MATHEMATICAL MODELING OF FOOT AND MOUTH DISEASE (FMD) IN CATTLE AND BUFFALOES USING VACCINATION AND CULLING: A NAMIBIAN PERSPECTIVE

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Abstract

Foot and mouth disease (FMD) is an acute, infectious viral disease for animals, and it is one of the most rapidly spreading diseases worldwide. Countries worldwide are putting efforts to curb the infection as it has devastating effects on agriculture and wildlife economies. Mathematical models have been used to analyse the transmission and control of FMD to enable better decision making for animal health policy makers. In this study, we developed and analysed a basic mathematical model of the dynamics of FMD with and without vaccination and culling as control measures replicating the FMD infection in the interface setting of Namibia communal areas and National parks. Furthermore, we fit the model with control measures to the yearly cumulative FMD cases in Namibia and discuss the results in order to identify the impact of these controls on the Namibian scenario. Vaccination and culling are the control methods mostly used in the Namibian setting for the control of FMD. Mathematical theories for systems of ordinary differential equations were used to establish the existence and uniqueness of model solutions, as well as the stability of equilibrium points and to ensure that the mathematical solutions were biologically reasonable. Important threshold parameters such as the reproduction numbers were established, which are critical indicators of disease spread. Results from the study showed that the models have two equilibrium points namely; the disease free equilibrium (DFE) and the endemic equilibrium points. The DFE was shown to be locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. The endemic equilibrium point was shown to exist when $\mathcal{R}_0 > 1$ and globally stable when $\mathcal{R}_0 > 1$. Numerical simulation results, using Namibia data relevant to the transmission dynamics of FMD, were presented to illustrate some of the main theoretical results and model projections. The results from this study suggest that increasing vaccination rate and efficacy has a positive impact on reducing the spread of FMD. Moreover, we have also observed that vaccination is not enough to protect both the cattle and buffaloes, therefore, more efforts from policy makers should be devoted to putting extra measures in place for buffaloes and cattle not to interact more often. Better results were observed when both vaccination and culling were implemented, hence it is advisable to Namibia to practice culling as one of the control measures.

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List of Acronyms/Abbreviations

FMD- Foot and mouth disease

FMDV- Foot and mouth disease virus

SEIR- Susceptible- Exposed-Infectious-Recovered

SEIRVC- Susceptible- Exposed-Infectious-Recovered-Vaccination-Culling

DFE- Disease-free-equilibrium

OIE- World Organisation for Animal Health

SAT- Southern African Territories

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Dedication

This work is dedicated, firstly to the Almighty God, the source of strength. I also dedicate this thesis to my sons C.N.T. Keendjele, C.T. Keendjele, my husband B. Keendjele, my parents Thresia Kamati and Elias Shikumwifa, my best friend Victoria Amuthenu, my elder sister Christiane Tshikumitha and our last born Angeline Shikumwifa, and my entire family for moral and spiritual support. Remain blessed always.

DECLARATION

I, Evalthine Shikumwifa, hereby declare that this study, MATHEMATICAL MODELING OF FOOT AND MOUTH DISEASE (FMD) IN CATTLE AND BUFFALO USING VACCINATION AND CULLING: A NAMIBIAN PERSPECTIVE, is my own work and is a true reflection of my research, and that this work, or any part thereof has not been submitted for a degree at any other institution.

		April 2022
Name of student	**Signature**	

Chapter 1

Introduction

1.1 Biological background

Foot and mouth disease (FMD) is an acute, infectious viral disease for animals, and it is one of the most rapidly spreading diseases worldwide [1]. Foot and mouth disease virus affects animals with cloven hooves, including domestic livestock such as cows, pigs, goats, deer, sheep, and certain wild animals, including buffaloes [3]. FMD is caused by viruses that belong to seven immunologically distinct serotypes (that can only be discovered in the laboratory) of the *Aphthovirus* genus, which is categorised within the *Picornaviridae* family [4]. Moreover, the serotypes of FMD are categorised as European types (O, A and C), African types (SAT-1, SAT-2, and SAT-3) and finally Asian type (Asian 1) [5]. However, some countries such as Australia have never experienced FMD.

