and the attachment to cell bases for cellular entrance. As a result, the nucleotide sequencing of FMDV VP1 has become the gold standard method for characterizing the genetic makeup of the virus (Grubman and Baxt, 2004; Jamal et al., 2011).

2. <u>The Virulency of Different</u> <u>Serotypes of Foot and Mouth Disease</u> <u>Virus:</u>

There are seven different FMD virus serotypes: O, A, C, Asia-1, SAT1, SAT2, and SAT3 (Longjam et al., 2011). A significant study was conducted in Pakistan in (2022) by Qureshi et al. to compare the pathogenicity of seven FMD serotypes. They came to the conclusion that compared to serotypes 'O' and 'Asia-1,' FMD virus serotype 'A' generates higher cytopathic effects. While serotypes "A" and "O" showed less variation in pathogenicity. Their results corroborated those of an earlier study carried out in the Republic of Korea, which showed that FMD virus serotype 'O' may spread easily to both cattle and pigs independent of the donor species. In contrast, serotype "A" is exclusively spread to swine and affects steers less severely whereas when pigs became infected with serotype "A" through direct contact, direct inoculum, or infection with a virus derived from cattle, they exhibit a severe, rapid, contagious illness (Pacheco et al., 2016).

These findings could be explained by the fact that the FMD virus uses v3 integrin as its major receptor for infection and that type O1 virus adaptation to cell culture allows the virus to use heparin sulphate (HS) as a receptor. Serotype A12 cannot reproduce in human cell line K562 because this integrin is missing, hence these cells must be transfected with cDNA encoding this integrin (K562-v3) in order for serotype A12 to do so. Contrarily, type O1 FMD viral infection requires cell surface heparin sulphate, and tissue culture FMDV type O1 virus was capable of multiplication in untransfected K562 cells (Neff et al., 1998).

3. <u>The disease in humans (Zoonotic</u> importance of FMD):

Even though FMD is a mild zoonotic disease that can infect humans, it does so slowly and with minimal impact. With the animal prevalence illness' substantial throughout history and in more recent outbreaks around the globe, its appearance in humans is unusual (Bauer, 1997). As a result, there are few reported cases of human infection. The last human case of foot and mouth disease in Britain was reported during the final pandemic of the disease in 1966 (Armstrong et al., 1967). All cases that have been documented have included close contact with sick animals, however it is

unclear why it sometimes affects people. One tale from 1834 claims that three veterinarians got sick after purposely ingesting raw milk from sick cows (Hertwig, 1834). The most often isolated viruses in humans are of type O, followed by type C and, less frequently, type A. The human incubation period lasts for two to six days. The majority of the symptoms, which include unpleasant tingling blisters on the hands, fever, sore throat, blisters on the feet, and blisters in the mouth, including the tongue, have been mild and self-limiting (Bauer, 1997). Generally, patients are fully recovered a week following the final blister creation. No reports of person-to-person transmission exist (Prempeh et al., 2001). It is important to distinguish between foot and mouth disease and hand, foot, and mouth disease in humans. The coxsackie A virus is primary cause of this unusual, generally mild viral infection, which mostly affects children (Chin, 2000).

4. <u>The disease in animals:</u>

Concerning small ruminants (SR), fever, depression, lameness, and infant mortality are the most prevalent clinical symptoms; while vesicles formations are less frequent (Kitching and Hughes, 2002). Although adults are frequently asymptomatic, the virus frequently spreads undetected among SRs despite the fact that lambs and kids might experience severe mortality (Kitching and Hughes, 2002). Moreover, most of diseased cases become carriers, but their epidemiological role isunclear. Additionally, there is a dearth of studies on goats, and there are few studies on sheep that were conducted under experimental conditions (Stenfeldt and Arzt, 2020; Stenfeldt et al., 2020).

Regarding large ruminants (LRs), Low mortality and significant morbidity are the disease's clinical characteristics (Alexandersen *et al.*, 2003). Vesicle in, lips, the tongue, muzzle, dental pad, hard palate, inter-digital space, coronary band, and gumsis the pathognomonic sign of FMD in accompanied large animals and is bydepression, salivation anorexia, and lamenesswhich results in low production (Kitching, 2002a; 2002b; 2002c; Knight-Jones and Rushton, 2013). Traditional definitions of persistent FMDV infection in cattle refer to a threshold of 28 days that was selected based more on experimental than biological evidence (Sutmoller et al., 1968). According to this accepted definition, an animal is regarded to be an FMDV carrier if infectious FMDV can be found in it more than 28 davs after infection (World Organization for Animal Health, 2017). However recent studies showed that cattle who recover from infection before becoming FMD carriers, typically do so much sooner than previously believed (Stenfeldt and Belsham, 2012; Stenfeldt et al., 2016). More specifically, it was shown in one set of trials that carrier status could be identified in vaccinated calves as early as ten days and in non-vaccinated cattle as late as 21 days. The transitional stage, which denotes the period during which infection clearance takes place, was made possible by the attainment of a more accurately defined timeline of the FMDV carrier state divergence (Stenfeldt et al., 2016; Arzt et al., 2019).

In Africa, the FMD infection in domestic animals is characterized by an acute clinical phase that lasts two weeks and includes fever and vesicles. Yet, FMDV also promotes, persistent subclinical infection in oropharyngeal sheep tonsils and nasopharvnx epithelial cells of cattle (Stenfeldt et al., 2016; 2019; Stenfeldt and Arzt, 2020). In addition, there is a subclinical infection that manifests early (neoteric phase), with higher levels of transmissibility and shedding than the persistent phase (Stenfeldt and Arzt, 2020). Acute clinical illness is uncommon in

African buffalo (Vosloo et al., 2002), and the palatine tonsil develops a persistent infection (Maree et al., 2016).

5. <u>Methods of transmission:</u>

During acute infection, viral shedding from burst vesicles and in body and excretions secretions facilitates transmission (Alexandersen et al., 2003). By direct contact with acutely infected animals or indirectly through the inhalation of from contaminated aerosols objects. susceptible ruminants can become infected with very low quantities of the inhaled virus. Moreover. pigs are largely resistant when contracted toinfection through inhalation (Alexandersen et al., 2003). A higher virus dose is needed for infection via other routes, such as ingestion or abrasions. FMDV can live in the environment and in different animal products, for days to months depending on the circumstances (Sellers, 1971). Nonetheless, certain ruminant hosts continuous to carry the virus and become carriers of FMDV in particular nasopharyngeal epithelial regions and associated lymphoid tissues (Stenfeldt et al., 2016).

6. <u>Reservoirs:</u>

The African (Cape) buffalo (Syncerus caffer) is the FMDV SAT serotypes' natural reservoir, but SATscan infect other wild animals and feral species, such as impalas (Aepyceros melampus melampus), and it can spread among domestic livestock in Africa (Vosloo et al., 2002; Thomson et al., 2003). Although some buffalo populations have significant FMDV seroprevalence (Hunter, 1998; Bronsvoort et al., 2008), and contact that occurs between animalsduring grazing in the same areas, it is unclear how often FMDV spreads from buffalo to livestock (Casey-Bryars et al., 2018; Omondi et al., 2020). By assessing the genetic similarity of isolated viruses taken from buffalo and cattle. molecular

epidemiology can provide a background to clarify FMD transmission (Omondi et al., 2019; 2020). One host can harbor a variety of viral genomes due to point mutations in RNA viruses during rapid viral replication and the RNA polymerase's lack of proofreading abilities (Andino and Domingo, 2015). African buffalo can also have concurrent multi-serotype FMDVpersistent infections, which creates the right conditions for related viruses to recombine (Maree et al., 2016; Ferretti et al., 2018). Hostrange, viral fitness and transmissibility may all change as a result of these evolutionary mechanisms.

According to Charleston (2011), Up to 28 days after infection, viruses may typically be isolated from cattle, and diseased cattle become carrier for the virus up to 3.5 years (Grubman and Baxt, 2004). According to this theory, carrier animals act as reservoirs of disease when previous waves of FMD have vaccinated the population, allowing reinfection when the immunity of non-carrier animals starts to decrease (Guyver-Fletcher et al., 2022).

Wildlife in other regions of the world may contribute significantly to the spread of FMD (Ward et al., 2007). Experimentally, all British deer species are contagious and capable of spreading viruses to domestic livestock (Gibbs et al., 1975). Despite exhibiting very moderate clinical symptoms, wild boars are vulnerable and can spread infection to domestic pigs (Breithaupt et al., 2012). After the 2001 outbreak, diagnostic andserologicaltests of wild deer and boar and in the UK, Holland, and Germany did not find any positive animals (Elbers et al., 2003; Mouchantat et al., 2005). Yet, after livestock epidemics in Thrace, seropositive roe deer and wild boar were discovered (Dhollander et al., 2016). In Europe, models typically come to the conclusion that if there are no outbreaks in cattle, deer and boar populations cannot maintain infection (Croft et al., 2019). Although there is no proof that deer or boar have contributed to FMD transmission in the UK, there is still a danger that they may have helped the illness spread locally. Although rodents and hedgehogs can carry the FMDV virus, they don't play a considerable role in its transmission (Thomson et al., 2003). When infected, animals such as scavengers like foxes, crows, and seagulls can mechanically spread FMDV (Sutmoller et al., 2003; Sellers, 1971).

7. <u>Sources of infection:</u>

FMDV is rapidly spread and can live in the environment (Cottral, 1969; Bartley et al., 2002). According to Gibbens et al. (2001); Sutmoller et al. (2003), people can spread FMDV to vulnerable animals by carrying it on their bodies, clothing, and shoes. Moreover, equipment and vehicles might serve as fomites (Btner and Belsham, 2012). In Japan in 2000, imported straw was determined to be the most likely entry point for an outbreak (Sugiura et al., 2001).

8. <u>The viral resistance and sensitivity:</u>

Temperature lower than 50°C, relative humidity higher than 55 %, and a pH of 7, all are favorable conditions for virus survival (Colenutt et al., 2018; 2020). Disease spread over long distances (up to 50 km on land and up to 200 km on water) and short distances (between adjacent farms within 2000 m) is also associated with airborne transmission (Donaldson, 1983; Gloster et al., 2003).

According to Cottral (1969); Bartley et al. (2002), FMD can live on the soil at temperatures greater than 16°C for 2-5 days, at 3-7.5°C up to 5 weeks, and less than 5°C over 20 weeks. The amount of time a virus may survive changes depending on the virus strain and is influenced by changing temperatures and rising R.H (Cottral, 1969). Regarding pH, The picornavirus that causes foot-and-mouth disease is likely the most sensitive to pH; at a slightly acidic pH, it begins to dissociate into pentamers, which is followed by genome uncoating and infection (van Vlijmen et al., 1998; Berryman et al., 2005).

For almost one hundred days at temperatures above 16°C in winter, FMDV can thrive on bedding and foodstuffs such as straw and bran (Cottral, 1969; Bartley et al., 2002; Auty et al., 2019). According to Btner and Belsham (2012), the virus can survive in manure at 20 °C for up to 9 days and at 5 °C for up to 14 weeks. Virus remains active even after drying to surfaces (Krug et al., 2012; 2018).

9. <u>Role of disinfectants in control of</u> <u>FMD outbreaks:</u>

In general, the primary eradication strategies for foot-and-mouth disease (FMD) are stamping out the disease and restriction of movement. It is also important to completely disinfect the infected area to prevent the spread of FMDV, including vehicles and people as well. Acidic ethanol disinfectants, alkaline cleaners and sodium hypochlorite had great effectagainst FMDV. On the other hand, neutral ethanol disinfectants, hand soaps, and quaternary ammonium compound sanitizers did not show great effect against FMDV. Therefore, it is presumed that acidic ethanol disinfectants are effective for human use and alkaline cleaners are effective for use in the infected environment for the control of a FMD outbreak (Harada et al., 2015).

10. <u>Global situation of FMD:</u>

FMDV affects a variety of animals with cloven hooves like cattle, pigs, goats and sheep (Arzt et al., 2011a; b). FMD normally has no fatal effects on adult animals but is extremely contagious and can lead to considerable losses in agricultural output, abortions, and higher mortalities in young animals. Outbreaks of FMD have indeed resulted in significant financial losses

across the globe (Knight-Jones and Rushton, 2013; Onono et al., 2013), making it a serious danger to the world's livestock business. According to estimates, FMDV has an annual impact on the world that ranges from 8 to 22.5 billion US dollars (Knight-Jones and Rushton, 2013). Despite this immense global impact, the genesis of the disease, its global dissemination, and its methods of transmission remain mostly poorly understood (Aiewsakun et al., 2020). According to Marqués et al. (2019), the illness is prevalent in portions of South America, Asia, the Middle East and Africa and occasionally produces outbreaks in formerly free nations and areas.

