Academic Journal of Animal Diseases 12(3): 48-59, 2023
ISSN 2079-200X

DOI: 10.5829/idosi.ajad.2023.48.59

# A Review on: Foot and Mouth Disease and Its Status in Ethiopia

*Gebeyehu Tefera Leye*

Gedeo Zone Bule Wereda Agricultural Office, Dilla, Ethiopia

**Abstract:** Foot and mouth disease (FMD) is an acute contagious and highly infectious disease of domesticated and wild cloven-hoofed animals characterized by the appearance of vesicular lesions at the epithelium of the mouth, nares, muzzle, feet and udder, causing repeated outbreaks every year in Ethiopia, thereby causing production loss and the banning of exportation of livestock, products and their by-products. The prevalence of foot and mouth disease in Ethiopia has been reported from different parts of the country, but a pooled quantitative estimate of foot and mouth disease has not been done so far; therefore, the objective of this study is to review Foot and Mouth Disease and its Epidemiology and Economic impact in Ethiopia. FMD is prevalent in Ethiopian cattle and the disease is a list A disease that could affect the export of live animals and their products since FMD is a transboundary disease. Therefore, it is imperative to have strategic implementation of effective prevention, control and robust eradication policies.

**Key words:** Economic Impact · Epidemiology · Ethiopia · Foot And Mouth Disease

## INTRODUCTION

Foot-and-mouth disease (FMD) is the most contagious and economically important acute Transboundary Animal Disease (TAD) affecting cloven-hoofed wild and domestic animals [1]. FMD is caused by a virus that belongs to the genus Aphthovirus of the family Picornaviridae. There are seven recognized serotypes of FMD (O, A, C, Asia1, SAT1, SAT2 and SAT3) which differ in distribution worldwide. Serotypes A and O have the widest distribution. Infection or vaccination against one serotype does not provide protection against the other serotypes [1]. Out of the seven serotypes of FMD virus, the existence of serotypes O, A, C, SAT 1 and SAT 2 were reported in Ethiopia [2].

The virus enters a new susceptible animal either orally (especially swine) or via the respiratory tract (especially cattle). Aerosol transmission is the major means of animal-to-animal spread within premises. The disease is characterized by vesicular lesions and erosions of the epithelium of the mouth, nose, muzzle, feet, teats and udder. FMD-infected animals usually develop blister-like lesions in the mouth, tongue lips, teats, or between the hooves, which causes them to salivate profusely or become lame. However, FMD virus (FMDV) is not typically considered a zoonotic disease and is not a threat to public health [3].

The accurate diagnosis of infection with FMDV is of prime importance for both control and eradication campaigns in FMD-endemic areas [4]. The disease is diagnosed based on clinical signs, however, the clinical signs can be confused with other diseases and thus, laboratory-based diagnosis is necessary [4]. For laboratory diagnosis, the sample of choice is tissue epithelium or vesicular fluid. Serum samples are also used for FMD diagnosis based on spiking of antibodies against a particular serotype [4]. Diagnosis of FMD in the laboratory is conducted by virus isolation and demonstration of the FMD viral antigens or nucleic acid in a sample tissue or fluid. Detection of virus-specific antibodies can also be used. Additionally, antibodies to viral nonstructural protein can be used as indicators of infection irrespective of vaccination status [4].

Foot and mouth disease preventive measures include control of national borders, prohibition of import of animals and livestock products from endemic countries in accordance with the OIE standards, emergency measures in the event of outbreaks through stamping-out, followed by cleaning and disinfection to reduce the risk of re-infection, strict movement controls, extending to movement on and off farms of livestock products. And also possible emergency vaccination is important [1].

**Corresponding Author:** Gebeyehu Tefera Leye, Gedeo Zone Bule Wereda Agricultural Office, Dilla, Ethiopia.

In Ethiopia, FMD has been a well-established endemic disease since its detection in 1957 for the first time [2]. Previous studies in the country reported FMD in different animal species with different prevalence levels. For example, in cattle, they reported FMD prevalence that ranges from 1.4 to 53.6% at the animal level and up to 61% at the herd level [5-7], in domestic small ruminants 4 to 11% and in ungulate wildlife 30% [8, 9]. Among the known FMD serotypes, four serotypes (A, O, SAT 1 and SAT 2) are maintained endemically in Ethiopia. Published articles on FMD show that type A and O are the main serotypes responsible for significant economic losses in the country [3, 10, 11].

Foot and mouth disease causes the highest economic impact on the poorest countries like Ethiopia where the livelihood of most of the people depends directly on livestock. This impact can be divided into two components: direct impacts due to production losses and change in herd structure and indirect impacts due to FMD control costs, limited access to market and limited use of new production technologies [12]. FMD impacts in terms of visible production losses and vaccination in endemic countries can cause losses of >USD 1.5 billion per year [12]. In Ethiopia FMD is posing a major threat thereby causing substantial economic losses through morbidity and mortality [13]. According to Jemberu *et al.* [3] the total annual losses due to FMD are estimated based on production losses, export losses and control costs to be greater than 1350 million Birr and the major cost is due to production losses. Therefore, the objective of this study is to review the foot and mouth disease, its epidemiology and economic impact in Ethiopia.

**Etiology:** Foot and mouth disease virus (FMDV) was the first recognized viral pathogen and is the sole member of the genus Aphthovirus belonging to the Picornaviridae family. Seven immunologically different serotypes of the FMD virus are known, namely, A, O, C, Asia-1,South African Territories (SAT) -1, -2 and -3, which comprise more than 65 subtypes. Initially 2 types were named: type O for Oise in France and type A for Allemagne (Germany). Later type C was recognized as an additional type in Germany [14]. Some 30 years later, work at Pirbright laboratory in England demonstrated 3 novel serotypes of FMDV in sample collected from the FMD outbreak in South Africa and called SAT1, SAT2 and SAT3. The seventh serotype, that is, Asia 1 was first recognized in a sample from Pakistan [15].

In Ethiopia serotypes O, A, C and SAT2 were responsible for FMD outbreaks between 1974 and 2003 [16, 17], while serotypes O, A and C caused FMD outbreaks in cattle from 1957 to 1979 [18]. Antibodies to SAT2 were also detected in 1971 from cattle in North Omo, south-western Ethiopia [18, 19]. The recent study conducted by Ayelet *et al*. [20] on FMD samples collected between 1981 and 2007 throughout the country from different species of animals showed the presence of serotype O, A, C, SAT1 and SAT2.