FMD virus can be found in excretions and secretions of infected animals, for instance saliva, milk, urine, semen and expired air [6]. Furthermore, the virus is airborne and can also be transmitted through direct contact from 60km overland and 300km by sea [6, 7, 8]. Additionally, many researchers, for instance [3, 4, 8, 9, 11], have emphasised more on the transmission of the virus through sexual contacts. In a similar manner, Aftosa in [11] added that sexual transmission might be a common route especially when it comes to SAT serotype, buffalo

populations pass the FMD virus. It is known that sheep pass FMDV through the placenta and infect the fetus [11]. Animals infected with FMD exhibit signs of clinical illness after an incubation period (time for the virus to be present in the animals before the first symptoms start to show) of about 2 to 14 days [1]. Clinical signs of FMD infection include high fever, and blisters called vesicles on the lips, tongue, in and around the mouth, on the mammary glands, and around the hooves, ruptured feed and small growth [12]. Lameness and excessive salivation may also be detected.

Nevertheless, animals hardly die from FMD. The FMD virus spreads faster within farms to the nearby farms and around areas that susceptible cattle can get in contact with infected buffaloes [6]. When infected and susceptible animals get in contact with each other, foot and mouth disease virus (FMDV) might be transmitted either from buffalo to buffalo, buffalo to cattle, cattle to buffalo or cattle to cattle. Aftosa in [11] notes that animals do not need the same dose of virus for them to be infected. For instance for the aerosolised virus, cattle are certainly susceptible while pigs need much higher doses to be infected through that route.

If the African buffaloes that pass through the borders from countries like Angola, Botswana, Zambia to Namibia happen to have FMD infection, and interact with susceptible cattle, then the cattle will be exposed to the infection. These are some of the ways through which FMD is transmitted because once cattle get the infection, then the infection will be transmitted to other susceptible animals, including other cattle and buffaloes. In the studies where transmission occurred, male buffaloes were mixed with female cattle, and cattle became infected only after the buffalo reached sexual maturity. This led to the hypothesis that FMD can be transmitted by the sexual route [11]. It is recognised that cattle are capable of sustaining FMDV for up to 3.5 years while African buffaloes for 5 years [13]. FMD is one of the most difficult animal infections to control because it can occur even in unaffected countries within a short period of time. FMD was recognised to be a zoonosis. Zoonosis is whereby the virus can be transmitted to human beings, for example by drinking infected cattle's milk [14]. Below in Figure 1.1 are

some FMD signs in cattle and buffaloes [15].