A revised theory about FMDV's widespread colonization mentioned that the increase in commerce and animal traffic from the Mediterranean to Northern European countries between the thirteenth and fifteenth centuries certainly helped the ancestor of the Euro-Asian FMDV strains spread to Europe. During the Period of Discovery (the fifteenth through the eighteenth centuries), the groundwork for European global trade was created, which probably aided the disease's spread from Europe to Asia. In the late 1860s, European immigrants shipped cattle from Europe to Argentina, which is likely what led to the spread of FMD throughout South America. On the other hand, the SAT strains are quite tightly restricted to the continent of Africa. They probably have been spreading among African agricultural and wild animals for a very long period, which is crucial to the ongoing spread of the disease throughout the continent. Yet, it is uncertain if the disease initially originated in Africa and subsequently moved to Mediterranean nations, or whether it happened the other way around (Aiewsakun et al., 2020).

According to estimates from the World Organization for Animal Health (2022a), FMD continues to be an endemic disease that mostly affects animals in tropical Africa and Asia. The disease is still believed to be common even though it is regarded as managed in many high- and middle-income environments (WOAH) "Disease free territories" with or without vaccination"). FMD outbreaks frequently recur in several of these countries even after the application of prevention and control measures like mobility restrictions, preemptive emergency or vaccination, movement increased biosecurity, bans. stepped-up surveillance. and social awareness and education programs (Maree et al., 2014; Blacksell et al., 2019). These methods have produced inconsistent to no results since different nations have different goals for animal health, varied financial and logistical resources, and different implementation and enforcement methods.

Many epidemiological aspects contribute to transmission of FMDV, the complication of the demonstrating mechanisms underpinning virus introduction, dissemination, and persistence in endemic areas (Dos Santos et al., 2017; Squarzoni-Diaw et al., 2021). In endemic locations, it can be challenging to collect data for a precise epidemiological picture of FMDV. The methods which cause FMDV endemic in some regions, however, can be addressed using outbreak records, a widely utilized and available data source for investigating contagious illnesses. Countries commonly keep databases of FMD epidemic because veterinary services data of government agencies play a vital role in identification response, outbreak and especially when the disease is of great economic value or is being targeted for control and eventual eradication. Of the 85 illnesses identified by WOAH and described in the animal terrestrial code. FMD is one whose official outbreak data has been gathered and made public via the WOAH-WAHIS database (World Organization for

Animal Health, 2022b). Regional veterinary services from all over the world produced reports that served as the basis for this data collection. The Progressive Control Pathway (PCP) for FMD (FAO et al., 2018), a global progressive control method for FMD led by the FAO, is one example of an evidencebasedFMDV circulation management method that heavily relies on outbreak information. Large-scale outbreak data are understanding crucial for the epidemiological situation, developing treatments, and evaluating the effectiveness of FMDV control approaches.

11. <u>The epidemiological situation of the</u> <u>disease in Africa:</u>

Three topotypes for serotype C, 6 for serotype O, 2 for serotype A, and 9, 14, and 5 topotypes for SAT1, 2 and 3, respectively, have been found in Africa (WRLFMD, 2003; Di Nardo et al., 2011). Three FMDV pools have been identified in Africa; Pool (4) for East Africa, Pool (5) for West Africa, Pool (6) for Southern Africa and Pool (7) for Southern Africa with serotypes SAT1, 2, and 3 (Paton et al., 2009).

11.1. <u>Occurrence of FMD in North</u> <u>Africa</u>

North African countries include Tunisia, Algeria, Morocco.Western Sahara, Egyptand Libya and According to Knowles et al. WRLFMD (2003) and (2007), the main means by which FMDV spreads to North African countries from sub-Saharan Africa and the Middle East is through the movement of livestock, and domestic animals are most likely where the virus is maintained (Hall et al., 2013). FMDV in this area has previously been linked to sheep populations (Rweyemamu et al., 2008). The prevalence of FMD was infrequent and typically resulted from exogenous causes in the Western portion of this region, which includes Morocco, Algeria, and Tunisia (Yehia and Primot,

2009). Although commerce between these countries and the rest of Africa is relatively minimal, the movement of small ruminant herds within the region may have contributed to the introduction of FMD from Tunisia to Moroccoin 1989 and 1991 (Aidaros, 2002).

I. <u>History of FMD epidemics in North</u> <u>Africa before 1999:</u>

O, A, C and SAT2 are the four serotypes that have historically been detected in North Africa. Serotype O is the most frequent serotype, after that serotypeA. Egypt and Tunisia, respectively, have recorded cases of SAT2 and serotype C, respectively. The last outbreaks that were documented in North African nations before 1999 were in Egypt (1997 serotype O), Tunisia (1994, serotype O), Libya (1994, serotype O) Morocco (1992, serotype O), and Algeria (1990, serotype O) (WRLFMD, 2017). The Algerian government planned a immunization monovalent type 0 administered annually solely to cattle in 1993 and 1994. However, due to the climate political at the time. the immunizationprogram was abandoned. The similar approach was used in Morocco during the same years, when cattle were vaccinated annually with a monovalent type O vaccine up until December 1997. From 1989. Tunisia has also vaccinated susceptible animals every year. Small ruminants received a monovalent O vaccine, whereas cattle got a trivalent vaccination (A, O and C) (FAO, 1999). Due to the lack of information on the state of animal health and the available management measures, there has been no data about the vaccination strategies for Libya (FAO, 1999). The illness has been recorded in Egypt since the 1950s, when a strain SAT2 outbreak led to the disease's first detection.InFebruary 1987, there were 230 pigs, 63,430 cattle, 11,178 sheep and goats infected with FMD in Egypt, the mortality rates were 100%, 4%,

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and 2%, respectively (FAO, 1999). Furthermore, an outbreak involving 1,827 buffaloes and 2,027 cattle was recorded in March 1993 in 11 governorates and 20 foci. As a result, immunization against FMD (serotype O) has been required and free of charge in Egypt since 1987 (FAO, 1999).

II. <u>The 1999 epidemics of FMD in North</u> <u>Africa:</u>

Over 78 million head of livestock were in danger for FMD in North African nations in 1999 due to the fact that the majority of these animals had not received any FMD serotype-specific vaccinations. Two possible FMD cases in cattle were discovered in the Algerian province of Algiers on February 20 and 21 of 1999. Aseptically obtained vesicular material was transferred to the Pirbright World Reference Laboratory (WRLFMD) for evaluation. As soon as the serotypes were verified to be type O, FAO was constantly informed (FAO, 1999). The virus was genetically distinct from the strains of type O virus that were detected at the same time in the MENA, according to a sequence analysis of the virus. The sequencing analysis revealed that the 1999 isolates from Guinea and Côte d'Ivoire had 99% similarity with the Algerian viruses, which belonged to the West African topotype (Samuel and Knowles, 2001). These findings supported the theory on the disease's origin. In fact, zebu cattle had been smuggled past the southern borders of Algeria in February 1999. These zebu cattle did not exhibit any clinical symptoms of FMD at the time of capture (Samuel and Knowles, 2001). Yet, their existence showed that there were transboundary animal movements along the southern border with FMD-endemic Niger and Mali. 179 outbreaks were documented from the start of the epizootic until June 22, 1999, in 36 of the epizootic's 48 districts. FMD cases were found on February 22, 1999, in Tlemcen (58 kilometers from the

Moroccan border) and the Souk Ahras region (50 kilometers) (FAO, 1999). Five days after the disease was declared in Algeria, on June 25, 1999, in the area of Oujda, there was suspicion of the first case of foot and mouth disease in Morocco. Foot disease-specific and mouth clinical symptoms and lesions were seen (FAO, 1999).The O/MOR/1/99 and O/MOR/2/99 serotypes, which shared 99% of their genetic makeup with the virus that first arose in Algeria, were confirmed by the WRLFMD. Additionally, the disease was discovered two weeks later in the Khouribga and Beni Mella district demonstrating the disease's contagiousness, spreading over large geographic areas, and infecting susceptible flocks (Samuel and Knowles, 2001: WRLFMD, 2017). Despite the launch of vaccination to stop the entry of FMD through the Algerian border, an FMD epidemic was discovered in the Nabeul province of Tunisia on March 1st, 1999. The WRLFMD verified the FMDV in all vulnerable animals. Serotype 0 (O/TUN/1/99 and5/99) was the strain in question, and it shares a striking genetic resemblance with the virus that was discovered in Algeria and Morocco in 1999 (WRLFMD, 2017). In Libya and Egypt, there was no breakout that was noted during this epidemic (OIE, 2017).

III. <u>FMD epidemic in North Africa from</u> 2000 to 2013:

Reporting from 2000 to 2013 between 2000 and 2013, there were a number of FMD outbreaks in Libya that were documented. The SAT2 strain of the virus, which started the outbreak's first wave in 2003 but eventually self-limited and never crossed international boundaries, was to blame. 2009 saw the detection of serotype A, topotype Asia lineage Iran-05. Another epidemic was reported in 2011. FMDV was found to be O serotype, topotype MESA, and lineage PanAsia2 (WRLFMD, 2017).

On February 27, 2012, Libya formally informed the OIE about FMDV serotype SAT2 outbreaks linked to recently imported cattle in Benghazi. Sequence analysis revealed that the serotype was identical to SAT2 viruses discovered in Nigeria in 2007 and the serotype that caused a prior FMD outbreak in Sudan in 2007. Serotype O and SAT2vwere verified from samples obtained in 2012 (WRLFMD, 2017). Information on the prevalence of the disease is becoming hard to get because of the political unrest in Control measures, Libya. like as vaccination, were put in place, especially after the release of novel serotype SAT2 strains in 2012. The OIE Reference Laboratory's (IZSLER) Istituto Zooprofilattico Sperimentale di Brescia, Italy, conducted a serosurveillance (Hall et al., 2013). Serotype O outbreaks had place in Egypt in 2000 and 2006. These outbreaks never crossed international borders and were self-contained (WRLFMD, 2017). Since 1972, no further serotypes have been recorded (OIE, 2017). Clinical FMD instances were discovered in January 2006 on a cow farm in Ismailia. WRLFMD samples for laboratory received confirmation, where serotype Awas determined to be EGY/2006. It arrived in Egypt from East Africa most likely through the sea route trade of live cattle from Ethiopia (WRLFMD, 2017). More than 7,500 animals were affected by 34 FMD outbreaks in 8 areas as of April 6, 2006. The majority of clinical FMD cases (96.7%) were cattle, and 411 of them, mostly calves, reportedly perished (OIE, 2017). Another serotype A epidemic was reported in 2009. The discovered strain was closely linked to A/EGY/06 and disseminated in 2006 before evolving till 2009 (WRLFMD, 2017), indicating the persistence of the African serotype A in Egypt. Despite a widespread immunization program being carried out in outbreaks were reported 2012. FMD

throughout Egypt in 2012. According to epidemiological study and laboratory results, the pattern of outbreaks may have been caused by uncommon FMDV serotypes or strains. The existence of SAT2 and other serotypes was confirmed by the actions taken to clarify the issue, which were taken from outbreak samplesand mortality rate reached 500 daily (OIE, 2017). There was no herd immunity or prior vaccination attempt when SAT2 was firstly introduced to Egypt, so the death rate increased particularly among calves and on small farms. Older livestock suffered severe losses. Both cattle and buffalo herds are affected by FMD, while the effects on cattle are typically more severe. Among Egypt's 27 governorates, 25 had already found the disease.Regionally, Libya notified SAT2 outbreaks in 2009 and 2012 (OIE, 2017), but there haven't been any FMD cases in Algeria, Morocco, or Tunisia since the large outbreaks in 1999 (OIE, 2017; WRLFMD, 2017).

IV. <u>The 2014-2015 epidemics in North</u> <u>Africa:</u>

There were over 100 million heads of livestock in North Africa that were vulnerable to and at danger from FMD. The epidemiological condition of FMD in North Africa and the regional control efforts were not consistent (OIE, 2017). Report on the situation in 2014-2015,2 cows with clinical FMD symptoms were observed on April 24 in the Tunisian region of Nabeul (OIE, 2017). The national laboratory (IRVT) carried out real-time PCR and phylogenetic analysis, which were both verified by IZSLER. The topotype that was identified was O/ME/SA/Ind 2001d. This topotype shares 99% of its nucleotide sequences with isolated from Saudi viruses Arabia (SAU/3/2013) and Libya (LIB/2/2013), respectively (WRLFMD, 2017). According to an OIE assessment, the illegal importation of animals from Libya was the outbreak's

primary cause (OIE, 2017). In 11 districts in May 2014, 32 new domestic sheep, goat, and cattle outbreaks were reported. Further instances were reported in June in Jendouba areas, which are 50 kilometers from the country's Algerian border (OIE, 2017). A FMD epidemic was discovered on July 23, 2014, in the Setif province of eastern Algeria, nearby the Tunisian border. The first outbreak took place on a cattle farm, brought and it was about bv the unauthorized importation of animals from Tunisia. Fever, blisters, lameness, and breast lesions were some of the disease's clinical indicators (OIE, 2017). Italian laboratory IZSLER, in Brescia, received samples. The 2014 FMD outbreaks in Tunisia led to the isolation of the O/ME/SA/Ind/2001d virus, which has 99.69% nucleotide identity with O/TUN/1054/2014 and O/TUN/1031/2014 (WRLFMD, 2017). During the final week of April, outbreaks were reported in 6 districts. 35 new outbreaks were recorded in 13 new districts during the first week of May. By the end of August, 33 separate districts had reported more than 350 outbreaks. Following that, cases were reported in Oran areas, 160 km from the country's western border with Morocco. All of the instances that were reported were cattle, and small ruminants showed no clinical or serological symptoms of FMD. However, 12 sheeprelated FMD infections returned in El Bavadh and El Oued regions in March 2015. After five months, they were the first FMD cases to be detected in Algeria (OIE, 2017). Many outbreaks with serotypes A, Oand SAT2 were reported in Egypt in 2014 despite immunization efforts. Regarding Libya, the political climate made it difficult to find information about FMD (OIE, 2017; WRLFMD, 2017).