**Transmission of FMDV:** FMD is a directly transmitted disease with the predominant means of spread being direct or close contact between infected and susceptible animals. However, less frequently, transmission may occur indirectly through infection enabled by transporting healthy animals in vehicles that have previously transported infected livestock or through people handling healthy animals soon after being in contact with infected ones. Other mechanisms of local spread such as short-distance airborne transmission during outbreaks are suspected but unequivocal evidence is yet to be provided in this respect [21].

Much confusion has resulted from the finding in northern Europe that long-distance transmission has very rarely occurred through virus-containing aerosols being transported for many kilometers by air currents (i.e. air-borne spread). For this form of transmission to occur, a number of climatic and epidemiological circumstances need to prevail including a potent source of infection usually large piggeries suffering an explosive outbreak (because pigs excrete FMD virus more efficiently than other animals) high density of susceptible animals, cool temperature, often involving temperature inversion that prevents convection, gentle wind blowing in a constant direction and cattle as recipients of the aerosols, because cattle are more susceptible to aerosol infection than other species owing to their large inspiratory volume. In tropical/sub-tropical climates these requirements are seldom, if ever, met. A recent publication has postulated that aerosols may be derived from the skin of infected animals but that remains to be proven [22].

Among ruminants, cattle certainly, infection usually occurs via the respiratory tract and cattle may be infected by small numbers of infectious virions [21]. Conversely, large amounts of infectivity are required to cause infection by the oral route in cattle. In pigs by contrast, the oral

route of infection is most common with infection resulting from the feeding of pigs with untreated swill being a common source of FMD outbreaks in Europe and Asia. There has only been one recorded case of this type of outbreak in southern Africa. It has been shown experimentally that animals infected with a FMD virus may excrete significant amounts of 'infectivity' for up to 3 days before obvious clinical signs develop and this has been considered epidemiologically important. Recently, however, it was shown in a series of experiments in cattle that the amounts of virus excreted before the development of clinical signs were insufficient to result in transmission; only about half a day after clinical signs developed did transmission occur [23].

**Epidemiology:** The serotypes of FMDV are not distributed uniformly around the world. The serotype O, A and C viruses have had the widest distribution and have been responsible for outbreaks in Europe, America, Asia and Africa. However, the last reported outbreak due to serotype C FMDV was in Ethiopia during 2005 [24] and so serotype C viruses may no longer exist outside of laboratories. The SAT1-3 viruses are normally restricted to sub-Saharan Africa. The current global burden of FMDV infection is maintained within three continental reservoirs in Asia, Africa and South America, which can be further subdivided into seven major virus pools of infection [24, 25]. Each of these contains at least three serotypes of virus and because virus circulation is mainly within these regional reservoirs, strains have evolved which are specific to the region and which often (in the case of type A and SAT viruses) require tailored diagnostics and vaccines for control [26].

In Africa, the FMDV serotypes are not uniformly distributed and each serotype results in different epidemiological patterns. The cumulative incidence of FMDV serotypes shows that six of the seven serotypes of FMD (O, A, C, SAT1, SAT2 and SAT3) have occurred in Africa [27, 28]. Based on the genetic characterization of the virus and antigenic relationship of FMDV in Africa, the virus distribution has been divided into three virus pools: namely, pool 4 covering East and North Africa, with the predominance of serotypes A, O, SAT1 and SAT2; pool 5 restricted to West and northern Africa, with serotypes O, A, SAT1 and SAT2; and pool 6 restricted mainly to South Africa, with SAT1, SAT2 and SAT3 serotypes. Recent studies in East and southern Africa have revealed genetic differences between viruses isolated at different times and places [29, 30].

Periodically, there have been incursions of types SAT1 and SAT2 from Africa into the Middle East, probably as a result of animal movement [27, 28]. The most recent reports include the spread of viruses of the SAT2 serotype to Yemen in 1990, to Kuwait and Saudi Arabia in 2000 and to the Palestinian Autonomous Territories and Bahrain in 2012 [31].

FMD is one of the major endemic transboundary livestock diseases of socioeconomic importance in Ethiopia and in other parts of the globe. The seroprevalence of FMD in different regions of Ethiopia as indicated by different studies accounted that, the prevalence in Borena zone of Oromia Regional State was 53.6% [32]. The other studies also indicated that the prevalence of FMD in Eastern zone of Tigray, Yeka (Addis Ababa) and Guji zone of Oromia Regional State was accounted 41.5%, 32.7% and 30% respectively. Moreover, other studies indicated that the prevalence in Bale zone was 21.9% [33] in Somali was 14.05% [34] and in around Dessie zuria and Koombolcha area was 5.59% [35].

Overall, the geographic and genetic clustering of FMDVs suggest ecological adaptation and/or separation, but in many endemically affected areas, the temporal and spatial dynamics of infection still need to be much more accurately determined by analysis of host animal distributions and contact opportunities, serosurveys to estimate weight of infection and use of the latest available techniques in genetic tracing of FMDV incursions into disease-free regions [36]. Generally, many of these factors are driven by climatic factors and socioeconomic changes centered on human behavior. Also, findings regarding the epidemiology of FMD involving wildlife within a particular ecosystem of Africa may not be applicable to other ecosystems because of ecological, host and viral variability differences [37]. Understanding of how these risk factors are clustered and associated in space and time may assist in effective disease control planning [38, 39].

**Susceptible Livestock Population:** FMD affects all cloven-footed animals. Cattle, sheep, goats and pigs are the main domesticated species infected. The Water Buffalo (Bubalus bubalis) can become infected and may also transmit the infection to other species (www.reuters.com). The World Organization for Animal Health (OIE) code chapter on FMD includes the Camelidae as susceptible to FMD, similar to cattle, pigs, sheep and goats, but infection dynamics vary across species [41]. The two closely related camel species of Bactrian and dromedary camels possess noticeably

Table 1: Samples received in 2005 by the World Reference Laboratory for Foot-and-Mouth Disease at Institute of Animal Health, Pirbright (UK)

| Country | Type O | Type A | SAT 1 | SAT 2 | C |
|---|---|---|---|---|---|
| Botswana | - | - | 8 | - | - |
| Cameroon | 25 | 3 | - | 54 | - |
| Ethiopia | 22 | 9 | - | - | 4 |
| Kenya | - | - | 1 | - | - |
| Mali | 3 | - | - | - | - |
| Sudan | 3 | - | - | - | - |
| Togo | 4 | 1 | - | - | - |
| Zambia | - | - | 2 | - | - |

Table 2: Distribution of Foot and mouth disease virus in different parts of Ethiopia

| No | Area | Pervalence | Authors |
|---|---|---|---|
| 1 | Borena zone of Oromia | 53.6% | [32] |
| 2 | Eastern zone of Tigray | 41.5% | [33] |
| 3 | Yeka Addis Abeba | 32.7% | [33] |
| 4 | Guji zone of Oromia | 30% | [33] |
| 5 | Somalia region | 14.05% | [34] |
| 6 | Dessie and Kombolcha | 5.59% | [35] |
| 7 | South Nations Nationalities | 5.6% | [40] |
| 8 | Afar | 3.9% | [40] |
| 9 | Amhara | 2.6% | [40] |
| 10 | Oromia | 20.7% | [40] |
| 11 | Tigray | 16.5% | [40] |

different susceptibility to FMD virus [42]. Dromedary camels appeared to be susceptible to FMD serotype O, but they are unlikely to play any significant role in the natural epidemiology of FMD [43].