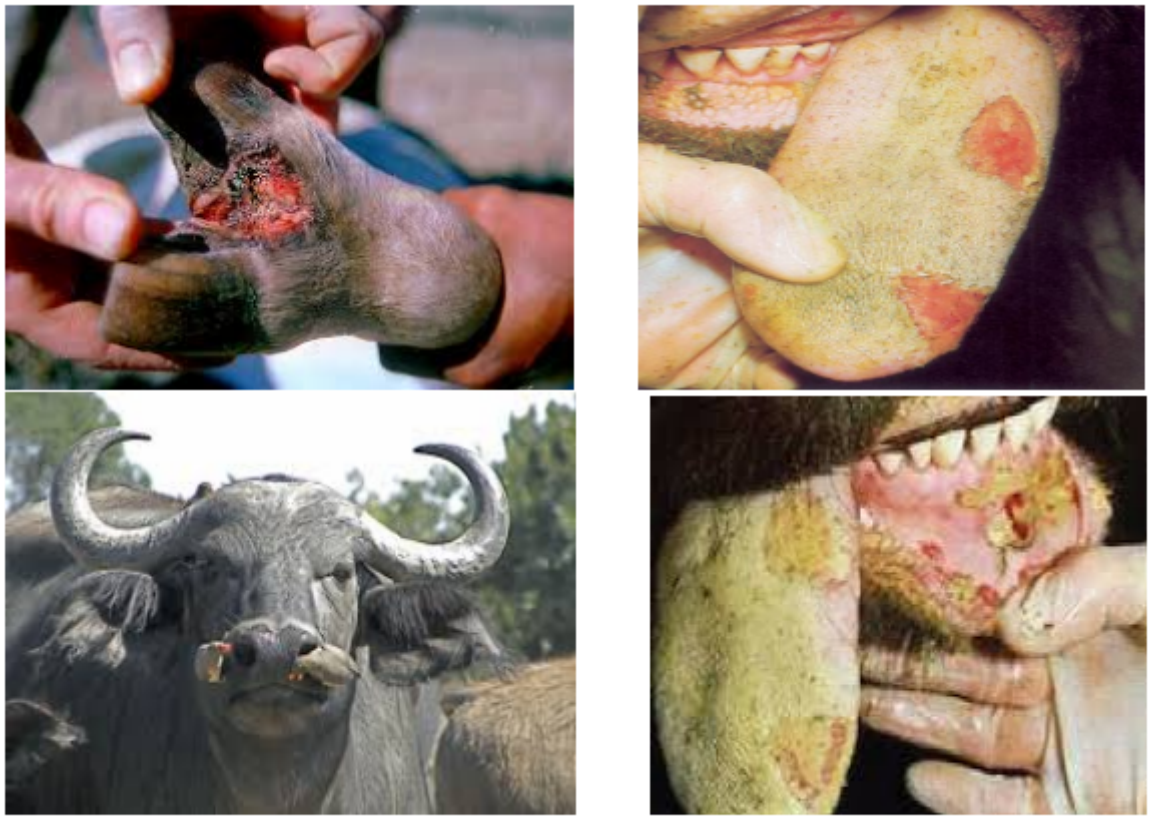

Figure 1.1: Pictures show some symptoms of FMD.

The following diagrams as summarised in short by [8] show how the FMD virus can be spread between susceptible animals using a simple S (susceptible), E (exposed), I (infected) and R (recovered) $(SEIR)$ model describing the cycles of FMDV transmission.

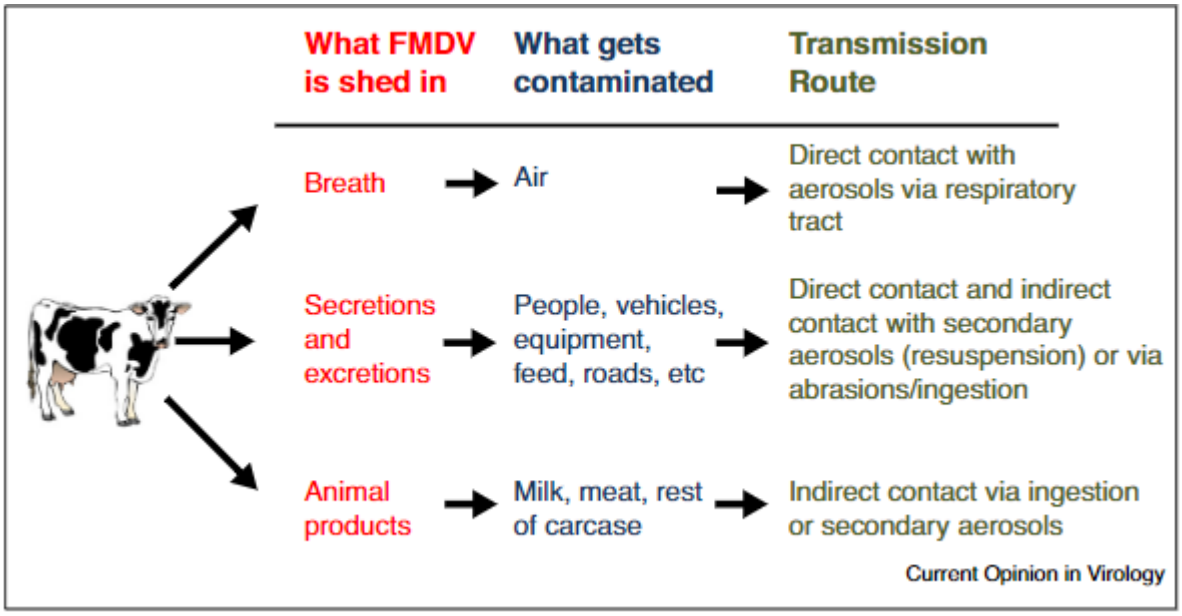

Figure 1.2: Principal routes by which infections FMD virus can be spread between susceptible animals.