V. <u>The 2017 epidemics in North Africa:</u>

Outbreaks of FMD wasobserved in Algeria's East (Bordj Bou Arréridj district), West (Relizane district) and Center (Medea

district) as of the end of March 2017 in cattle (OIE, 2017). Sequencing analysis showedserotype A that was genetically related to strains that were prevalent in neighboring Libya (Asia/ Iran 05BAR 08) (WRLFMD, 2017). The infection's source was not known. However, the phylogenic study revealed that the Algerian serotypes shared 98.4% of their nucleotides with field strains discovered in Nigeria in 2015 lineage (African topotype G IV) (WRLFMD, 2017).

11.2. Occurrence of FMDV in West, Central and East Africa (from 2000 to 2014):

Mauritania, Cameroon, Mali, Niger, Benin, Cape Verde, Senegal, Ghana, Gambia, Guinea-Bissau, Sierra Leone, Cote d'Ivoire, Liberia, Burkina Faso, Nigeria, and Togo are the countries that make up West Africa. Equatorial Guinea, Gabon, the Central African Republic (CAR), Chad, and the DRC are all nations in central Africa (DRC). The nations of Eritrea, Burundi, Rwanda, Kenya, Sudan, South Sudan, Uganda, Djibouti, Ethiopia, Somalia, and Tanzania make up the East African countries. Six FMD serotypes (A, O, C, SAT 1-3) have been found in this area. Outbreak of SAT-3 that was stated in DRC (Sumption et al., 2007) in 2005 is not included since no virus genotype or affected species was identified.

Two (4 and 5) viral pools have been found in the East, West and Central Africa region (Paton et al., 2009). There is a significant co-occurrence of virus serotypes and topotypes in the two pools. As, the serotype and topotype viruses found in Ethiopia (2005-2008, and 2010–2012), Eritrea (2004 and 2011)and Sudan (2005, and 2008–2011) in East Africa were genetically related to pool 5 isolates of serotype O (EA-3) virus from Nigeria (2007 and 2009) and Niger (2007) in West Africa (WRLFMD, 2003). serotype A (G-IV

AFRICA topotype) was identified in cattle samples from Nigeria, Togoand Cameroon. Sequence analysis of the 1D coding region (WRLFMD, 2003; Bronsvoort et al., 2004b) revealed a close relationship between these isolates and the East African serotype A viruses from Sudan (2006 and 2011) and Eritrea (1998). Moreover, comparable isolates for the serotype SAT2 topotype VII were detected in the two pools in East and West African countries (WRLFMD, 2003; Bronsvoort et al., 2004b). According to Roeder and Knowles (2008), serotype C was last discovered from cattle in 2004 in Kenya. At the time, the isolate was believed to be a field reintroduction of the vaccine strain (Sangula et al., 2011).Modern serological tests show the existence of antibodies against serotype C (Rufael et al., 2008; Ayelet et al., 2009; Ayebazibwe et al., 2010b; Tekleghiorghis et al., 2014). To establish that serotype C is probably no longer circulating, more serological research must be conducted in this part of Africa utilizing particular diagnostic techniques.

SAT 2, A and O serotypes were widely circulated throughout the region, whereas SAT1 was only found in a small area in East Africa. In the DRC, Kenya, Tanzania, Ethiopia, Uganda, and Ethiopia, SAT-1 has been found. The first incidence of FMDV serotype SAT1 in Ethiopia was discovered in 2007 (Ayelet et al., 2009). Furthermore, isolates of serotypes SAT2 and SAT1 and were detected and genotyped in 2007 in Uganda (Ayebazibwe et al., 2010a). Although no information has been found on the isolation of SAT1 virus from domestic or wild animals in Central and West Africa in the previous 3 decades, WRLFMD revealsSAT1 to be one of the circulating serotypes. According to Ehizibolo et al. (2014), there is serological evidence of antibodies against SAT1 and SAT3 in sheep and cattle in Nigeria. Nevertheless, virological methods must be used to confirm this. Serotype SAT-3 has only been found in East Africa twice, in 1970 (Roeder et al., 1994) and 1997 (Bastos et al., 2003a; b; Kalema-Zikusoka et al., 2005). Serotype SAT3 was detected in Uganda using African buffaloes; however, no animals have ever been used in this region to isolate this serotype (Rweyemamu et al., 2008). Because ofinsufficient surveillance, it is likely that SAT-1 has either ceased to exist or is still present but unrecognized because it has not been isolated from wild and domestic animals for the previous 3 decadesin West African countries.

11.3. <u>Occurrence of FMDV in southern</u> <u>Africa</u>

Zimbabwe. Zambia. Angola, Sevchelles, Madagascar, South Africa, Mozambique Namibia, Malawi, Botswana, Swaziland, Comoros. Lesotho. and Mauritius, are among the nations that make up southern Africa. Strong evidence exists in southern Africa that the livestock-wildlife interface affects FMD dynamics in livestock populations, particularly for the transmission of the SAT (1-3) virus (Bastos et al., 2003b; Vosloo et al., 2005; Miguel et al., 2013). Since 1991, FMDV serotypes (O, SAT 1-3) have been discovered in this area, with SAT 1-3 serotypes being the most commonly found (Rweyemamu et al., 2008; Paton et al., 2009). Serotypes C and A were documented prior to 1991, however they appear to have vanished after then.

I. <u>Epidemics during the period from 2000</u> <u>to 2013:</u>

In southern Africa, serotype O is the least predominantserotype. Serotype O was also isolated at Mbala in Zambia's Northern Province. Serotype O was detected in the region bordering East and Central Africa and spread through the illegal trafficking of unwell animals, as in 2010 and 2012, for example (Sinkala et al., 2013). The serotype O EA-2 topotype of this virus showed the strongest correlation with viruses isolated from Uganda, DRC and Tanzania (OIE, 2005). Pigs and cattle were impacted by an outbreak of the serotype O (ME-SA PanAsia1 topotype) in 2000 in South Africa. The virus was discovered in raw sewage from a ship in Durban, where it was most likely brought from Asia (Sangare et al., 2001; Knowles et al., 2005). South Africa has reported no new cases of pan-Asian serotype O virus since 2000. To

SAT serotypes are kept by large herds of African buffalo in Africa and represent a major source of infection for domestic and other wildanimalswith cloven hooves, like impala, which can briefly contract the disease and spread it to susceptible animals (Hargreaves et al., 2004). In southern Africa, it has been shown that FMDV epidemics in domestic animals frequently arise as an overflow from the buffalo reservoir to cattle (Hargreaves et al., 2004; Miguel et al., 2013).In southern Africa, the latest SAT1 outbreaks were reported in Zambia in 2012, Botswana in 2013, South Africa in 2010 and 2013, and Namibia in 2011 and 2013, but the majority countries have previously of these discovered SAT1 in cattle, buffalo, and impala (Vosloo et al., 2006; 2007). Significant domestic cow epidemics caused by serotype SAT2 occurred in Zambia, Botswana, and Namibia in 2011 and 2012. In the same year, serotype SAT2 was observed in Mozambique and South Africa (OIE, 2005). SAT serotypes have a restricted range, with SAT3 occurring primarily in southern Africa and Uganda (Vosloo et al., 2002; Thompson et al., 2003). SAT3 has been detected in 2010 in Mozambique and in 2006, 2008, 2010, and 2011 in South Africa (WRLFMD, 2003).

II. <u>The epidemics in the period from 2014</u> <u>to 2018:</u>

The only outbreak identified during the study period occurred in 2016 on Mauritius; it was determined that FMDV serotype O, which genetically related toME-SA (Middle East South Asia topotype), was to blame. The disease was successfully managed by vaccination (Meenowa, 2020). It cannot be overstated how important it is to maintain strategic FMD vaccine banks and maintain high levels of emergency readiness given that the disease's invasion into this country shows that no country is immune to FMDV (Jamal and Belsham, 2013).

Thirty out of the 33 outbreaks that were reported were brought on by FMD viruses in Pool 6 (SAT1-3), while only three out of the 33 outbreaks were brought on by FMD viruses in Pool 4. Serotype SAT2 contributed to 20 of the outbreaks brought on by Pool 6 virus, SAT1 to six of the outbreaks brought on by the virus, and SAT3 to four of the outbreaks brought on by the virus. Limited sampling and sporadic reporting during outbreaks may have contributed to the issue of sampling bias, which causes virulent strains with greater transmission propensities to be sampled more frequently. The incorrect perception that the sampled strains are the ones that predominate in the field may result from this sampling bias.Sampling during low incidence periods could eliminate sample bias (Machira and Kitala, 2017).

SAT3 outbreaks had not been reported in southern African countries for years after its recent introduction in Mozambique and Zambia. The outbreak in Zambia appears to have originated in the western province's Shangombo district, where infected cases were first observed in2015. Then, clinical cases spread to the neighboring districts of Sikongo and Kalabo, and the spread of disease was stopped by a ring vaccination of 109, 211 herds of cattle (Sinkala, 2016). Shangombo's main affected neighborhood is bordered by Angola.The

disease originated from un controlled movement of infected cattle between neighboring countriesand lack of effective coordinated surveillance programs, based on the topotype of the virus isolate, topotype II (WZ) (Vosloo et al., 2002; Jori et al., 2009; Scoones et al., 2010; Sinkala et al., 2014). Owing to the low level of surveillance between these neighbors, little is known about the actual prevalence of FMD in either country, and there is no official data on the isolation of serotype SAT3 from Angola. In 2009, there was an FMD outbreak, but no virus could be found (Maree et al., 2014). The lack of FMD epidemiological data in this nation makes it difficult to effectively control FMD in southern Africa because successful vaccination depends on this data (Sangula et al., 2010).

12. <u>Risk factors involved in FMD</u> transmission in Africa:

I. Long distance transmission:

According to Mekibib and Arin (2016), airborne transmission is the term used to describe particles that are expired from an infected animal and evaporate into the surrounding air. These tinyparticles, which have a diameter of less than 5 m, have a lengthy infectious half-life and can be spread by air currents over great distances (greater than 1 m), possibly leading to long-distance transmission events (Kutter et al., 2018 and Siegel et al., 2021).

Gloster et al. (1982); (2003); Donaldson (1983) found that infectious aerosols released into the atmosphere have the potential to cause long-distance transmission events that represent a severe danger to the management of FMD epidemics. Transmission via airborne virus can spread quickly and result in sickness outside of the established quarantine zones (Klausner et al., 2015). Long-distance transmission of the virus requires several factors. For example, (i) High viral shedding and (ii) Prevailing climatic conditions, (iii)

High virus viability, such as R.H of at least 55%, and (iv) large numbers of carriers (Gloster et al., 1982).

When determining the likelihood of airborne dissemination, the stability of such strains in aerosolized FMDV is a crucial consideration. In a recent study, the durability of viral strains was examined in an experimental setting employing a Collison nebulizer to produce aerosols into a Goldberg drum and a Proton impinger sampler made entirely of glass to collect aerosol samples. Using this technique, Donaldson (1972) evaluated eight FMDV serotypes O, A, and C strains and discovered that when exposed to 55% and 70% R.H, serotype A viruses were more persistent in aerosols than the serotype O and C strains. In addition, Donaldson et al. (1970) demonstrated that the virus' ability to survive in aerosols declined as the humidity level rose. Similar findings were made by Barlow (1972), who used the same methods to demonstrate that a serotype O (O1 BFS 1860) had a higher survival rate at relative humidity levels above 50%. In addition, Barlow (1972) demonstrated that the virus's ability to survive diminished after aerosols were kept in the drum for five minutes as opposed to being tested right away. However, it has since been reported that sampling should wait until after a period of mixing when using rotating drums in order to account for deposition (Thompson et al., 2011; Fischer et al., 2016). When relative humidity exceeded 50%, Barlow and Donaldson (1973) stated that O1 BFS 1860 strain more stable in aerosols when suspended in bovine saliva, whereas Brown et al. (2021) demonstrated that in aerosols, FMD recovery was higher for serotype A than for serotypes O and Asia 1.