A wide range of wild cloven-footed animals contract FMD including deer and wild pigs. African buffalos play an important role in the maintenance of FMDV infection within National Parks in Uganda. Both SAT 1 and SAT 2 viruses were isolated and serological data indicate that it is also likely that FMDV serotypes O and SAT3 may be present in the buffalo population [44]. FMD is not considered zoonotic. Although clinical cases have been proven in human, these are extremely rare in relation to human exposure during outbreaks [45]. In the recent outbreaks during 2011 in different countries the majority of species affected are cattle, swine and sheep. Source of recent outbreak due to wild life species has been reported in South Africa and Namibia [41].

**Pathogenesis:** The pathogenesis of FMD has recently been reviewed in detail; these reviews not only reveal the complexity of FMD's pathogenesis but identify many gaps in the level of present understanding altogether 33 knowledge gaps are listed in the two papers, so a simple account of the pathogenesis of FMD is currently impossible [46]. The route of infection of cloven hoofed animals, other than in pigs where it is generally oral, is thought to be respiratory. In cattle the tissues most consistently infected during the pre viraemic phase of the disease are the epithelia of the nasopharynx and larynx [3]. It is therefore likely this is the primary replication site in ruminants. The tissues of the nasopharynx and FMD viruses have a complex relationship because not only does initial infection of ruminants take place there but the nasopharynx is also the site of viral persistence in chronically infected animals (so called carriers). Vesicle formation, cell lysis and significant inflammation occur at secondary replication sites (oral mucosa, skin of the horn hoof junction & skin of the teats) but not in the epithelium of the primary replication site. The cells which support viral replication are located in the basal layer of nasopharyngeal epithelium [46]. However, the mechanism by which viral replication occurs in the nasopharyngeal epithelium without causing cell lysis is unknown; nor is there an explanation as to why virus can be readily cultured from pharyngeal scrapings (obtained using probing cups) that, in recently infected animals, may contain high levels of antibody (mainly IgA) directed against the infecting virus. In pigs, delayed clearance of viral RNA from pharyngeal and lymphoid tissues has been observed but that has not been shown for infectious virus [46]. It is currently concluded that persistent infection of pigs does not occur or at least is not epidemiologically important. One or two days before the onset of clinical signs, cattle and pigs develop viraemia which may endure for up to 3 days.

The source of virus in the circulation remains a matter of conjecture (i.e. another knowledge gap) but viraemia ensures distribution of virus to all parts of the animal's body. In infected animals the vesicles which develop at the sites of secondary replication contain by far the highest levels of infectivity; however, high concentrations of virus can also be found in lymph nodes, myocardium, lungs and skin even in the absence of obvious lesions [47- 49]. Virus may also accumulate in the spleen, liver, adrenals, myocardium, pancreas, thyroid and mammary glands. In mammary tissue and myocardium, however, viral replication occurs in secretory epithelial cells of the alveoli and myocytes respectively, resulting in clear microscopic lesions. There is an association between FMDV and dendritic cells in lymph nodes that results in localization of virus in germinal centres but the details of this association remain to be elucidated [3].

Epithelial lesions at secondary replication sites are initiated by infection of single cells in the stratum spinosum [50]. Following infection of these cells, bullae

develop either by lysis of cells swollen as a result of ballooning degeneration and the release of intracellular fluid, or by the formation of areas of focal intercellular oedema. The bullae then coalesce, rupture or, more rarely, the fluid seeps away resulting in desiccation of the lesion. The development of characteristic vesicular lesions in FMD is dependent on persistent local irritation or friction. In transplantation studies in guinea pigs, it was shown that epithelium from predilection sites grafted to other body areas lost that predilection and *vice versa* [51].

In various parts of the world including South America, East Africa and India/Pakistan, a heat-intolerance syndrome (sometimes referred to as 'hairy panters') has been associated with previous infection or 'chronic FMD', with a putative endocrine-related pathogenesis. The limited information available on this syndrome has been reviewed recently indicating that the extent of the syndrome's association with FMD remains speculative [46].

**Clinical Signs:** FMD is characterized by fever, profuse salivation, vesicles in the mouth and on the feet and a drastic reduction in milk production; sudden death in young stock may occur [47]. A sequel to FMD frequently described in African cattle is the complex of clinical signs referred to as 'heat-intolerance syndrome (HIS). The condition is characterized by intolerance to heat and affected animals show pronounced panting, increased body temperature and pulse rate during hot weather and abnormal hair growth [3, 46, 52, 53].

**Diagnoses:** The accurate diagnosis of FMDV infection is of utmost importance for the control and eradication of the disease in endemic regions. The initial diagnosis of FMD is normally based on clinical signs, but this can easily be confused with other vesicular diseases [54]. Hence, it is vital that the recognition of signs of the disease by the farmer is promptly conveyed to the relevant veterinary authorities to verify clinical symptoms and suspect samples should then be sent to the reference laboratory for confirmation. Rapid and precise data generated by laboratories provide vital support to FMD control and vaccination programs. However, in many African countries, samples received by the laboratory can be of poor quality due to an ineffective cold chain and long transport periods. These factors make laboratory diagnosis challenging and it is evident that sub-Saharan Africa requires diagnostic tools that are fit for purpose in these settings to allow for rapid diagnosis and the appropriate measures taken for control.

Existing diagnostic techniques for the detection of FMD are mainly based on the following principles:

- The identification of the infectious agent by virus isolation involving propagation on susceptible cell cultures [55].
- The detection of viral antigens by ELISA systems using FMDV-specific antibodies or capturing reagents [56, 57].
- Molecular detection of viral nucleic acid by reverse-transcription polymerase chain reaction (RT-PCR) and the genetic analysis of the nucleotide sequence, mostly of the VP1-coding region [58].
- Detection of FMDV-specific antibodies in animals previously exposed to the virus. The VNT is usually used as a confirmatory test for sera found positive by ELISA [59].