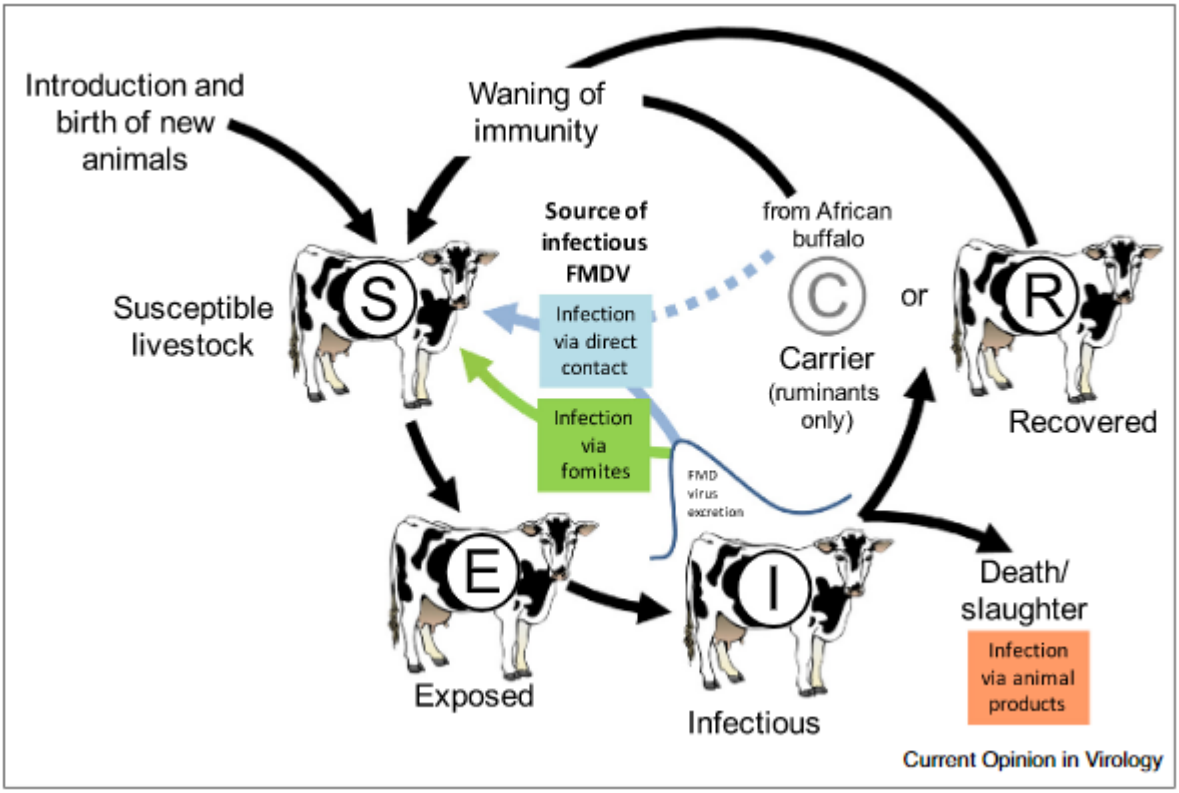

Figure 1.3: Model describing cycles of FMDV replication and transmission in livestock.

1.2 Historical background

Foot and mouth disease (FMD) has been recognised as a significant epidemic disease, threatening the cattle industry since the sixteenth century. In the late nineteenth century, it was shown by Loeffler and Frosch [16] to be caused by a submicroscopic, filterable transmissible agent, smaller than any known bacteria. It was not until 1920 that a convenient animal model for the study of FMD virus was established by Waldmann and Pape [16]. African buffaloes are one of the wildlife animals that have been recognised to transfer foot and mouth disease virus (FMDV) to livestock with serotypes: South African Territories (SAT) 1, 2 and 3 [17]. African buffaloes are known to live in many different environments such as woodlands, grasslands, swamps and flood plains, provided that there is a sufficient supply of water and great quality grazing, as their significant amount of time is spent near or in water [17, 19]. African buffaloes are known to be carriers of the infection of FMD. The diagnosis of FMD in buffaloes is more complicated compared to domestic livestock because FMD is asymptomatic in African buffaloes, which perform as natural reservoirs of the infection in an endemic cycle [17, 20]. It is known that even though cattle can also be carriers of FMD viruses, they are less effective carriers compared to buffaloes [20].

The population of buffaloes in Namibia has been found to increase almost every year in interfaces [21]. For instance, it was reported in 2018 that in Okakarara, at Waterberg plateau park in the Otjozondjupa region, there is an increase in the number of buffaloes [21]. This is a risk when it comes to FMD outbreaks because it appears that due to the high number of buffaloes entering the park, some tend to escape from the park's perimeters [21].