Research shows that FMDV from a contaminated environment can be resuspended into aerosols. Colenutt et al. (2018); (2020) shown that sick animals'

surroundings get contaminated in the wild, which could present a chance for virus aerosolization during animal and human movement or routine cleaning duties.

II. <u>Livestock management systems:</u>

In Africa, the availability of grazing and watering areas varies by region, which has a diverse effect on the system of animal husbandry. Robinson et al. (2011) identified five distinct livestock production systems in Africa based on the volume of animal movement: (i) Complete nomadism: absence of a fixed place of abode and regular agriculture, (ii) Semi-nomadic lifestyle: a permanent home and supplemental farming are conducted, but for extended periods of time, animal owners migrate to far-off grazing places, (iii) Transhumance: Herds are transported to far-off grazing sites, typically on seasonal cycles, despite having a permanent home, (iv) Partial nomadism: Farmers continuously reside in permanent communities and are in possession of animals that graze nearby.

Cattle, sheep, and goat husbandry are primarily sedentary occupations in southern Africa. Nonetheless, the risks of FMD spread have increased and will undoubtedly continue to rise in the future due to the establishment of game farming conservancies in cattle ranching areas and the creation of Trans frontier Conservation Areas (TFCAs). There may therefore be more contact between domestic animals and wildlife even when cattle are not moving (Osofsky et al., 2005).

III. <u>Livestock trade in Africa:</u>

The published and presently available livestock data show that over 780 million cattle, buffaloes, camels and other small ruminants were farmed throughout Africa. The main imported goods entering Morocco, Algeria, and Tunisia are cattle and small ruminants. Other dairy and meat products are imported from Australia, EU, Argentina, and New Zealand, and for certain imports, but not all, this database (<u>http://www.africalivestockdata.org/afrlivest</u> <u>ock/content/about-us</u>) gives information on the source of the animals.

Although such trading is expected to be significant throughout Africa, neither the FAO data nor the cross-border movements of cattle from nomadic and transhumance herds were included. In the north-west of Africa, for instance, the Sahara Desert separates it from sub-Saharan Africa, this making informal trade from southern countries into the north-west of Africa is constrained. In contrast, in the north-east of Africa, no information on formal trade from Sudan to Egypt and Libya is found, but informal trade is highly likely. Additionally, the FAO numbers do not include animal products that can be a transmission risk. North Africa imports more milk and beef than any other region on the continent, according to African Livestock Data (http://www.africalivestockdata.org/afrlivest ock/content/about-us).

Well-established livestock trade routes run through the dry and semi-arid sub-Saharan region of West, Central, and East Africa. In 2010, 2.9 million animals were imported into West, Central, and East Africa, with small ruminants making up two thirds of those and cattle making up the remaining third. With more than 1.4 million animals imported in 2010-including more than 1 million live cattle, sheep, and goats from Niger-Nigeria is the largest importer in this category. There aren't many trade figures available for the Greater Horn of Africa, which includes the countries of Ethiopia, Eritrea, Somalia, Sudan, and Djibouti.Mostly to the Arabian Peninsula and the Gulf States, sheep and goats are traded along these routes. Exports from East Africa to the Gulf States and the Arabian Peninsula primarily consist of small ruminants, cattle, and Arabian one-hump

camels. According to (http://www.africalivestockdata.org/afrlivest ock/content/about-us), Somalia, Sudan, and Ethiopia account for 61% of all sheep meat exports on the continent. Although though there is a low chance of FMD spreading within Africa as a result of exports, this trade nonetheless has a big impact on the continent because not all animals come from the countries that sell to the Arabian Peninsula and the Gulf States. In order to be shipped to the Arabian Peninsula and the Gulf States, they are assembled along trade routes and then transported by vehicle or foot to sea ports in the Red Sea and the Gulf of Aden. Many are imported from nearby African countries. Large cattle markets can be found in major East African cities like Nairobi and Mombasa in Kenya because to the high local demand, which facilitates trade throughout the area. Although Kenya technically lacks meat, it imports it from nations like South Sudan. Tanzania. Ethiopia, and Ethiopia to make up the shortfall (Aklilu, 2008). The spread of new FMD strains depends heavily on these animal movements (Fevre et al., 2006). For example, serotype SAT-2(SAU/6/00) isolates from Saudi Arabia in 2000 were genetically related to isolates from Eritrea in 1998 (ERI/1/98) (Bronsvoort et al., 2004), suggesting that Northeastern Region as Most Likely Source of Virus (Bastos et al., 2003b). Due to the trade ties between East, Central and West Africa. а more comprehensive regional approach to control should be taken.

According to FAO data, South Africa imports the most livestock in southern Africa—650 000 head. Furthermore, it is challenging to gather reliable data on actual cattle trade, and in many places, illegal trade is likely to outweigh legal trade (Scoones and Wolmer, 2006). The majority (94%) of Southern Africa's beef exports are beef from Botswana, South Africa, and Namibia, according to (http://www.africalivestockdata.org/afrlivest ock/content/about-us). The majority (57%) of Botswana's meat exports go to South Africa, and a sizeable amount (40%) travels to the European Union, according to FAO data (EU). The risk of transmissionof FMDV to countries without the disease could be decreased by importing animals and animal products from FMDVfree zones acknowledged by the OIE.

IV. <u>Involvement of African wildlife in</u> <u>FMD transmission</u>

It's possible that some wild animals help spread FMD. It has been shown that the African buffalo (Syncerus caffer) is a genuine maintenance host for serotype SAT (1-3) viruses and that it is constantly evolving (Vosloo and Thomson, 2004; Mwiine et al., 2010). Most healthy buffalo populations still harbor SAT viruses, which frequently cause subclinical illnesses (Vosloo et al., 2007). The virus can also be shown to persist in a single buffalo for at least five years and in small herds of freeliving animals for at least 24 years (Condy et al., 1985). According to theories about the SAT serotypes, the majority of buffalo sick during "childhood" calves get epidemics or while the mother's protective antibodies are decreasing (Vosloo and Thomson, 2004). The buffalo calves may infect susceptible cattle during this time (Jori et al., 2009).

How frequently diseased animals contribute to epidemics is still up for debate (Salt, 2004). Contrary to FMDV transmission from buffalo to cattle, which was viewed as an uncommon event (Dawe et al., 1994 and Thomson, 1995), SAT serotype transmission from carrier buffalo to cattle has been successfully confirmed under both experimental and natural conditions (Bastos et al., 2000). African buffalo in the Kruger National Park in South Africa have spontaneously transmitted FMDV to impala (Bastos et al., 2000). FMDV transmission from carrier male buffalo to female cattle was observed in one experiment, suggesting that the buffalo bull may have sexually transferred the illness to the cows (Bastos et al., 1999).

Some wild species that are briefly infected with the SAT serotypes can transmit the illness to susceptible animals (Thomson et al., 2003). Other wild species may have a role in the spread of serotype A and O, even if this hasn't been studied in Africa. Deer (Gibbs et al., 1975), impala (Aepyceros melampus), bush pig, warthog antelope, kudu (Hedger et al., 1972; Bengis et al., 1984; Bengis and Erasmus, 1988) and giraffe are other wild species of clovenhoofed mammals that are vulnerable to FMD (Vosloo et al., 2011). Contrary to farmed pigs, warthogs are not powerful amplifiers, despite virus being experimentally and clinically susceptible to FMD infection (Bengis, 2012). Although the FMDV SAT serotypes have not been shown to persist in any other wild species than buffalo (Bengis, 2012), transiently infected wild mammals with cloven hooves may aid in the spread of the virus. However, it is believed that in southern Africa, the primary contact necessary for the transmission of SAT FMDV serotypes is between cattle and buffalo or impala (Bastos et al., 2000; Vosloo et al., 2006). Ayebazibwe et al. (2010); Kalema-Zikusoka et al. (2005) both found evidence of serotype O and C infection in African buffalo from Kenva.

V. Social and economic development:

Socio-economic traits were underrepresented in FMD outbreak models despite the well-established association between low socio-economic position and contagious diseases (Wijayanti et al., 2016; Loi et al., 2019). Social aspects of animal health should be considered in models developed to understand and anticipate FMD epidemics (Card et al., 2018). The findings of recent studies that suggest that the socioeconomic realities of daily life in communities affected by FMD may be linked to an increase in FMD outbreaks (Guerrini et al., 2019) and that in many cases the same communities experience difficulties adhering to the numerous restrictions put in place as a result of the detection of the outbreaks can be supported by this evidence (Limon et al., 2020; MacPhillamy et al., 2022). It would be possible to evaluate socio-economic factors' importance and, if there is a connection, to come up with ideas for a comprehensive FMD control strategy that does not just rely on the conventional methods (such as market closures, movement controls, and vaccination) (Loi et al., 2019; Wajid et al., 2020). This would be made possible by incorporating socio-economic factors into FMD spatio-temporal modelling.

VI. <u>Ecology and environment:</u>

Climatic and environmental factors may have an impact on FMD's viral stability, survival, or transmission, which may have an impact on risk. For example, the risk of airborne transmission, which is a longdistanceroute for FMD spread, varies across geographic regions due to differences in animal husbandry, proximity of susceptible and infected species, numbers of animals, and local climate (Brown et al., 2022). On the other hand, weather-related factors may disclose the increased contact between animals produced by extreme climatic occurrences like drought, a factor that produces movements and natural migrations in animals.Cool temperatures, a neutral Ph, and a relative humidity of at least 55% are necessary for stability of FMDV and permit the prolonged survival of virus particles in the environment (Mielke and Garabed, 2020). The virus is therefore expected to survive the wet season longer than the dry

season since the favorable damp and cool temperature conditions delay the virus's desiccation (Mielke and Garabed, 2020). It is possible that regions with FMDV friendly conditions would be better for preserving the illness and more likely to see outbreaks. Considering this. meteorological and microclimatic variables such as relative humidity, temperature, windspeed, and precipitationmay represent a number of processes linked to direct and indirect FMDV transmission routes (Gordon et al., 2022).

VII.<u>Effect of water availability on annual</u> <u>variation of FMD outbreaks:</u>

Decreased water accessibility in dry conditions is likely to increase contact with animals in the few remaining water sources, resulting in the spread of FMD between animal houses. This is why water accessibility was taken into consideration as a potential risk factor. Although data on water availability were lacking, rainfall over a year at the end of the rainy season was used as a proxy for the amount ofreplacement of water reservesand was believed to have the potential to influence the frequency of FMD outbreaks for 12 months (Guerrini et al., 2019).

VIII.<u>Season:</u>

In addition to environmental factors (like temperature and rainfall) that can have an impact on FMD epidemiology, seasons also govern the calendars for cropping and herding. The majority of the peak FMD outbreaks were observed in the second half of each year that was recorded. Variations in animal mobility may be related to seasonal changes in FMD epidemics. This might be connected to the increased animal transports brought on by the rise in meat consumption during the Amhara area's Christian Easter celebrations (March–April), one of the two major Orthodox Christian religious holidays in Ethiopia generally and in the Amhara region particularly. The increase in demand for meat for the impending Muslim holidays of Milad-Un-Nabi and Ramadan (Gunasekera et al., 2017; Aman et al., 2020).

13. <u>Control and vaccination strategy:</u>

Effective FMD control and prevention requires a multiple control strategy including physical isolation of wildanimals and livestock by using gameproof fences, repeated vaccination of susceptible herds, restriction of animal movements, and careful risk assessment of FMDV introduction into FMD-free areas (Bruckner et al., 2002; Jori et al., 2009). These prevention measures, however very expensive, have been implemented in southern Africa and have significantly decreased the disease's prevalence there. Southern Africa uses inactivated whole particle FMD vaccines adjuvanted with saponin and aluminum hydroxide gel to control FMD through vaccination. Several manufacturers of these vaccinations assert that each dosage has 3PD50 homologous potency. Yet, there is little publicly available information on the efficacy of African FMD vaccinations, which restricts debates of their effectiveness. Codon fences have been used to delineate disease zones in a few nations in the region, including Botswana and Zimbabwe, in order to conduct effective FMD control strategies (Derah and Mokopasetso, 2005). "Green Zones" have been designated as regions outside of game conservancies, and animals there are free of FMD without the need for vaccination. "Red zones" are locations that are adjacent to game conservancies and are thought to be FMD-infectious. These areas routinely administer trivalent vaccines including the SAT1. 2. and to cattle. 3 strains Strategically, the bivalent FMD vaccine has been used primarily in southern African countries (SAT1 and SAT2). Trivalent FMD vaccination use has only been reported in

Botswana and Republic of South Africa (SAT1, SAT2, and SAT3). These vaccines are offered in two purification levels: semi purified and highly purified. The latter level, known as DIVA or marker vaccines, enables the distinction between infection- and vaccine-induced antibodies (Fana et al., 2021).