These techniques are primarily suited for well-equipped laboratories which are usually either national or regional reference laboratories [24]. The virus cell culture system, for example, requires careful handling of specimens to prevent environmental and cross-contamination, trained personnel and a BSL3 (biosecurity level 3) laboratory. The success of virus isolation is dependent on the sample quality and requires special transport conditions from the sampling point to the laboratory [55]. Both the solid-phase competition ELISA and the liquid phase blocking ELISA for serological detection of FMDV specific antibodies against structural proteins are relatively simple procedures and easily implementable in diagnostic laboratories in endemic regions [60, 61].

**Prevention and Control:** The existing vaccines against FMD consist of complete, chemically inactivated virions combined with an adjuvant [62]. The adjuvant used in the vaccine formulation has undeniably a huge effect on the efficacy and potency of the vaccine and has been reviewed elsewhere [62, 63].

Despite successful application in the developed world, the effective administration and optimal induction of protective immunity are hampered by several factors in developing countries. In addition to the vaccine-matching constraints that have been discussed in the previous section, some viruses are very difficult to adapt to cell culture, slowing the introduction of new vaccine strains, reducing vaccine yield and potentiating through prolonged passage the selection of undesirable antigenic changes [64, 65]. Vaccination does not induce sterile immunity and animals may still be able to infect non

vaccinated animals and may also become persistently infected [62, 66].

The presence of contaminating non structural proteins in some vaccine formulations makes it problematic to distinguish between vaccinated and convalescent animals, impacting on the ability to export from FMD controlled regions. In addition, the hot climate in many African regions calls for vaccines with improved stability and which are less reliant on a cold chain. During production, the manufacturer also has to compensate for this instability by increasing the quantity of antigen per vaccine dose, which is expensive and reduces vaccine yield [66]. It is believed that unstable vaccines are less immunogenic due to degradation before and after inoculation. Therefore, FMD vaccines require frequent booster vaccinations in order to be effective. Lastly, the current vaccines are relatively expensive, especially for small and subsistence farmers.

Vaccines used in the control of FMD in endemic regions are mostly used for mass prophylactic application. Such vaccines are multivalent to provide protection against multiple serotypes and should have a potency of at least 3 PD50 per dose [24]. Generally, prophylactic vaccines incorporate 146S particles combined with saponin hydrogel or oil adjuvant [24].

Oil adjuvanted vaccines have been used successfully in FMD eradication campaigns in South America [13, 68, 69]. A study evaluating different adjuvants for SAT vaccines has shown that a double water-in-oil-in-water adjuvant, ISA206, elicited protective antibody responses against SAT2 serotype in cattle [70]. Inactivated vaccines induce short lived immunity and it is recommended that naïve animals receive two initial vaccinations (a primary and secondary dose) 3-4 weeks apart, followed by re-vaccination every 4-6 months to prevent spread of disease within populations [70, 71].

However, in the African environment, this may differ for different manufacturers, depending on the potency of the vaccine and some manufacturers recommend five vaccinations per annum. There is a definite need to assess whether different adjuvants may enhance the duration of immunity against SAT antigens. For these reasons vaccination campaigns should be performed regularly, based on; - 1) the epidemiological circumstances and risk of disease spread, 2) the value and life expectancy of species and 3) the economic status of the country. The interval between vaccinations is critical to prevent a "window of susceptibility" and where the continuous or sporadic presence of virus in carrier animals is present [24]

The PCP is the strategy proposed by OIE and FAO to control and ultimately eradicate FMD from endemic countries. Different regions in sub-Saharan Africa are at different developmental stages of control and are thus facing unique challenges and priorities in terms of FMD control. In many African endemic countries, there are various knowledge gaps, such as disease occurrence and mechanisms of virus maintenance and transmission and therefore no routine vaccination campaigns are implemented (PCP Stage 1 countries). In other African endemic countries, even where surveillance is conducted to provide knowledge about high-risk populations, often the implementation of effective, scheduled vaccination campaigns still does not take place (PCP Stage 2 countries). There are various reasons why governments do not subsidize FMD vaccines, leading to individuals needing to carry the cost and implement their own vaccine schedules. Additionally, individuals would need to source vaccines without knowledge of the current circulating strains in their region, leading to a poor vaccine match. This often leads to no or ineffective control in endemic African regions. The development of new vaccines against FMD in endemic countries in Africa should therefore take into account the ecosystem-based synchronization as FMD control strategies employed in these regions [24].

**Economic Impacts of FMD in Ethiopia:** The impact of the disease is not equal across all countries and livestock populations due to differences is not only FMD status, incidence and risk of incursion but also (a) the genetics of the national herd; (b) prevailing livestock management practices; (c) prevailing prices of livestock production inputs and outputs [72] and (d) their ability to supply livestock for export markets. Countries infected with FMD cannot trade live animals with FMD free countries. Typically the countries with the best meat prices are FMD free (i.e. EU, USA and Japan) [4] where prices are typically 50% higher [73]. The trade of livestock products is also restricted. If regular outbreaks occur only processed, tinned products can be exported to free countries; if FMD is effectively controlled with vaccination by a competent veterinary service able to detect outbreaks then deboned meat can be exported. Also, trade of fruit and vegetables can be affected by FMD status. Even if a country is FMD free, if it trades with FMD infected countries it will experience trade restrictions [4].

Ethiopia has the largest cattle population in Africa; in 2006 there were >43 million cattle with slightly fewer sheep and goats [74]. Large numbers of ruminants are exported;

in the Ethiopian financial year (July 2010-July 2011), meat and livestock export revenue was $211.1 million, mostly from live animal trade with the Middle East (>472,041 heads of live animals, 70% of which were cattle) [75]. However, production costs are high compared to other meat exporting nations, such as Australia or Brazil, limiting the potential for export market access regardless of FMD status. Difficulties in meeting export Sanitary and Phyto Sanitary standards results in greater numbers of livestock being purchased by traders for export through unofficial channels where prices are lower. Due to the presence of FMD and other OIE listed trade limiting diseases the export of live cattle and their products to FMD free countries is an unlikely prospect [74]. This raises the case for investment in veterinary service infrastructure to improve the control of all trade limiting diseases for international market access. Having an economy that is highly dependent on smallholder and animal based agriculture, including the widespread use of beasts of burden, the direct impacts of FMD are substantial in Ethiopia. In agro-pastoral areas, FMD infected oxen are unable to work for the entire season when affected at cropping time. Pastoralists are particularly vulnerable to FMD as their living depends entirely on their livestock [76]. By reducing the supply of milk FMD impacts on food security, particularly when outbreaks occur during times of the year when other food sources are limited and dependency upon milk is greatest [53].