According to the report on FMD in Namibia [15], in 2007 an outbreak of FMD occurred in Caprivi (now renamed to Zambezi) region in the area called Malindi, located east of Katima Mulilo. There, out of a cattle population of close to 45 000, a total of 32 000 heads of cattle were vaccinated. Furthermore, it was confirmed by the Botswana Vaccine Institute's laboratory

that Caprivi region was affected by serotype SAT 2 [22]. Since then, Namibia was free from FMD until another outbreak occurred in 2011 and 2012 in the same region. According to the report by The World Organization for animal health (OIE) in [23], the FMD outbreak occurred in Masikili village in Caprivi region in Namibia, where 37 out of 1510 susceptible cattle were infected. Some control measures, such as control of livestock movements within the country, quarantine, and zoning were applied [15]. The movement of cattle from Masikili village caused an outbreak again in 2012, although the necessary strategy was considered to vaccinate the cattle within a 40 km radius of the primary outbreak [15].

The Namibian veterinary authorities reported in August 2013 [23] that two outbreaks of FMD in Caprivi region affected 706 susceptible animals. According to the OIE report [23], one kraal of 156 cattle was inspected, whereby two cattle had fresh ulcers on their tongues and 11 had old healing ulcerative lesions. In addition, it was reported that the same kraal was last vaccinated for FMD in March 2013, while the outbreak occurred in August 2013. This shows that vaccination is helpful although it needs to be repeated every 6 months. In 2015, OIE reported seven cases of FMD that were thought to be caused by wild species in the Northeast of the Kavango region, and this was the first FMD outbreak in this region since the last one reported in 2009 [24]. Moreover, 2015 was also the same year that the Northern parts of Namibia experienced an outbreak, where 14 cattle out of the susceptible population of 2458 village cattle were found with oral and hoof lesions [24]. In the same way, two outbreaks of FMD occurred in the Caprivi region where 36 cattle from four different kraals were found with lesions that were suspected to be FMD.

The setting of our study interfaced in Namibian communities especially in Zambezi, Kavango regions and Northern parts of Namibia, where buffaloes and cattle interact in one place.

1.3 Economic consequences

Every country, whether developed or not, wants to avoid such diseases like FMD for the safety of the country's economy. Countries worldwide are putting efforts to curb the infection, as it has devastating effects on agriculture and wildlife economies [25, 26]. Furthermore, once FMD is suspected, there will be restricted market access for livestock and livestock products from the affected areas. FMD is, and will remain a serious economic problem in Southern Africa, with the main obstacle to eradication being the presence of large numbers of the principal maintenance host; the African buffalo in border areas between Southern African countries [4].

Namibia exports more than 80% of its livestock and livestock products [27]. Moreover, it was reported in 2016 and 2017 that the export of Namibian products to other countries as well as selling of livestock productions increased [27]. Namibia exports livestock productions to South Africa, Scandinavian countries as well as European Union, in order to bring profit into the country [27]. The agricultural sector is one of the largest employers and supports the Namibian population directly and indirectly. Furthermore, according to the National planning Commission (NPC) [28], Namibia has a goal for vision 2030 to reduce poverty and hunger by creating prosperity and more jobs for the nation. However, for as long as FMD is a threat to the communities, this will be hard to achieve. Baluka in [25] explains that farmers with small and medium herds are the ones affected the most by FMD, as they experienced the most milk losses and suffered higher control costs. Hence, this has became an economic consequence for those countries whose economy depends more on export of livestock [29].

1.4 Foot and mouth disease control mechanisms

At the moment, it is known that there is no cure for FMD [30]. Hence, control mechanisms are required to reduce the spread of the infection. In most cases, strategies (major control mechanisms) that have been used against the spread of the disease from infected to susceptible animals are restrictions on animals movement, especially across the borders, and in addition,

preventing the movement of animal products, quarantine and educating the public about FMD outbreaks. Decreasing the total number of susceptible animals through immunisation (vaccination) has been adopted by many countries [31]. According to a report by the FMD world reference laboratory, all FMD-free countries have practiced vaccination when affected by the outbreaks [32].