Studies that matched vaccines over this time (2014-2018) produced r1- values greater than or equal to 0.3. These findings demonstrate the vaccinations' ability to provide protection against exposure to FMD viruses in the field (Rweyemamu et al., 1978). These findings coincide with those of the phylogenetic analysis. The VP1 sequences from epidemic strains were grouped with other vaccination strains used in the area and in their respective serotypes, indicating a tight evolutionary link. The results are consistent with earlier studies done in the area and show that some outbreaks clustered with sequences from buffalo, demonstrating the role this species plays in maintaining FMD outbreaks in the area (Vosloo et al., 2005). In phylogenetic trees, the grouping of individuals shows a close relationship in ancestry. Nonetheless, great care must be taken when extrapolating nucleotide homology between and antigenicity because there is insufficient data on the impact of amino acid variations on antigenicity (Paton et al., 2005).

Some nations revised their vaccination plans in response to the reappearance of FMDV serotypesOand SAT3in southern Africa; now, Zambia has toimmunize against 3 serotypes in Pool 6 as well as serotype O from Pool 4. In order to manage FMD, Namibia and Mozambique now use utilize a trivalent vaccine (SAT1-3) rather than a bivalent vaccine (SAT 1-2). Zambia now has a heavier burden of FMD management through vaccination, and it's possible that they won't be able to stop Pool 4's exotic virus outbreak from spreading to

Zimbabwe, Botswana, and Namibia.In order to effectively control and prevent FMD caused by exotic viruses moving into the region from eastern Africa, it is recommended that coordinated measures led by SADC and supported by international donor organizations like the World Bank, FAO, and/or EU be begun as soon as possible (Fana et al., 2021).

Throughout other parts of the African continent, vaccination strategies against FMD are not frequently carried out. Because the antibody decay following vaccination is about 6 months, many especially southern countries. vaccinate animals Africacountries, in response to occurrence of outbreaks rather than doing regular preventative vaccinations (Parida, 2009; Dekker et al., 2016). The majority of the continent's nations are underdeveloped. lacking in competent human resources, good veterinary infrastructure, and the ability to regulate the movement of animals. Thus, they are unable to afford the traditional FMD vaccines available today. Due to these restrictions, several African nations are now vulnerable to the development of FMD (Doel, 2003; Sutmoller et al., 2003; Knight-Jones and Rushton, 2013). Despite these limitations, in order to get the optimum effects with these vaccines, it is crucial that governments in African countries continent follow the recommendations for the preventive immunization of significant susceptible hosts, particularly cattle. Future research should focus on developing FMD vaccines that are more affordable than the ones now in use. These vaccines may persuade more nations implement regular African to preventive immunizationprograms to curb and prevent the spread of FMD. In addition to facilitating regional and global trade of these routine animals, immunization campaigns will stop disease outbreaks in susceptible animals, eradicating poverty and ensuring prosperity and food security (Fana et al., 2021).

REFERENCES

- Aidaros, H. A. (2002). Regional status and approaches to control and eradication of foot and mouth disease in the Middle East and North Africa. *Revue Scientifique et Technique* (*International Office of Epizootics*), 21(3), 451-458.
- Aiewsakun, P., Pamornchainavakul, N., &Inchaisri, C. (2020). Early origin and global colonisation of foot-andmouth disease virus. *Scientific reports*, 10(1), 1-9.
- Aklilu, Y. (2008): Livestock marketing in Kenya and Ethiopia: a review of policies and practice. Pastoral Areas Coordination, Analysis and Policy Support (PACAPS). Project of the Feinstein International Centre, Tufts University, funded by USAID East Africa, Addis Ababa.
- Alexandersen, S., & Mowat, N. (2005). Foot-and-mouth disease: host range and pathogenesis. *Foot-and-mouth disease virus*, 288: 9-42.
- Alexandersen, S., Zhang, Z., Donaldson, A. I., & Garland, A. J. M. (2003). The pathogenesis and diagnosis of footand-mouth disease. *Journal of comparative pathology*, *129*(1), 1-36.
- Aman, E., Molla, W., Gebreegizabher, Z., &Jemberu, W. T. (2020). Spatial and temporal distribution of foot and mouth disease outbreaks in Amhara region of Ethiopia in the period 1999 to 2016. *BMC veterinary research*, 16(1), 1-8.
- Andino, R., & Domingo, E. (2015). Viral quasispecies. *Virology*, 479, 46-51.
- Armstrong, R., Davie, J., & Hedger, R. S. (1967). Foot-and-mouth disease in man. British Medical Journal, 4(5578), 529.

- Arzt, J., Baxt, B., Grubman, M. J., Jackson, T., Juleff, N., Rhyan, J., ... & Rodriguez, L. L. (2011a). The pathogenesis of foot-and-mouth disease II: viral pathways in swine, ruminants, and wildlife; small myotropism, chronic syndromes, and molecular virus-host interactions. Transboundary and emerging diseases, 58(4), 305-326.
- Arzt, J., Juleff, N., Zhang, Z., & Rodriguez, L. L. (2011b). The pathogenesis of foot-and-mouth disease I: viral pathways in cattle. *Transboundary* and emerging diseases, 58(4), 291-304.
- Arzt, J., Fish, I., Pauszek, S. J., Johnson, S. L., Chain, P. S., Rai, D. K., ... &Stenfeldt, C. (2019). The evolution of a super-swarm of foot-and-mouth disease virus in cattle. *PLoS One*, 14(4), e0210847.
- Auty, H., Mellor, D., Gunn, G., & Boden, L.
 A. (2019). The risk of Foot and Mouth Disease transmission posed by public access to the countryside during an outbreak. *Frontiers in Veterinary Science*, 6, 381.
- Ayebazibwe, C., Mwiine, F. N., Balinda, S. N., Tjørnehøj, K., Masembe, C., Muwanika, V. B., ... &Alexandersen, S. (2010a). Antibodies against foot-and-mouth disease (FMD) virus in African buffalos (*Synceruscaffer*) in selected national parks in Uganda (2001–2003). *Transboundary and emerging diseases*, 57(4), 286-292.
- Ayebazibwe, C., Mwiine, F. N., Tjørnehøj, K., Balinda, S. N., Muwanika, V. B., AdemunOkurut, A. R., ... &Alexandersen, S. (2010b). The role of African buffalos (Synceruscaffer) in the maintenance of foot-andmouth disease in Uganda. *BMC veterinary research*, 6(1), 1-13.

- Ayelet, G., Mahapatra, M., Gelaye, E., Egziabher, B. G., Rufeal, T., Sahle, M., ... & Knowles, N. J. (2009).
 Genetic characterization of foot-andmouth disease viruses, Ethiopia, 1981–2007. *Emerging infectious diseases*, 15(9), 1409.
- Bari, F. D., Parida, S., Tekleghiorghis, T., Dekker, A., Sangula, A., Reeve, R., ... & Mahapatra, M. (2014). Genetic and antigenic characterization of serotype A FMD viruses from East Africa to select new vaccine strains. Vaccine, 32(44), 5794-5800.
- Barlow, D. F. (1972). The effects of various protecting agents on the inactivation of foot-and-mouth disease virus in aerosols and during freezedrying. *Journal of General Virology*, *17*(3), 281-288.
- Barlow, D. F., & Donaldson, A. I. (1973). Comparison of the aerosol stabilities of foot-and-mouth disease virus suspended in cell culture fluid or natural fluids. *Journal of general Virology*, 20(3), 311-318.
- Bartley, L. M., Donnelly, C. A., & Anderson, R. M. (2002). Review of foot-and mouth disease virus survival in animal excretions and on fomites. *Veterinary record*, 151(22), 667-669.
- Bastos, A. D., Bertschinger, H. J., Cordel, C., Van Vuuren, C. D., Keet, D., Bengis, R. G., ... & Thomson, G. R. (1999). Possibility of sexual transmission of foot-and-mouth disease from African buffalo to cattle. *Journal ofPediatric Surgery*, 19, 205.
- Bastos, A. D. S., Boshoff, C. I., Keet, D. F., Bengis, R. G., & Thomson, G. R. (2000). Natural transmission of footand-mouth disease virus between African buffalo (Synceruscaffer) and impala (Aepyceros melampus) in the

Kruger National Park, South Africa. *Epidemiology* & *Infection*, 124(3), 591-598.

- Bastos, A. D., Anderson, E. C., Bengis, R. G., Keet, D. F., Winterbach, H. K., & Thomson, G. R. (2003a). Molecular epidemiology of SAT3-type foot-and-mouth disease. *Virus genes*, 27(3), 283-290.
- Bastos, A. D. S., Haydon, D. T., Sangare,
 O., Boshoff, C. I., Edrich, J. L., &
 Thomson, G. R. (2003b). The
 implications of virus diversity within
 the SAT 2 serotype for control of
 foot-and-mouth disease in subSaharan Africa. *Journal of General Virology*, 84(6), 1595-1606.
- Bauer, K. (1997). Foot-and-mouth disease as zoonosis. Viral Zoonoses and Food of Animal Origin, 95-97.
- Belsham, G. J., & Martinez-Salas, E. (2019). Genome organisation, translation and replication of foot-and-mouth disease virus RNA. *Foot and Mouth Disease*, 19-52.
- Bengis, R. G. (2012). Some epidemiological aspects of foot-and-mouth disease in wildlife in sub-Saharan Africa. *Global Foot-and Mouth Disease Research Alliance* (*GFRA*), Hazyview, South Africa.
- Bengis, R. G., & Erasmus, J. M. (1988). Wildlife diseases in South Africa: a review. *Revue Scientifique et Technique de l'OIE*.
- Bengis, R. G., R. S. Hedger, V. de Vos, and L. Hurter, (1984): The role of the elephant (Loxodonta African africana) in the epidemiology of foot-and-mouth disease in the Kruger National Park. In: Proceedings of the 13th World Congress of Diseases in Cattle. World **Buiatrics** Association. World Congress of Diseases in Cattle, pp. 39–44.

- Berryman, S., Clark, S., Monaghan, P., & Jackson, T. (2005). Early events in integrin $\alpha\nu\beta6$ -mediated cell entry of foot-and-mouth disease virus. *Journal of virology*, 79(13), 8519-8534.
- Blacksell, S. D., Siengsanan-Lamont, J., Kamolsiripichaiporn, S., Gleeson, L.
 J., & Windsor, P. A. (2019). A history of FMD research and control programmes in Southeast Asia: lessons from the past informing the future. *Epidemiology* & *Infection*, 147.
- Bøtner, A., &Belsham, G. J. (2012). Virus survival in slurry: analysis of the stability of foot-and-mouth disease, classical swine fever, bovine viral diarrhoea and swine influenza viruses. *Veterinary*

microbiology, 157(1-2), 41-49.

- Breithaupt, A., Depner, K., Haas, В., Alexandrov, T., Polihronova, L., Georgiev, G., ... & Beer, M. (2012). Experimental infection of wild boar and domestic pigs with a foot and mouth disease virus strain detected in the southeast Bulgaria of in 2010. Veterinary December of microbiology, 159(1-2), 33-39.
- Brito, B. P., Rodriguez, L. L., Hammond, J. M., Pinto, J., & Perez, A. M. (2017).
 Review of the global distribution of foot-and-mouth disease virus from 2007 to 2014. *Transboundary and emerging diseases*, 64(2), 316-332.
- Bronsvoort, B. D. C., Radford, A. D., Tanya, V. N., Nfon, C., Kitching, R. P., & Morgan, K. L. (2004). Molecular epidemiology of foot-andviruses mouth disease in the Adamawa province of Cameroon. Journal of Clinical Microbiology, 42(5), 2186-2196.
- Bronsvoort, B. M. D. C., Parida, S., Handel, I., McFarland, S., Fleming, L.,

Hamblin, P., & Kock, R. (2008). Serological survey for foot-andmouth disease virus in wildlife in eastern Africa and estimation of test parameters of a nonstructural protein enzyme-linked immunosorbent assay for buffalo. *Clinical and Vaccine Immunology*, 15(6), 1003-1011.

- E., Nelson, N., Gubbins, Brown, S.. &Colenutt, C. (2021). Environmental and air sampling are efficient detection and methods for the quantification of foot-and-mouth disease virus. Journal of virological methods, 287, 113988.
- Brown, E., Nelson, N., Gubbins, S., & Colenutt, C. (2022). Airborne transmission of foot-and-mouth disease virus: A review of past and present

perspectives. Viruses, 14(5), 1–14.