**Direct Economic Impact**

**Visible Losses:** Production losses due directly to FMD include reduced milk production [76], affecting both the humans and calves that depend on it. This can account for 33% of losses in endemic settings [77]. Not only crucial to commercial dairy operations, milk is an important source of nutrition for many pastoralists, particularly for children [53]. Although FMD typically has a short-term effect on an animal's health, chronic FMD typically reduces milk yields by 80% [53, 76].

Livestock growth rates are also suppressed and mortality amongst young stock is typically 2-3% [78] although occasionally much higher [53, 79]. Loss of traction power where draught animals are used is particularly damaging if it occurs during harvest [80, 81]. FMD can result in abortion, the cost of which is high as the farmer will have to pay to keep the cow without it producing anything for another year or more, or cull the animal. Visible production losses are most prominent in pigs in intensive production systems and dairy cattle. These two systems are key sources of animal protein in poor countries and their importance continues to grow [82].

**Invisible Losses:** A compound effect of fertility problems due to abortion and reduced conception rates is a need to have a greater proportion of breeding animals in a population for a given output. This invisible loss means that for every kilo of meat or milk produced there is an additional fixed cost to maintain more breeding stock [72].

**Indirect Economic Impacts**

**Control Costs:** The cost of control carried out by the state veterinary services (e.g. vaccination, outbreak control, culling and compensation) is borne by the tax payer. In addition significant amounts are spent by the private sector. These costs are enormous with an estimated 2.35 billion doses of FMD vaccine administered in the world every year [83] at a cost of $0.4-3 or occasionally $9 per dose including delivery and application [13, 53, 84]. Due to the short duration of immunity induced by FMD vaccines, ongoing control programmes vaccinate cattle one to five times a year and sheep and goats once a year; limiting resources available to combat other diseases.

Wildlife is sometimes kept out of FMD free zones with fencing which is both costly and affects wildlife ecology [85]. Even if a country is FMD free there are ongoing costs due to efforts to prevent disease introduction, including import controls and sometimes vaccination. In addition, maintaining FMD early detection and control capability, including vaccine banks, is costly. Other costs include FMD-related research and permanent restrictions on the livestock sector (such as post-movement standstills and bans on feeding swill). The cost of surveillance is significant including proving disease freedom after an outbreak; >3 million serum samples were tested after the UK 2001 outbreak [59] in addition to approximately 3.5 million sera tested during the outbreak.

**Market Access:** Countries infected with FMD cannot trade live animals with FMD free countries. Typically the countries with the best meat prices are FMD free (i.e. EU, USA and Japan) [4] where prices are typically 50% higher [73]. The trade of livestock products is also restricted. If regular outbreaks occur only processed, tinned products can be exported to free countries; if FMD is effectively controlled with vaccination by a competent

Table 3: Different impacts of foot and mouth disease

| Direct impacts | | Indirect impacts | |
|---|---|---|---|
| Visible losses [76] | Invisible losses[72] | Additional costs [59] | Revenues forgone [4] |
| Loss of milk production | Fertility problem | Vaccine and Vaccine delivery | Use of suboptimal breeds |
| Loss of draft power | Change in herd structure | Movement control | |
| Loss of weight gain | Delay in sale of animals and / or animal products | Diagnostic tests | Denied access to markets both local and international |
| Dead animals | | Culled animals | |

veterinary service able to detect outbreaks then deboned meat can be exported [4]. Even if a country is FMD free, if it trades with FMD infected countries it will experience trade restrictions [4].

FMD is highly contagious, affects many species and is not easily contained within one farm or one population. The presence of FMD creates problems to all livestock owners who are connected to populations where FMD is present. This connection may be geographical or via market chains. Therefore, FMD creates what economists call externalities. If an outbreak occurs because one farmer did not protect his animals' others may suffer. Conversely, when livestock owner protects their animals from FMD infection they will generate a positive externality as they are less likely to become infected and transmit the pathogen to other farms [81].

The positive and negative impacts of FMD on different players in a dynamic market are complex; when FMD outbreaks create increased demand for vaccines, pharmaceutical companies benefit. When a free country experiences an outbreak poultry prices may increase due to public reluctance to consume products from FMD susceptible species, particularly if through ignorance there is a reluctance to eat products from FMD vaccinated animals. Where externalities exist there is a need for public investment as one farmer's actions create costs and benefits for others. These externalities are not equally shared amongst different livestock sectors with production losses being particularly severe for commercial dairy farms. Even when individuals reap positive returns from successful FMD control there is less of an incentive to undertake such a programme if there is a high risk of reinfection from those that do not attempt FMD control [81]. Effective control of infectious diseases with vaccination often requires high levels of vaccine coverage to develop herd immunity; with a sufficient proportion of immune animals outbreaks will tend to die out due to a lack of susceptible hosts. If left in the hands of individual farmers a lack of action by those less visibly affected by FMD will result in pockets where control is poor, undermining the entire control programme. Impacts on the livestock producer have ripple effects along the entire market chain, impacting on other players, such as markets, abattoirs and dairies to mention a few [86].

FMD control can be both an externality, with benefits not captured by the market and a regional or global public good, as the reduction in risk of FMD is also experienced by countries other than ones controlling the disease; external funding and cooperation is therefore required [84].

## CONCLUSION AND RECOMMENDATIONS

Foot and mouth disease (FMD) is a highly contagious viral disease of cloven-footed animals and is one of the most important economic diseases of livestock. The disease is characterized by fever and vesicular eruptions in the mouth, on the feet and on the teats. It is caused by a virus of the genus Aphthovirus, in the family Picornaviridae, of which there are seven immuno-logically district serotypes; O, A, C, South African Territories (SAT) 1, SAT2, SAT3 and Asia1. FMD is the most important disease limiting the trade of animals and animal products throughout the world.FMD is one of the major endemic transboundary livestock diseases of socio-economic importance in Ethiopia. FMD is the most economically important disease in Ethiopia and can cause both direct and indirect impacts on the economy. These economic losses are due to production losses (i.e., reduction of milk production, loss of draft power, mortality), restriction of export, control costs and prevention costs. From the above conclusion the following recommendations are forwarded:

- ✓ Implementing strict animal movement control both across national and international boundaries to limit across national and international boundaries to limit the spread of existing serotypes and the introduction of new serotypes.
- ✓ The multivalent vaccine candidates should be formulated containing all serotypes isolated.