There are two basic forms of vaccination, namely: prophylactic vaccination and ring vaccination. Prophylactic vaccination is the type of vaccination given during pre-outbreaks of FMD in order to prevent the introduction of the infection, while ring vaccination is given during an outbreak of FMD and this is carried out in neighboring infected farms [6]. Moreover, the aim of prophylactic vaccination is to prevent the infection before it occurs. Since there are many serotypes (60 subtypes of the FMD virus), vaccination of animals for just one serotype does not guarantee the protection of animals against the other serotypes because the protection will not be complete [6]. Therefore, it is advisable to re-vaccinate approximately after 6 - 12 months as vaccine immunity does not last long.

Another mechanism used to control the spread is culling (slaughtering of infected animals). There are two methods of culling, namely, contagious premises (CP) and infected premises (IP). CP is when slaughtering takes place in farms based on their proximity to infected farms and IP is when slaughtering takes place in the infected farm [6].

Namibia is one of those African countries that put more effort into vaccination even though it is considered to be expensive. Although not all the regions receive vaccines after six months; Kavango and Caprivi regions receive vaccination due to the buffaloes that are in their surroundings, thus cattle need to be always protected. According to the study done by Kitching et al. in [31], FMD vaccines will not prevent the infection if domestic animals, particularly cattle, are already exposed to the existing virus. It is not advisable for livestock to get in contact with the buffaloes as they are known to be carriers and sources of FMD and there is no strategy of

culling or vaccinating the buffaloes.

When the Namibian government officials suspect FMD, they put in place border patrols and roadblocks to try and curb meat and cattle movements. Hence, all cloven-hoofed animals and animal fodder such as grass, hay, straw and firewood are prohibited from passing through roadblocks until the specific region that had been suspected of FMD is declared free from the disease [15]. According to import regulations, an infected area should be closed for at least 6 months from the date it is declared free from the disease and the slaughtering of cattle is stopped as well for the same duration [22].

1.5 Mathematical modeling background on FMD

Mathematical modeling is the art of interpreting problems from an application area into manageable mathematical formulations. Mathematical models are applied to analyse the transmission and control of FMD including the possible control mechanisms to enable better decision making for health policy makers [33].

It all started with Kermack and McKendrick in 1927, when they developed a susceptible-infected-removed (SIR) epidemic model and became famous among researchers [34]. The SIR model is one of the most common models used for modeling infectious diseases including FMD. In 2001, in United Kingdom (UK), mathematical models played a significant role to develop policies in the control of the FMD epidemic. Moreover, since Ferguson, Donnelly and Anderson in [35] developed a mathematical model for UK FMD outbreak, quite a number of researchers took an interest in the mathematical modeling of FMD (see, for instance [1, 6, 10, 36].

Hayama et al. [37] recognised that vaccination is not the only strategy to reduce the epidemic outbreak, by developing a mathematical model for 2010 FMD epidemic in Japan to evaluate the control measures: vaccination and culling. A simulation model was developed on FMD

transmission between cattle and pigs farms in order to generate the disease spread in the affected area. The focus was more on the two types of culling: preemptive and prompt, where their simulation results suggested that prompt culling on infected farms after detection could be a better option to reduce the spread of the infection.

Extensive literature studied show that in Namibia, mathematical modeling was not done on the FMD epidemic, a gap this research will address.

1.6 Statement of the problem

FMD spreads rapidly if no control strategies are implemented on time. Although, according to Aftosa in [11], it is known that FMD is not fatal in healthy adult animals, it is still a risk and a major threat to Namibia and other countries. The problem is that buffaloes are still increasing in the country every year, the infection is still growing and no strategies are in place to control this phenomena, this puts the domestic livestock at risk of FMDV, as there will always be a competition for water and grazing and buffaloes do not die but carry the infection for life. If FMD outbreak occurs, the main export markets for meat and dairy products would stop, and as a result, the country's income would be reduced. Moreover, sometimes the communities or farmers lack the knowledge to identify or to suspect FMD on time and once the damage is already done then it will be too late to handle and vaccinate the susceptible animals. In the Namibian scenario there is no culling taking place only vaccination, but we are going to include culling as a potential control strategy as other countries. The control measures' impact is hampered by the uncontrolled movement and mixing of cattle and buffaloes. Hence, not much attention has been given to these factors in the attempt to understand FMD prevalence in Namibia in order to come up with comprehensive and effective policies to suppress the spread of the infection.