- Bruckner, G. K., Vosloo, W., Plessis, B. J.
 A. D., Kloeck, P. E. L. G., Connoway, L., Ekron, M. D., ... & Marais, T. (2002). Foot and mouth disease: the experience of South Africa. *Revue scientifique et technique-Office international des épizooties*, 21(3), 751-761.
- Card, C., Epp, T., & Lem, M. (2018). Exploring the social determinants of animal health. Journal of Veterinary Medical Education, 45(4), 437–447.
- Casey-Bryars, M., Reeve, R., Bastola, U., Knowles, N. J., Auty, H., Bachanek-Bankowska, K., ... &Lembo, T. (2018). Waves of endemic foot-andmouth disease in eastern Africa suggest feasibility of proactive vaccination approaches. *Nature ecology & evolution*, 2(9), 1449-1457.
- Charleston, B., Bankowski, B. M., Gubbins, S., Chase-Topping, M. E., Schley, D., Howey, R., ... &Woolhouse, M.

E. (2011). Relationship between clinical signs and transmission of an infectious disease and the implications for control. *Science*, *332*(6030), 726-729.

- Chin, J. (2000). Control of communicable diseases manual. 17th ed. Washington, DC: American Public Health Association. *Coxsackievirus diseases*; pp. 129–131
- Colenutt, C., Brown, E., Nelson, N., Wadsworth, J., Maud, J., Adhikari, B., ... &Gubbins, S. (2018). Environmental sampling as a lowtechnology method for surveillance of foot-and-mouth disease virus in an area of endemicity. *Applied and environmental microbiology*, 84(16), e00686-18.
- Colenutt, C., Brown, E., Nelson, N., Paton, D. J., Eblé, P., Dekker, A., ... &Gubbins, S. (2020). Quantifying the transmission of foot-and-mouth disease virus in cattle via a contaminated environment. *MBio*, 11(4), e00381-20.
- Condy, J. B., Hedger, R. S., Hamblin, C., & Barnett, I. T. R. (1985). The duration of the foot-and-mouth disease virus carrier state in African buffalo (i) in the individual animal and (ii) in a free-living herd. *Comparative immunology, microbiology and infectious diseases,* 8(3-4), 259-265.
- Cottral, G. E. (1969). Persistence of footand-mouth disease virus in animals, their products and the environment. *Bulletin-Office international des épizooties*, 71(3-4), 549-568.
- Croft, S., Aegerter, J. N., Massei, G., & Smith, G. C. (2019). The risk of footand-mouth disease becoming endemic in a wildlife host is driven

by spatial extent rather than density. *Plos one*, *14*(6), e0218898.

- Dawe, P. S., Flanagan, F. O., Madekurozwa, R. L., Sorensen, K. J., Anderson, E. C., Foggin, C. M., ... & Knowles, N. J. (1994). Natural transmission of foot-and-mouth disease virus from African buffalo (Synceruscaffer) to cattle in a wildlife area of Zimbabwe. *The Veterinary Record*, 134(10), 230-232.
- Dekker, A., Chénard, G., Stockhofe, N., &Eblé, P. L. (2016). Proper timing of foot-and-mouth disease vaccination of piglets with maternally derived antibodies will maximize expected protection levels. Frontiers Veterinary in Science, 3, 52.
- Derah, N., &Mokopasetso, M. (2005). The control of foot and mouth disease in Botswana and Zimbabwe. *Tropicultura*, 2005(23), 3-7.
- Dhollander, S., Belsham, G. J., Lange, M., Willgert, K., Alexandrov, Т., Chondrokouki, E., ... &Bøtner, A. Assessing the potential (2016).spread and maintenance of foot-and-mouth disease virus infection in wild ungulates: general principles and application to a specific scenario in Т hrace. Transboundary and emerging diseases, 63(2), 165-174.
- Di Nardo, A., Knowles, N. J. and Paton, D. J. (2011): Combining livestock trade patterns with phylogenetics to help understand the spread of foot and mouth disease in sub-Saharan Africa, the Middle East and Southeast Asia. *Rev. Sci. Tech.* 30, 63–85.
- Doel, T. R. (2003). FMD vaccines. Virus research, 91(1), 81-99.
- Donaldson, A. I. (1972). The influence of relative humidity on the aerosol

stability of different strains of footand-mouth disease virus suspended in saliva. *Journal of General Virology*, *15*(1), 25-33.

- Donaldson, A. I. (1983). Quantitative data on airborne foot-and-mouth disease virus: its production, carriage and deposition. *Philosophical Transactions of the Royal Society of London. B*, *Biological Sciences*, 302(1111), 529-534.
- Donaldson, A. I., Herniman, K. A. J., Parker, J., & Sellers, R. F. (1970). Further investigations on the airborne excretion of foot-and-mouth disease virus. *Epidemiology & Infection*, 68(4), 557-564.
- Dos Santos, D. V., Silva, G. S. E., Weber, E. J., Hasenack, H., Groff, F. H. S., Todeschini, B., Borba, M. R., Medeiros, A. A. R., Leotti, V. B., Canal, C. W., Corbellini, L. G., Dos Santos, D. V., Sousa e Silva, G., Todeschini, B., Borba, M. R., Corbellini, L. G., Weber, E. J., Hasenack, H., Groff, F. H. S., ... Corbellini, L. G. (2017). Identification of foot and mouth disease risk areas using a multi-criteria analysis approach. *PLoS* One, 12(5), e0178464.
- Ehizibolo, D. O., Perez, A. M., Carrillo, C., Pauszek, S., AlKhamis, M., Ajogi, I.,Umoh, J. U., Kazeem, H. M., Ehizibolo, P. O., Fabian, A., Berninger, M., Moran, K., Rodriguez, L. L. and Metwally, S. A. (2014): Epidemiological analysis, serological prevalence and genotypic analysis of foot-and-mouth disease in Nigeria 2008–2009. *Transbound. Emerg. Dis.* 63, 500– 510.
- Elbers, A. R. W., Dekkers, A., & Dekker, L. J. M. (2003).Serosurveillance of wild deer and wild boar after the epidemic

of foot-and-mouth disease in the Netherlands in 2001. *Veterinary Record*, *153*(22), 678-681.

- Fana, E. M., Mpoloka, S. W., Leteane, M., Seoke, L., Masoba, K., Mokopasetso, M., ... &Hyera, J. (2021). A Five-Year Retrospective Study of Footand-Mouth Disease Outbreaks in Southern Africa, 2014 to 2018. Veterinary Medicine International, 2021.
- FAO, OIE, GF-TADs, & EU-FMD. (2018). The progressive control pathway for foot and mouth disease control(PCP-FMD).
- Ferretti, L., Di Nardo, A., Singer, B., Lasecka-Dykes, L., Logan, G., Wright, C. F., ... &Ribeca, P. (2018). Within-host recombination in the foot-and-mouth disease virus genome. *Viruses*, 10(5), 221.
- Fevre, E. M., Bronsvoort, B. M., Hamilton, K. A. and Cleaveland, S. A. (2006): Animal movements and the spread of infectious diseases. *Trends Microbiol.*, 14, 125–131.
- Fischer, R. J., Bushmaker, T., Judson, S., & Munster, V. J. (2016). Comparison of the aerosol stability of 2 strains of Zaire ebolavirus from the 1976 and 2013 outbreaks. *The Journal of Infectious Diseases*, 214(suppl_3), S290-S293.
- Food and Agricultural Organization (FAO)(1999). The 1999 Session of the Research group of the standing technical committee of EuFMD. <u>http://www.fao.org/ag/againfo/comm</u> issions/eufmd/commissions/ eufmd-<u>home/reports/archive/63rd-session-</u> of-theexecutive-committee/sessionof-the-research-groupof-thestanding-technical-committee-of-theeufmdheld-at-maisons-alfort-france-29-september-to-1october-1999/en/.

- Gibbens, J. C., Wilesmith, J. W., Sharpe, C.
 E., Mansley, L. M., Michalopoulou,
 E., Ryan, J. B. M., & Hudson, M.
 (2001). Descriptive epidemiology of the 2001 foot-and-mouth disease epidemic in Great Britain: the first five months. *Veterinary Record*, 149(24), 729-743.
- Gibbs, E. P., Herniman, K. A., Lawman, M. J., & Sellers, R. F. (1975). Foot-andmouth disease in British deer: transmission of virus to cattle, sheep and deer. *The Veterinary Record*, 96(26), 558-563.
- Gloster, J., Sellers, R. F., & Donaldson, A. I. (1982). Long distance transport of foot-and-mouth disease virus over the sea. *The Veterinary Record*, *110*(3), 47-52.
- Gloster, J., Champion, H. J., Sørensen, J. H., Mikkelsen, T., Ryall, D. B., Astrup, P., ... & Donaldson, A. I. (2003). Airborne transmission of foot-and-mouth disease virus from Burnside Farm, Heddonon-the-Wall, Northumberland, during the 2001 epidemic in the United Kingdom. *Veterinary Record*, 152(17), 525-533.
- Gordon, L. G., Porphyre, T., Muhanguzi, D., Muwonge, A., Boden, L., & de C Bronsvoort, B. M. (2022). A scoping review of foot-and-mouth disease risk, based on spatial and spatio-temporal analysis of outbreaks in endemic settings. *Transboundary and Emerging Diseases*.
- Grubman, M. J., & Baxt, B. (2004). Footand-mouth disease. *Clinical microbiology reviews*, 17(2), 465-493.
- Guerrini, L., Pfukenyi, D. M., Etter, E., Bouyer, J., Njagu, C., Ndhlovu, F., Bourgarel, M., de Garine-Wichatitsky, M., Foggin, C., Grosbois, V., & Caron,

A. (2019). Spatial and seasonal patterns of FMD primary outbreaks in cattle in Zimbabwe between 1931 and 2016. *Veterinary Research*, 50(1), 73.

- C., Sivasothy, Gunasekera, U. A., Wedasingha, N., Thayaparan, S., Rotewewa, B., Muralithas, M., ... &Punyapornwithaya, V. (2017).Analyzing the Foot and Mouth Disease outbreak as from 2008 to 2014 in cattle and buffaloes in Sri Lanka. *Preventive* veterinary medicine, 148, 78-88.
- Guyver-Fletcher, G., Gorsich, E. E., &Tildesley, M. J. (2022). A model exploration of carrier and movement transmission as potential explanatory causes for the persistence of foot-and-mouth disease in endemic regions. *Transboundary* and *Emerging Diseases*, 69(5), 2712-2726.
- Hall, M. D., Knowles, N. J., Wadsworth, J., Rambaut, A. andWoolhouse, M. E. (2013): Reconstructing geographical movements and host species transitions of foot-and-mouth disease virus serotype SAT 2. *mBio* 4, 1–10.
- Hammond, J. M., King, D., Knowles, N., Mioulet, V., & Li, Y. (2012, June).
 Analysis of the worldwide foot and mouth disease situation, trends and regional differences. In *Proceedings* of the FAO/OIE Global Conference on Foot and Mouth Disease Control, Bangkok, Thailand (pp. 27-29).
- Harada, Y., Lekcharoensuk, P., Furuta, T., & Taniguchi, T. (2015). Inactivation of foot-and-mouth disease virus by commercially available disinfectants and cleaners. *Biocontrol science*, 20(3), 205-208.
- Hargreaves, S. K., Foggin, C. M., Anderson, E. C., Bastos, A. D., Thomson, G. R., Ferris, N. P. and

Knowles, N. J. (2004): An investigation into the source and spread of foot and mouth disease virus from a wildlife conservancy in Zimbabwe. *Rev. Sci. Tech.* 23, 783–790.

- Hedger, R. S., Condy, J. B. and Golding, S. M. (1972): Infection of some species of African wild life with foot-andmouth disease virus. J. Comp. Pathol. 82, 455–461.
- Hertwig, C. A. (1834).bertragungtierischerAnstecku ngsstoffe auf den Menschen. *Med Vet Z, 48.*
- Hunter, P (1998). Vaccination as a means of control of foot-and-mouth disease in sub-saharan Africa, Vaccine, 16, 261-264.
- Hussein, H. A., El Nashar, R. M., El-Sherbiny, I. M., & Hassan, R. Y. (2021). High selectivity detection of FMDV-SAT-2 using a newlydeveloped electrochemical nanosensors. *Biosensors and Bioelectronics*, 191, 113435.
- Jamal, S. M., &Belsham, G. J. (2013). Footand-mouth disease: past, present and future. *Veterinary research*, *44*, 1-14.
- Jamal, S. M., Ferrari, G., Ahmed, S., Normann, P., Curry, S., &Belsham, G. J. (2011). Evolutionary analysis foot-and-mouth of serotype Α disease viruses circulating in Pakistan and Afghanistan during General 2002–2009. Journal of Virology, 92(12), 2849-2864. doi:10.1099/vir.0.035626-0.
- Jori, F., W. Vosloo, B. Du Plessis, R. G. Bengis, D. Brahmbhatt, B. Gummow, and G. R. Thomson, (2009): A qualitative risk assessment of factors contributing to foot and mouth disease outbreaks in cattle along the western boundary of

the Kruger National Park. *Rev. Sci. Tech.* 28, 917–931.