- ✓ Regular FMD outbreak investigations should be conducted to have more detailed information about the serotypes and topotypes circulating in the country and regular vaccination programs should be started to control the outbreak of the disease.
- ✓ The epidemiology of FMD in Ethiopia along with the associated risk factors should be studied further in different areas nationally.

## REFERENCES


1. Sumption, K., J. Pinto, J. Lubroth, S. Morzaria, T. Murray, S.D.L. Rocque and F. Njeumi, 2007. Footand-Mouth disease situation worldwide and major epidemiological events in 2005-2006. Food and Agriculture Organization, Rome, Italy. http://www.fao.org/3/a-ai339e.pdf
2. Ayelet, G., E. Gelaye, H. Negussie and K. Asmare, 2012. Study on the epidemiology of foot and mouth disease in Ethiopia. Rev. Sci. Tech., 31: 789-98.
3. Jemberu, W.T., M.C.M. Mourits, M. Sahle, B. Siraw, J.C.M. Vernooij and H. Hogeveen, 2016. Epidemiology of foot and mouth disease in Ethiopia: a retrospective analysis of district level outbreaks (2007-2012). Transbound Emerg. Dis., 63: e246-59.
4. Jamal, S.M. and G.J. Belsham, 2013. Foot-and-mouth disease: Past, present and future. Vet. Res., 44: 1-14.
5. Mekonen, H., D. Beyene, T. Rufael, A. Feyisa and A. Fufa, 2011. Study on the prevalence of foot and mouth disease in Borana and Guji zones. S Ethiop Vet. World, 4(7): 293-6.
6. Tesfaye, A., M. Sahle, A. Abebe, A. Muluneh and D. Gizaw, 2016. Sero-prevalence of foot and mouth disease in cattle in Borena zone, Oromia regional state, Ethiopia. Ethiop Vet. J., 20(1): 55-66.
7. Wagari, A., 2016. Sero-prevalence of foot and mouth disease in bulls of Borana origin quarantined in Adama. J. Bio. Biophy Mol. Bio., 1(1): 1-10.
8. Sahle, M., 2004. An epidemiological study on the genetic relationships of foot and mouth disease viruses in east Africa. South Africa: PhD thesis, Faculty of Veterinary Science, University of Pretoria.
9. Beyene, B., T. Tolosa, T. Rufael, B. Hailu and T. Teklue, 2015. Foot and mouth disease in selected districts of western Ethiopia: sero-prevalences and associated risk factors. Rev. Sci. Tech., 34(3): 939-52.
10. Rufael, T., A. Catley, A. Bogale, M. Sahle and Y. Shiferaw, 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.
11. Gelaye, E., G. Ayelet, T. Abera and K. Asmare, 2009. Sero prevalence of foot and mouth disease in Bench Maji zone, south western Ethiopia. J. Vet. Med. Anim Health, 1(1): 00 5-0010.
12. Knight-Jones, T.J.D. and J. Rushton, 2013. The economic impacts of foot and mouth disease. Prev. Vet. Med., 112: 161-173.
13. Abdela, N., 2017. Sero-prevalence, risk factors and distribution of foot and mouth disease in Ethiopia. Acta Trop., 169: 125-132.
14. Waldmann, O. and K. Trautwein, 2003. Experimentelle untersuchungen ueber die pluralitet des maulund klauenseuche virus. Berliner und Münchener tierärztliche Wochenschrift, 91: 3-7.
15. Brooksyby, J.B. and J. Rogers, 1957. Method of Typing and Cultivation of Foot and Mouth Disease Virus. Paris, France: OEEC. Methods used in typing the virus of foot and mouth disease at Pirbright, pp: 31-34.
16. Sahle, M., 2004. An epidemiological study on the genetic relationship on FMDV in east Africa. A thesis submitted in partial fulfillment of the requirement for the degree of Doctor of Philosophy in the department of veterinary tropical diseases, Faculty of Veterinary Science University of Pretoria, South Africa, unpublished.
17. Gelaye E., B. Beyene and G. Ayelet, 2005. Foot and Mouth Disease virus serotypes identified in Ethiopia. Ethiopa, J.9, 75-80.
18. Roeder, P.L., G. Abraham., G.Y. Mebratu and R.P. Kitching, 1994. Foot and mouth disease in Ethiopia from 1988 to 1991. Trop. Anim. Health Prod. 26: 163-167.
19. Martel, J.L., 1974. Foot and mouth disease in Ethiopia. Distribution of viral serotypes. Rev. Elev. Med. Vet. Pays. Trop., 27: 169-175.
20. Ayelet, G.M., E. Mahapatra, G. Gelaye, T. Berhe, M. Rufeal, N. Sahle, J. Ferris, G.H. Wadsworth and N.J. Knowles, 2009. Genetic characterization of Foot and Mouth disease viruses, Ethiopia, 1981-2007. Emerg. Infect. Dis., 15: 1409-1417.
21. Thomson, G.R. and A.D.S. Bastos, 2004a. Foot and mouth disease. In: Infectious Diseases of Livestock, 2nd edn. JAW Coetzer & RC Tustin (eds.). Vol. 2,1324-1365. Cape Town: Oxford University Press Southern Africa.
22. Dillon, M.B., 2011. Skin as a potential source of infectious foot and mouth aerosols. Proceedings of the Royal Society B, 278: 1761-1767.