We develop deterministic mathematical models to capture the dynamics of FMD in Namibia

in cattle incorporating buffalo as a reservoir. The same models can be modified to incorporate vaccination and culling when domestic livestock migrate and mix with buffaloes. Ultimately, the predictive potential of these models shall be used to determine the impact of intervention strategies in the control and case management of the disease. Epidemiological modeling helps describe FMD infections and how they spread in the population. Mathematical models help us to study the dynamics of the biological system at disease-free equilibrium point in order to find out whether FMD can be contained or not.

1.7 Aims and objectives of the study

Aim: The aim of the study is to use mathematical models to assess the potential impact of FMD control using vaccination and culling on the spread of FMD on the interface settings of Namibia communal areas and National parks.

The specific objectives are to:

1. Develop and analyse the basic mathematical model of the dynamics of FMD without control measures replicating the FMD infection in the interface setting of Namibia communal areas and National parks;

2. Develop and analyse a mathematical model incorporating vaccination and culling as control measures available in the Namibian setting for the control of FMD;

3. Fit the model with control measures to the yearly cumulative FMD cases in Namibia;

4. Discuss the results and identify the impact of these controls measures on the Namibian scenario.

1.8 Significance of the study

Mathematical modeling of the spread of infectious diseases has become part of veterinary policy decision making in various countries [38]. In a similar manner, Fraser and Grassly

in [39] state that mathematical modeling is the foremost method to use in order to clarify the theories and to make decisions. Furthermore, FMD models have been studied to understand the transmission dynamics of FMD. However, to the best of our knowledge, no mathematical models were ever developed using Namibian data to control FMD. Namibia needs strategies to warn communities that there is a threat of FMD in the country. Additionally, the transmission interactions in a community are complicated and it is hard to understand the enormous size dynamics of the spread of FMD without a suitable structure of mathematical models [39]. Moreover, mathematical models for FMD transmission have been used to interpret observed data on outbreaks in order to make predictions to new situations. Given the above, having well-developed mathematical models, the World Organisation for Animal Health (OIE), the Ministry of Agriculture, Water and Forestry especially in the department of veterinary services and the Ministry of Environment and Tourism, may use the findings for eradication programmes on FMD.

Mathematical models serve as epidemiological modeling tools in epidemiology to understand the predictions and possible control to the spread of infections [34]. Moreover, mathematical models have become invaluable monitoring tools for shedding light on the mechanisms underlying the dynamics of FMD and for quantitative assessment of the effectiveness of different control strategies [1]. The application of mathematical models to FMD surveillance data can be used to discuss both scientific hypotheses and disease-control policy questions [39].

It is important to come up with FMD models that capture the scenario of the spread of FMD in animals, considering the presence of buffalo population in the Namibian context. There will be models that inform policy on the threshold conditions to be attained, and policymakers such as Veterinary offices, public health professions and national parks personnel will benefit. These models will help Namibians living or working on the interface between national parks and surrounding communities to determine the vaccination strategies that can help keep the disease contained. Therefore, this work will contribute to the body of knowledge of mathematical

modeling of FMD in Namibia.

1.9 Outline of the thesis

This chapter has focused more on giving the background, the aims and objectives that were expected to be achieved at the end of the investigation and significance of the study. Chapter 2 reviews the literature that relates to FMD. Chapter 3 gives preliminary concepts which were used for mathematical analysis. In chapter 4 and 5, models were formulated and analysed in detail. Chapter 6 provides numerical simulations of the two models namely: basic model in the absence of vaccination and vaccination model and discussions of the results from the simulation. Lastly, chapter 7 is the discussions of the results, conclusions, strengths and weaknesses of the models and future work to be done.

Chapter 2

LITERATURE REVIEW

2.1 Introduction

This chapter provides contextual information from different researchers on dynamics of FMD models. It focuses more on the mathematical models on FMD in cattle and buffaloes incorporating two major themes; vaccination and culling as control mechanisms of FMD. We shall use or select some studies as our building blocks to our study. Furthermore, this chapter outlines in detail the numerous methods employed in the modeling of FMD. It then gives a detailed review on the vaccination and culling, outlining its benefits and shortcomings and results obtained to identify research gaps entailed this study.