- Kalema-Zikusoka, G., Bengis, R. G. Michel,
 A. L. and Woodford, M. H.
 (2005): A preliminary investigation of tuberculosis and other diseases in African buffalo (Synceruscaffer) in Queen Elizabeth National Park, Uganda. *Onderstepoort J. Vet. Res.* 72, 145–151.
- Kitching, R. P. (2002a). Clinical variation in foot and mouth disease: Cattle. OIE Revue Scientifique et Technique, 21(3), 499–504.
- Kitching, R. P. (2002b). Clinical variation in foot and mouth disease: Pigs. OIE Revue Scientifique et Technique, 21(3), 513–518.
- Kitching, R. P. (2002c). Clinical variation in foot and mouth disease: Sheep and goats. OIE Revue Scientifique et Technique, 21(3), 505–512.
- Klausner, Z., Klement, E., &Fattal, E. (2015). Modeling long distance dispersal of airborne foot-and-mouth disease virus as a polydisperse aerosol–Application to the emergence of a new strain from Egypt to Israel. *Atmospheric Environment*, 122, 332-342.
- Knight-Jones, T. J., & Rushton, J. (2013). The economic impacts of foot and mouth disease–What are they, how big are they and where do they occur?. *Preventive veterinary medicine*, *112*(3-4), 161-173.
- Knowles, N. J., & Samuel, A. R. (2003). Molecular epidemiology of foot-andmouth disease virus. *Virus research*, 91(1), 65-80.
- Knowles, N. J., A. R. Samuel, P. R. Davies, R. J. Midgley, and J. F. Valarcher, (2005): Pandemic strain of foot-and-mouth disease virus serotype O. *Emerg.* Infect. Dis. 11, 1887–1893.

- Knowles, N. J., J. Wadsworth, S. M. Reid, K. G. Swabey, A. A. El-Kholy, A. O. A. El-Rahman, H. M. Soliman, K. Ebert, N. P. Ferris, G. H. Hutchings, R. J. Statham, D. P. King, and D. J. Paton, (2007): Foot-andmouth disease virus serotype A in Egypt. *Emerg. Infect. Dis.* 13, 1593– 1596.
- Knowles, N. J., Nazem Shirazi, M. H., Wadsworth, J., Swabey, K. G., Stirling, J. M., Statham, R. J., ... & Paton, D. J. (2009). Recent spread of a new strain (A-Iran-05) of foot-and-mouth disease virus type A in the Middle East. *Transboundary and emerging diseases*, 56(5), 157-169.
- Krug, P. W., Larson, C. R., Eslami, A. C., & Rodriguez, L. L. (2012). Disinfection of foot-and-mouth disease and African swine fever viruses with citric acid and sodium hypochlorite on birch wood carriers. *Veterinary microbiology*, 156(1-2), 96-101.
- Krug, P. W., Davis, T., O'Brien, C., LaRocco, M., & Rodriguez, L. L. (2018). Disinfection of transboundary animal disease viruses on surfaces used in pork packing plants. *Veterinary Microbiology*, 219, 219-225.
- Kutter, J. S., Spronken, M. I., Fraaij, P. L., Fouchier, R. A., &Herfst, S. (2018). Transmission routes of respiratory viruses among humans. *Current* opinion in virology, 28, 142-151.
- Limon, G., Ulziibat, G., Sandag, B., Dorj, S., Purevtseren, D., Khishgee, B., Basan, G., Bandi, T., Ruuragch, S., Bruce, M., Rushton, J., Beard, P. M., & Lyons, N. A. (2020). Socioeconomic impact of foot-and-mouth disease outbreaks and control measures: An analysis of Mongolian outbreaks in 2017. Transboundary

and Emerging Diseases, 67, 2034–2049.

- Loi, F., Laddomada, A., Coccollone, A., Marrocu, E., Piseddu, T., Masala, G., Bandino, E., Cappai, S., & Rolesu, S. (2019). Socio-economic factors as indicators for various animal diseases in Sardinia. PLoS One, 14(6), e0217367.
- Longjam, N., Deb, R., Sarmah, A. K., Tayo, T., Awachat, V. B., & Saxena, V. K. (2011). A brief review on diagnosis of foot-and-mouth disease of livestock: conventional to molecular tools. *Veterinary medicine international*, 2011.
- Machira, D. N., &Kitala, P. (2017). Epidemiological analysis of passive surveillance data on foot and mouth disease occurrence in Nakuru County, Kenya. J Dairy Vet Anim Res, 6(3), 00178.
- MacPhillamy, I., Young, J., Earp, F., Khounsy, S., Windsor, P., Toribio, J. A., & Bush, R. (2022). Foot-and-mouth disease seroprevalence and reporting behaviours nine northern in provinces in Lao PDR: The current situation and challenges for control. Transboundary and Emerging Diseases, 69, 645-659.
- Maree, F. F., Kasanga, C. J., Scott, K. A., Opperman, P. A., Chitray, M., Sangula, K., Sallu, A. R., Sinkala, Y., Wambura, P. N., King, D. P., Paton. D.. & Rweyemamu, M. (2014). Challenges and prospects for the control of foot-and-mouth African disease: An perspective. *Veterinary* Medicine: Research and Reports, 5, 119–138.
- Maree, F., de Klerk-Lorist, L. M., Gubbins, S., Zhang, F., Seago, J., Pérez-Martín, E., ... &Juleff, N. (2016).

Differential persistence of foot-andmouth disease virus in African buffalo is related to virus virulence. *Journal of virology*, 90(10), 5132-5140.

- Marqués, F. J., Battistessa, E. I., Peek, S. F., Raabis, S. M., & Darien, B. J. (2019). The effect of foot-and-mouth disease vaccination on early pregnancy loss in beef heifers in Argentina. *Preventive veterinary medicine*, 170, 104716.
- "Information Meenowa, D. (2020): Received on 14/09/2016 from Dr DeodassMeenowa, Assistant-director (livestock and veterinary services), division of veterinary services. Ministry of Agro-Industry and Food Security. REDUIT, Mauritius", https://www.oie.int/wahis _2/public/wahid.php/Reviewreport/R eview?reportid=20849.
- Mekibib, B., &Ariën, K. K. (2016). Aerosol transmission of filoviruses. Viruses, 8(5), 148.
- Mielke, S. R., & Garabed, R. (2020). Environmental persistence of foot-and-mouth disease virus applied to endemic regions. Transboundary and Emerging Diseases, 67(2), 543–554.
- Miguel, E., Grosbois, V., Caron, A., Boulinier, T., Fritz, H., Cornélis, D., V., Foggin, C., Makaya, P. Tshabalala, P. T. and de Garine-Wichatitsky, M. (2013): Contacts and foot and mouth disease transmission from wild to domestic bovines in Africa. *Ecosphere*, 4, art51.
- Mouchantat, S., Haas, B., Lutz, W., Pohlmeyer, K., &Frölich, K. (2005). Absence of antibodies to foot-andmouth disease virus in free-ranging roe deer from selected areas of

Germany (2001-2002). *Journal of Wildlife Diseases*, *41*(3), 599-605.

- Mwiine, F. N., Ayebazibwe, C., Olaho-Mukani, W., Alexandersen, S., Balinda, S. N., Masembe, C., Okurut, A. R. A., Christensen, L. S., Sorensen, K. J. and Tjornehoj, K. (2010): Serotype specificity of antibodies against foot-and-mouth disease virus in cattle in selected Uganda. Transbound. districts in *Emerg. Dis.* 57, 365–374.
- Neff, S., Sá-Carvalho, D., Rieder, E., Mason, P. W., Blystone, S. D., Brown, E. J., & Baxt, B. (1998).
 Foot-and-mouth disease virus virulent for cattle utilizes the integrin αvβ3 as its receptor. Journal of virology, 72(5), 3587-3594.
- OIE (2005–2013): World Organisation for Animal Health (OIE), World Animal Health Information Database (WAHID) Interface reports. Available at http://www.oie.int/links/ (accessed December 10, 2013).
- Omondi, G., Alkhamis, M. A., Obanda, V., Gakuya, F., Sangula, A., Pauszek, S., ... &VanderWaal, K. (2019).
 Phylogeographical and cross-species transmission dynamics of SAT1 and SAT2 foot-and-mouth disease virus in Eastern Africa. *Molecular ecology*, 28(11), 2903-2916.
- Omondi, G. P., Gakuya, F., Arzt, J., Sangula, A., Hartwig, E., Pauszek, S., ... &VanderWaal, K. (2020). The role of African buffalo in the epidemiology of foot-and-mouth disease in sympatric cattle and populations buffalo in Kenya. *Transboundary* and emerging diseases, 67(5), 2206-2221.
- Onono, J. O., Wieland, B., & Rushton, J. (2013). Constraints to cattle

production in a semiarid pastoral system in Kenya. *Tropical animal health and production*, 45(6), 1415-1422.

- Osofsky, S. A., Cleaveland, S. A.. Karesh, W. B., Kock, M. D., Nyhus, P. J., Starr, L. and Yang, A. (2005): Conservation and development interventions at the wildlife/livestock interface: implications for wildlife, livestock human health. and In: S. A. Osofsky (ed.), xxxiii + 220 pp. IUCN Gland, Switzerland and Cambridge, UK.
- Pacheco, J. M., Lee, K. N., Eschbaumer, M., Bishop, E. A., Hartwig, E. J., Pauszek, S. J., ... &Arzt, J. (2016).
 Evaluation of infectivity, virulence and transmission of FDMV field strains of serotypes O and A isolated in 2010 from outbreaks in the Republic of Korea. PLoS One, 11(1), e0146445.
- Parida, S. (2009). Vaccination against footand-mouth disease virus: strategies and effectiveness. *Expert review of vaccines*, 8(3), 347-365.
- Paton, D. J., Valarcher, J. F., Bergmann, I. E., Matlho, O. G., Zakharov, V. M., Palma, E. L., & Thomson, G. R. (2005).Estudio de la selección de cepasvacunales contra la fiebreaftosa. *Revue Scientifique et Technique de l'OIE*, 24(3), 981-993.
- Paton, D. J., K. J. Sumption, and B. Charleston, (2009): Options for control of foot-and-mouth disease: knowledge, capability and policy. *Philos. Trans. R Soc. Lond.* A 364, 2657–2667.
- Phologaine, B. S., Dwarka, R. M., Haydon, D. T., Gerber, L. J. andVosloo, W. (2008): Molecular characterization of SAT-2 foot-andmouth disease virus isolates obtained

from cattle during a four-month period in 2001 in Limpopo Province, South Africa. *Onderstepoort J. Vet. Res.* 75, 267–277.

- Prempeh, H., Smith, R., & Müller, B. (2001). Foot and mouth disease: the human consequences: The health consequences are slight, the economic ones huge. *Bmj*, 322(7286), 565-566.
- Qureshi, S. S., Khan, B., Khan, S., Ur Rahman, H., & Qureshi, M. S. (2022). Comparative Study of the Virulency of Different Serotypes of Foot and Mouth Disease Virus by Using Baby Hamster Kidney-21 Cell Line. Sarhad Journal of Agriculture, 38(3), 778-783.
- Robinson, T. P. K., Franceschini, G., Kruska, R. L., Chiozza, F., Notenbaert, A., Cecchi, G., Herrero, M., Epprecht, M., Fritz, S., You, L., Conchedda, G. and See, L. (2011): Global Livestock Production Systems, p. 152. Food and Agriculture Organization of the United Nations (FAO) and International Livestock Research Institute (ILRI), Rome.
- Roeder, P. L., and Knowles, N. J. (2008): *Foot-and-mouth Disease Virus Type C Situation: The Target for Eradication?*.Erice, Sicily, Italy.
- Roeder, P., Abraham, G., Mebratu, G. and Kitching, R. (1994): Foot-andmouth disease in Ethiopia from 1988 to 1991. *Trop.* Anim. Health Prod. 26, 163–167.
- Rowlands, D. J. (2008). Foot and mouth disease viruses. In B. W. J. Mahy & M. H. V. Van Regenmortel (Eds.), *Encyclopedia of virology* (3rd ed., pp. 265–274). Academic Press. https://doi.org/10.1016/B978-012374410-4.00402-7

- Rufael, T., Catley, A., Bogale, A. Sahle, M. and Shiferaw, Y. (2008): Foot and mouth disease in the Borana pastoral system, southern Ethiopia and implications for livelihoods and international trade. *Trop. Anim. Health Prod.* 40, 29–38.
- Rweyemamu, M. M., Booth, J. C., Head, Pay. T. W. М., & (1978). Microneutralization tests for serological typing and subtyping of foot-and-mouth disease virus strains. *Epidemiology* Å Infection, 81(1), 107-123.
- Rweyemamu, M., Roeder, P., Mackay, D., Sumption, K., Brownlie, J., Leforban, Y.,Valarcher, J. F., Knowles, N. J. and Saraiva, V. (2008): Epidemiological patterns of foot-and-mouth disease worldwide. *Transbound. Emerg. Dis.* 55, 57–72.
- Salt, J. S., (2004): Persistence of foot-andmouth disease virus. In: F. Sobrino, and E. Domingo (eds), Foot and mouth Disease: Current Perspectives, pp. 103–143. Horizon Bioscience, Wymondham, UK.
- Samuel, A. R., and Knowles, N. J. (2001): Foot-and-mouth disease type O viruses exhibit genetically and geographically distinct evolutionary lineages (topotypes). J. Gen. Virol. 82, 609–621.
- Sangare, O., Bastos, A. D., Marquardt, O., Venter, E. H., Vosloo, W. and Thomson, G. R. (2001): Molecular epidemiology of serotype O footand-mouth disease virus with emphasis on West and South Africa. *Virus Genes* 22, 345–351.
- Sangula, A. K., Belsham, G. J., Muwanika, V. B., Heller, R., Balinda, S. N., Masembe, C., &Siegismund, H. R. (2010). Evolutionary analysis of foot-and-mouth disease virus

serotype SAT 1 isolates from east Africa suggests two independent introductions from southern Africa. *BMC* evolutionary *biology*, 10, 1-8.