23. Charleston, B., B.M. Bankowski and S. Gubbins, 2011. Relationship between clinical signs and transmission of an infectious disease and the implications for control. Science, 332: 726-729.
24. Rweyemamu, M., P. Roeder and D. Mackay, 2008. Epidemiological patterns of foot and mouth disease worldwide. Transbound Emerg Dis. 55(1):57-87.
25. Tully, D.C. and M.A. Fares, 2008. The tale of a modern animal plague: tracing the evolutionary history and determining the time-scale for foot and mouth disease virus. Virology, 382(2): 250-256.
26. 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 B Biol. Sci., 364(1530): 2657-2667.
27. Valarcher, J.F., N. Knowles and R. Fernandez, 2004. Global FMD situation 2003-2004. In: Report of the Session of the Research Group of the Standing Technical Committee of the European Commission for the Control of Foot and Mouth, pp: 137-148.
28. Donaldson, A., 1999. The global status of foot and mouth disease and its relevance to control and eradication efforts in South East Asia. Report of 33rd session of the European Commission for the Control of Foot-and-Mouth Disease (EUFMD Commission); April 7-9, Rome.
29. Sangula, A.K., G.J. Belsham and V.B. Muwanika, 2010. Co circulation of two extremely divergent serotype SAT 2 lineages in Kenya highlights challenges to foot and mouth disease control. Arch Virol., 155(10): 1625-1630.
30. Kasanga, C.J., J. Wadsworth and C.A. Mpelumbe-Ngeleja, 2014. Molecular characterization of foot and mouth disease viruses collected in Tanzania between 1967 and 2009. Transbound Emerg Dis.
31. Hall, M.D., N.J. Knowles, J. Wadsworth, A. Rambaut and M.E. Woolhouse, 2013. Reconstructing geographical movements and host species transitions of foot and mouth disease virus serotype SAT 2. MBio., 4(5): e00591-13.
32. Habtamu, M., B. Desta, R. Tesfaye, F. Ashenafi and A. Fufa, 2011. Study on the prevalence of foot and mouth disease in Borana and Guji Zones, Southern Ethiopia. Vet. World, 4(7): 293-296.
33. Misgana, D., J. Yasmin, I. Ahmed and H. Addisalem, 2013. Seroprevalence of foot and mouth disease of cattle in Bale Zone, Oromiya Regional State, Ethiopia. Global Vet., 11(1): 59-64.
34. Abdulahi, M., T. Esaya and D. Hailu, 2013. Seroprevalence of bovine foot and mouth disease (FMD) in Awbere and Babille districts of Jijiga zone, Somalia Regional State, Eastern Ethiopia. Afr. J. Microbiol. Res., 5(21): 3559-3563.
35. Abraha, G. and I. Ahmmed, 2013. Sero-prevalence of foot and mouth disease in cattle in Dessie Zuria and Kombolcha Area, South Wollo, Ethiopia. Vet. Res., 6(1): 1-9.
36. Cottam, E.M., G. Thebaud and J. Wadsworth, 2008. Integrating genetic and epidemiological data to determine transmission pathways of foot-and-mouth disease virus. Proc Biol. Sci., 275(1637): 887-895.
37. Vosloo, W., P.N. Thompson, B. Botha, R.G. Bengis and G.R. Thomson, 2009. Longitudinal study to investigate the role of impala (Aepyceros melampus) in foot and mouth disease maintenance in the Kruger National Park, South Africa. Transbound Emerg Dis., 56(1-2): 18-30.
38. Molla, B., G. Ayelet and Y. Asfaw, 2010. Epidemiological study on foot-and-mouth disease in cattle: seroprevalence and risk factor assessment in South Omo zone, south-western Ethiopia. Transbound Emerg Dis., 57(5): 340-347.
39. Megersa, B., B. Beyene and F. Abunna, 2009. Risk factors for foot and mouth disease seroprevalence in indigenous cattle in Southern Ethiopia: the effect of production system. Trop. Anim. Health Prod., 41(6): 891-898.
40. Gelagy, A., 2008. National Veterinary Institute, Ethiopia. EUFMD. Sicily, Italy
41. Office International des Epizooties (OIE), 2009. Foot and mouth disease. OIE Terrestrial Mannual. Chapter2.1.5.http:www.oie.int/ eng/ A_FMD2012/ docs/ 2.01.05_FMD.pdf
42. Larskar, M., U. Wernery, J. Kinne, R. Schuskr, G. Aexandersen and S. Aexandersen, 2009. Differences in the susceptibility of dromedary and Bactrian camels to foot and mouth disease virus. Epidemo. Infect., 137: 549-554.
43. Yousef, M.R.., K.S. Mazoum and H.M. A-Nakhali, 2012. Serological evidence of natural exposure of camels (camelus dromedaries) to foot and mouth disease virus. Vet. World, 5(4): 197-200.
44. Ayebazibwe, C., F.N. Mwiine, K. Tjornehoj, S.N. Bainda, V.B. Muwanika, A.R.A. Okurut, G.J. Besham, P. Normann, H.R. Seigismund and S. Aexandersen, 2010. The role of African Buffaloes (Syncerus caffer) in the maintenance of foot and mouth disease in Uganda. BMC Veterinary Research, 6: 54.

45. Davies, G., 2002. Foot and mouth disease. Research in Veterinary Science, 73: 195-199.
46. Arzt, J., N. Juleff., Z. Zhang and L. Rodriguez, 2011b.The pathogenesis of foot-and-mouth disease I: Viral pathways in cattle. A review on Transboundary and Emerging Diseases 58(4): 291-304. doi:10.1111/ j.1865-1682.2011.01204.x.
47. Burrows, R., J.A. Mann, A. Garland, A. Grieg and D. Goodridge, 1981. The pathogenesis of natural and simulated natural foot and mouth disease infection in cattle. Journal of Comparative Pathology, 91: 599-629.
48. Zhang, Z. and S. Alexandersen, 2004. Quantitative analysis of foot and mouth disease viral loads in bovine tissues: implications for the site of viral persistence. Journal of General Virology, 85: 2567-2575.
49. Arzt, J., J.M. Pacheco and L.L. Rodriguez, 2010. The early pathogenesis of foot-and-mouth disease in cattle after aerosol inoculation: identification of the naso-pharynx as the primary site of infection. Veterinary Pathology, 47: 1048-1063.
50. Woodbury, E.L., 1995. A review of the possible mechanisms for persistence of foot and mouth disease virus. Epidemiology & Infection, 114: 1-13.
51. Platt, H., 1960. The localization of lesions in experimental foot and mouth disease. British Journal of Experimental Pathology, 41: 150-159.
52. Catley, A., R.T. Chibunda., E. Ranga., S. Makungu., F.T. Magayane., G. Magoma., M.J. Madege and W. Vosloo, 2004. Participatory diagnosis of a heat intolerance syndrome in cattle in Tanzania and association with foot and mouth disease. Preventive Veterinary Medicine, 65: 17-30.
53. Barasa, M., A. Catley, D. Machuchu, H. Laqua, E. Puot, D. Tap Kot and D. Ikiror, 2008. Foot-and-mouth disease vaccination in South Sudan: benefit-cost analysis and livelihoods impact. Transbound. Emerg. Dis., 55: 339-351.
54. Remond, M., C. Kaiser and F. Lebreton, 2002. Diagnosis and screening of foot and mouth disease. Comp Immunol Microbiol Infect Dis., 25(5-6): 309-320.
55. Jamal, S.M. and G.J. Belsham, 2013. Foot-and-mouth disease: past, present and future. Vet Res., 44: 116.
56. Abu Elzein, E.M. and J.R. Crowther, 1997. Enzyme labelled immunosorbent assay techniques in foot and mouth disease virus research. J. Hyg., 80(3): 391-399.
57. Ferris, N.P., N.G. Abrescia and D.I. Stuart, 2005. Utility of recombinant integrin ávâ6 as a capture reagent in immunoassays for the diagnosis of foot-and-mouth disease. J. Virol. Methods., 127(1): 69-79.
58. Di Nardo, A., NJ. Knowles and D.J. Paton, 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(1): 63-85.
59. Paton, D.J., J.F. Valarcher and I. Bergmann, 2005. Selection of foot-and-mouth disease vaccine strains-a review. Rev Sci Tech., 24(3): 981-993.
60. Paiba, G.A., J. Anderson and D.J. Paton, 2004. Validation of a foot and mouth disease antibody screening solid phase competition ELISA (SPCE). J. Virol. Methods. 115(2): 145-158.
61. Mackay, D.K., A.N. Bulut, T. Rendle, F. Davidson and N.P. Ferris, 2001. A solid phase competition ELISA for measuring antibody to foot and mouth disease virus. J. Virol. Methods., 97(1-2): 33-48.
62. Doel, T.R., 2003. FMD vaccines. Virus Res., 91(1): 81-99.
63. Kitching, R.P., 1997. Vaccination of calves against FMD in the presence of maternally derived antibody. In: European Commission for the Control of Foot and Mouth Disease; Israel, pp: 191-195.
64. Sa-Carvalho, D., E. Rieder and B. Baxt, 1997. Tissue culture adaptation of foot-and-mouth disease virus selects viruses that bind to heparin and are attenuated in cattle. J Virol., 71(7): 5115-5123.
65. Zhao, Q., J.M. Pacheco and P.W. Mason, 2003. Evaluation of genetically engineered derivatives of a Chinese strain of foot and mouth disease virus reveals a novel cell binding site which functions in cell culture and in animals. J. Virol., 77(5): 3269-3280.
66. Salt, J.S., 1993. The carrier state in foot and mouth disease an immunological review. Br Vet J., 149(3): 207-223.
67. Doel, T.R. and P.J. Baccarini, 1981. Thermal stability of foot and mouth disease virus. Arch Virol., 70(1): 21-32.
68. Bahneman, H.G. and J.A. Mesquita, 1987. Oil adjuvanted vaccine against foot and mouth disease. Bol Centro Panam Aftosa, 53: 25.
69. Dora, J.F.P., T. Coelho and J.C. Nunes, 1980. Epidemic of foot-and-mouth disease in Bage, RS, Brazil. Evaluation of two systems of vaccination. Bol Centr Panam Fiebre Aftosa, pp: 49-50.
70. Cloete, M., B. Dungu, L.I. Van Staden, N. Ismail-Cassim and W. Vosloo, 2008. Evaluation of different adjuvants for foot-and-mouth disease vaccine containing all the SAT serotypes. Onderstepoort J. Vet. Res., 75(1): 17-31.