- A. K., Siegismund, H. Sangula, R., Belsham, G. J., Balinda, S. N.. Masembe, C. and Muwanika, V. B. (2011): Low diversity of foot-andmouth disease serotype C virus in Kenva: evidence for probable vaccine strain re-introductions in the field. *Epidemiol*. Infect. 139, 189-196.
- Scoones, I. and Wolmer, W. (2006): Livestock, Disease, Trade and Markets: Policy Choices for the Livestock Sector in Africa. *IDS WORKING PAPER 269*. Institute of Development Studies at the University of Sussex, Brighton, UK.
- Scoones, I., Bishi, A., Mapitse, N., Moerane,
 R., Penrith, M. L., Sibanda, R., ...
 &Wolmer, W. (2010). Foot-andmouth disease and market access:
 challenges for the beef industry in
 southern Africa. vol. 1, no. 2
- Seago, J., Jackson, T., Doel, C., Fry, E., Stuart, D., Harmsen, M. M., ... &Juleff, N. (2012). Characterization of epitope-tagged foot-and-mouth disease virus. *Journal of general virology*, 93(11), 2371-2381.
- Sellers, R. F. (1971). Quantitative aspects of the spread of foot and mouth disease. *Vet. Bull.*, *41*, 431-439.
- Siegel J.D., Rhinehart E., Jackson M., ChiarelloL.andThe Healthcare Infection Control Practices Advisory Committee (2007). Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings. [(accessed on 25 February 2021)]; Available online: <u>https://www.cdc.gov/infectio</u>

ncontrol/guidelines/isolation/index.ht ml

- Sinkala, Y. (2016): FMD Zambia Information Received on 03/03/2016 from Dr Yona Sinkala, Director, Department of Veterinary Services, Ministry of Fisheries and Livestock, Lusaka,Zambia, <u>https://www.oie.int/</u> <u>wahis 2/public/wahid.php/Reviewre</u> <u>port/Review?reportid=198.</u>
- Sinkala, Y., Simuunza, M., Pfeiffer, D. U., Munang'Andu, H. M., Mulumba, M., Kasanga, C. J., ... &Mweene, A. S. (2014). Challenges and economic implications in the control of foot and mouth disease in sub-Saharan Africa: Lessons from the Zambian experience. Veterinary medicine international, 2014.
- Squarzoni-Diaw, C., Arsevska, E., Kalthoum, S., Hammami, P., Cherni, A., Karim J., Daoudi, Laoufi. M., Lezaar, Y., Rachid, I., OuldElmamy, K., Seck, B., Yahya, B., Dufour, B., Hendrikx, P., Cardinale, E., Muñoz, F., Lancelot, & Coste, R., C. (2021). Using participatory a qualitative risk assessment to estimate the risk of introduction and spread of transboundary animal diseases in scarce-data environments: A Spatial Qualitative Risk Analysis applied to foot-and-mouth disease in Tunisia 2014-2019. Transboundary and Emerging Diseases, 68(4), 1966-1978. https://doi.org/10.1111/tbed.1

1978. https://doi.org/10.1111/tbed.1 3920

Stenfeldt, C., &Belsham, G. J. (2012). Detection of foot-and-mouth disease virus RNA in pharyngeal epithelium biopsy samples obtained from infected cattle: investigation of possible sites of virus replication and persistence. *Veterinary microbiology*, *154*(3-4), 230-239.

- Stenfeldt, C., &Arzt, J. (2020). The carrier conundrum; a review of recent advances and persistent gaps regarding the carrier state of footand-mouth disease virus. *Pathogens*, 9(3), 167.
- Stenfeldt, C., Eschbaumer, M., Rekant, S. I., Pacheco, J. M., Smoliga, G. R., Hartwig, E. J., ... &Arzt, J. (2016). The foot-and-mouth disease carrier state divergence in cattle. *Journal of virology*, 90(14), 6344-6364.
- Stenfeldt, C., Pacheco, J. M., Singanallur, N. B., Vosloo, W., Rodriguez, L. L., &Arzt, J. (2019). Virulence beneath the fleece; a tale of foot-and-mouth disease virus pathogenesis in sheep. *Plos one*, 14(12), e0227061.
- Stenfeldt, C., Bertram, M. R., Smoliga, G. R., Hartwig, E. J., Delgado, A. H., &Arzt, J. (2020). Duration of Contagion of Foot-And-Mouth Disease Virus in Infected Live Pigs and Carcasses. *Frontiers in Veterinary Science*, 7, 334.
- Sugiura, K., Ogura, H., Ito, K., Ishikawa, K., Hoshino, K., & Sakamoto, K. (2001). Eradication of foot and mouth disease in Japan. *Revue Scientifique et Technique-Office International des Epizooties*, 20(3), 701-714.
- Sumption, K. J., Pinto, J., Lubroth, J., Morzaria, S., Murray, T. and Rocque, S. (2007): Foot-andmouth disease: situation worldwide and major epidemiological events in 2005–2006. EMPRES Focus On Bulletin 1.
- Sutmoller, P., McVicar, J. W., &Cottral, G. E. (1968). The epizootiological importance of foot-and-mouth disease carriers. Archiv für die gesamteVirusforschung, 23(3), 227-235.

- Sutmoller, P., Barteling, S. S., Olascoaga, R.
 C., & Sumption, K. J. (2003).
 Control and eradication of foot-and-mouth disease. *Virus research*, 91(1), 101-144.
- Tekleghiorghis, T., Moormann, R. J., Weerdmeester, K., & Dekker, A. (2014). Serological Evidence Indicates that Foot-and-Mouth Disease Virus Serotype O, C and SAT 1 are most Dominant in E ritrea. *Transboundary and emerging diseases*, 61(6), e83-e88.
- Tekleghiorghis, T., Moormann, R. J., Weerdmeester, K., & Dekker, A. (2016). Foot-and-mouth disease transmission in Africa: Implications for control, a review. *Transboundary and emerging diseases*, 63(2), 136-151.
- Thomson, G. R. (1995): Overview of foot and mouth disease in southern Africa. *Rev. Sci. Tech.* 14, 503–520.
- Thomson, G. R., Vosloo, W., & Bastos, A. D. S. (2003). Foot and mouth disease in wildlife. *Virus research*, *91*(1), 145-161.
- Thompson, K. A., Bennett, A. M., & Walker, J. T. (2011). Aerosol survival of Staphylococcus epidermidis. *Journal of Hospital Infection*, 78(3), 216-220.
- van Vlijmen, H. W., Curry, S., Schaefer, M., & Karplus, M. (1998). Titration calculations of foot-and-mouth disease virus capsids and their stabilities as a function of pH. Journal of molecular biology, 275(2), 295-308.
- Vosloo, W. and Thomson. G. R. (2004): Natural habitats in which foot-and-mouth disease virus is maintained. In: F. Sobrino and E. Domingo (eds), Foot and Mouth Disease: Current Perspectives.

Horizon Bioscience, Wymondham, UK.

- Vosloo, W., Bastos, A. D. S., Sangare, O., Hargreaves, S. K., & Thomson, G. R. (2002). Review of the status and control of foot and mouth disease in sub-Saharan Africa. *Revue* scientifique et technique-Office international des épizooties, 21(3), 437-445.
- Vosloo, W., Bastos, A. D. S., Sahle, M., Sangare, O. and Dwarka, R. M. (2005): Virus topotypes and the role of wildlife in Foot-and-mouth disease in Africa. In: S. A. Osofsky (ed.), Conservation and Development Interventions at the Wildlife/Livestock Interface. Implications for Wildlife Livestock, and Human Health, pp. 67-73. The Species **IUCN** Survival Commission, Durban, South Africa.
- Vosloo, W., Bastos, A. D. and Boshoff, C. I. (2006): Retrospective genetic analysis of SAT-1 type foot-andmouth disease outbreaks in southern Africa. *Arch. Virol.* 151, 285–298.
- Vosloo, W., de Klerk, L. M., Boshoff, C. I., Botha, B., Dwarka, R. M., Keet, D. Haydon, D. T. and (2007): Characterisation of a SAT-1 outbreak of foot-and-mouth disease in captive African buffalo (Synceruscaffer): clinical symptoms, characterisation genetic and phylogenetic comparison of outbreak Microbiol. 120, 226isolates. Vet. 240.
- Vosloo, W., Thompson, P. N., Botha, B., Bengis, R. G. and Thomson, G. R. (2009): Longitudinal study to investigate the role of impala (Aepyceros melampus) in foot-andmouth disease maintenance in the Kruger National Park, South

Africa. *Transbound. Emerg. Dis.* 56, 18–30.

- Vosloo, W., Swanepoel, S. P., Bauman, M., Botha, B., Esterhuysen, J. J., Boshoff, C. I..Keet. D. F. and Dekker, A. (2011): Experimental infection of giraffe (Giraffa camelopardalis) with SAT-1 and SAT-2 foot-and-mouth disease virus. Transbound. Emerg. Dis. 58, 173–178.
- Wajid, A., Chaudhry, M., Rashid, H.
 B., Gill, S. S., & Halim, S.
 R. (2020). Outbreak investigation of foot and mouth disease in Nangarhar province of war-torn Afghanistan, 2014. Scientific Reports, 10(8).
- Ward, M. P., Laffan, S. W., &Highfield, L. D. (2007). The potential role of wild and feral animals as reservoirs of foot-and-mouth disease. *Preventive veterinary medicine*, 80(1), 9-23.
- Wekesa, S. N. (2012): Foot-and-mouth Disease Virus (FMDV) in the African Buffalo (*Synceruscaffer*) in Kenya,Jerez, Spain. Open Session of the Standing Technical and Research Committee of the EuFMD Commission, Jerez, Spain.
- Wijayanti, S. P. M., Porphyre, T., Chase-Topping, M., Rainey, S. M., McFarlane, M., Schnettler, E., Biek, R., & Kohl, A. (2016). The importance of socio-economic versus environmental risk factors for reported dengue cases in Java, Indonesia. PLoS Neglected Tropical Diseases, 10(9), e0004964.
- World Organisation for Animal Health (2017). Infection with foot-andmouth disease virus. In *Terrestrial Animal Health Code*; OIE: Paris, France.
- World Organisation for Animal Health (OIE)(2017). World Animal Health Information Database (WAHID).

Available

http://www.oie.int/wahis_2/public/wahid.

php/Wahidhome/Home/indexcontent /newlang/en.

at:

- World Organization for Animal Health (2022a). Official disease status. <u>https://www.woah.org/en/dise</u> <u>ase/foot-and-mouth-disease/#ui-id-2</u>
- World Organization for Animal Health (2022b). World animal health information system (WAHIS-OIE). <u>https://wahis.woah.org/#/home</u>
- World Reference Laboratory Pirbright FMD (WRLFMD)(2017). Molecular Epidemiology/Genotyping, OIE/FAO FMD Reference Laboratory Network Reports. <u>http://</u>

www.wrlfmd.org/fmd_genotyping/2 017.htm.

- WRLFMD (2003–2013): Molecular epidemiology/genotyping, OIE/FAO FMD reference laboratory network reports. Available at <u>http://www.wrlfmd.org/fmd_genot</u> <u>yping/index.html</u> (accessed December 19, 2013).
- Yehia, G., andPrimot, P. (2009): Foot and mouth disease control strategies in North Africa and the Middle East – the current situation. First OIE/FAO Global Conference on Foot and Mouth Disease: The way Towards Global Control. OIE, Asuncion, Paraguay.