71. Hunter, P., 1998. Vaccination as a means of control of foot and mouth disease in sub Saharan Africa.Vaccine., 16(2-3): 261-264.
72. Rushton, J., 2009. CAB International; Oxfordshire & Massachusetts. The Economics of Animal Health and Production, pp: 193-197.
73. Jarvis L.S., J.P. Cancino and J.E. Bervejillo, 2005. The Effect of Foot and Mouth Disease on Trade and Prices in International Beef Markets; American Agricultural Economics Association Annual Meeting, Providence, Rhode Island, 24-27.
74. Rich, K.M., D. Perry and S. Kaitibie, 2009. Commodity-based trade and market access for developing country livestock products: the case of beef exports from Ethiopia. Int. Food Agribus. Manag. Rev., 12: 1-22.
75. SPS-LMM., 2011. Focus on Ethiopia's Meat and Live Animal Export Ethiopia Sanitary & Phytosanitary Standards and Livestock & Meat Marketing Program Trade Bulletin.
76. Bayissa, B., G. Ayelet, M. Kyule, Y. Jibril and E. Gelaye, 2011. Study on seroprevalence, risk factors and economic impact of foot and mouth disease in Borena pastoral and agro-pastoral system, southern Ethiopia. Trop. Anim. Health Prod., 43: 759-766.
77. Ellis, P.R. and A.D. James, 1976. Foot and Mouth Disease: India. International Symposia on Veterinary Epidemiology and Economics proceedings, ISVEE 1: New Techniques in Veterinary Epidemiology and Economics, Proceedings of a Symposium, University of Reading; England, pp: 118-122.
78. Rufael, T., A. Catley, A. Bogale, M. Sahle and Y. Shiferaw, 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.
79. Office International des Epizooties (OIE), 2007. Disease card Foot and Mouth Disease. Center for Food Security and Public Health, Ames, Iowa, USA, pp: 1-6.
80. Perry B.D., W. Kalpravidh, P.G. Coleman, H.S. Horst, J.J. Mc Dermott, T.F. Randolph and L.J. Gleeson, 1999. The economic impact of foot and mouth disease and its control in South East Asia: a preliminary assessment with special reference to Thailand. Revue scientifique technique (OIE), 18: 478-497.
81. Perry, B.D., T.F. Randolph, S. Ashley, R. Chimedza, T. Forman, J. Morrison, C. Poulton, L. Sibanda, C. Stevens, N. Tebele and I. Yngström, 2003. International Livestock Research Institute (ILRI); Nairobi, Kenya. The Impact and Poverty Reduction Implications of Foot and Mouth disease Control in Southern Africa, with Special Reference to Zimbabwe, pp: 152.
82. Delgado, C., M. Rosegrant, H. Steinfeld, S. Ehui and C. Courbois, 1999. IFPRI; Washington, DC, USA. Livestock to 2020. The Next Food Revolution. Food, Agriculture and the Environment Discussion Paper, 28: 72.
83. Hamond, J., 2011. An event organised by NFUS, Moredun and Scottish Government. FMD Vaccine: Practical Applications from an International Perspective-FMDV Vaccine to Live.
84. Forman, S., F. Le Gall, D. Belton, B. Evans, J.L. François, G. Murray, D. Sheesley, A. Vandersmissen and S. Yoshimura, 2009. Moving towards the global control of foot and mouth disease: an opportunity for donors. Revue scientifique et technique (OIE), 28: 883-896.
85. Gadd, M.E., 2011. Barriers the beef industry and unnatural selection: a review of the impact of veterinary fencing on mammals in Southern Africa. In: Somers M.J., Hayward M., editors. Fencing for Conservation. Springer; New York, USA, pp: 153-186.
86. Le Gall, F. and N. Leboucq, 2004. The Role of Animal Disease Control in Poverty Reduction, Food Safety, Market Access and Food Security in Africa; Recueil des thèmes techniques présentés au Comité international ou aux Commissions régionales Vol. 2003, 87-106 et 107-126, Paris, France.