2.2 Literature review

There is no specific treatment for FMD, except controlling and preventing the infection. Hence, researchers have studied the dynamics of FMD transmission to come up with better control measures to reduce the prevalence of the disease in different countries. Thomson [4], wrote an overview of FMD in Southern Africa in 1995 as a historical study, to show that FMD has been there and immunisation has always been recommended as a better tool against FMD. The aim was to investigate the extent and nature of antigenic variation within the Southern

African Territories (SAT) types of FMD. It was further emphasised more on the role of wildlife, especially African buffalo, in the spread of FMD in the community. It was found that even though the cattle are immunised, the benefits were eroded away because of cattle gathering together with wildlife at water points [4]. Thomson's study provided information that was useful as building blocks/information to this study especially on the interaction between cattle and buffalo or the study on SAT virus types. However, they concentrated on experiments and no mathematical analysis was carried out.

The transmission and control of FMD in buffaloes and cattle have been a matter of concern. Jori and Etter [40] developed a stochastic quantitative model to assess the annual risk of FMD transmission from buffalo to cattle herds. Their research was conducted at Kruger National park interface in South Africa based on the ecological and epidemiological data. The aim was to suggest a better control mechanism that so as to reduce the infection in the cattle population and to know how the disease transmits between cattle and buffalo individuals. In their model, it was found that the immunisation of the cattle population is the best option to protect the cattle against FMDV. Moreover, it was found that FMDV is transmitted more when cattle and buffaloes interact within the park especially by sharing water at water sources. This is in contrast with the number of small groups of buffalo that escape from the park and infect the individuals. The authors suggested that without any intervention, the best choice is to avoid cattle herds entering the park to reduce FMD transmission. The study employed the use of stochastic quantitative models, but this study made use of mathematical deterministic models. In addition, although this model has touched more on the importance of vaccination in the interface, this study used it as building blocks on the concept of vaccination and to further investigate other control mechanisms such as culling that is not researched by their study.

Carpenter et al. [9] presented a numerical simulation study of intra-herd transmission of the FMDV. The aim was more on how the FMDV can be diagonosed before and after clinical signs among the cattle herd. Their results showed that FMD would not be diagnosed in the herd until

10 to 13.5 days after the index case cattle had become infected. In addition, the model predicted that after FMDV is diagnosed the number of infectious cattle increased rapidly during the first few days. It was concluded that for as long as there are susceptible enough animals to sustain the transmission of FMDV, the transmission will continue rapidly through the herd. The study suggested that proper biosecurity strategies such as slaughtering infected cattle and separating susceptible animals herd from the infected animals will minimize the transmission of FMD provided early diagnosis is made. Effective biosecurity measures could actually prevent the invasion of farms by the FMDV [9, 11]. Moreover, their study also suggested that intraherd disease dynamics must be estimated before a model can be used to accurately predict FMDV transmission between herds. It was advised that in real-time the diagnostic surveillance that used to detect FMDV is required before clinical signs start to develop. Their study helps us to understand how the transmission of FMD occurs within herds and on diagnosis test since we focused more on the interface and no experiments were conducted. Nevertheless, they did not focus on buffaloes and their study did not include any numerical simulations.

Since the outbreak of FMD occurred in the United Kingdom in the year 2001, several studies on mathematical models have been proposed in order to understand the dynamics of FMD transmission. Tildesley et al. [41] proposed a probabilistic FMD transmission model by investigating the optimal deployment of limited capacity reactive ring vaccination of cattle. The aim was to develop an optimal reactive and responsive vaccination programme based on the 2001 United Kingdom (U.K) epidemic data. In their work, it was suggested that vaccinated animals do not have to be culled. Their results highlighted the significance of reactive vaccination as a tool to control future FMD epidemic. Their work suggested that optimal ring size was highly dependent upon logistical constants although it was more robust to epidemiological parameters. It was further suggested that there are other ways for reactive vaccination to significantly reduce the epidemic size, for instance, not paying more attention to the ordering in which infections are reported and just vaccinating those farms closest to any previously reported case. Tildesley et al. study contributes to our study as building blocks, and

we extend and examine the effects that vaccination and culling have in the Namibian context in order to control the FMD epidemic. We further research and discuss more on these control measures via mathematical modeling for better estimates.

Mushayabasa and Tapedzesa [1] proposed and analysed an optimal control problem where the control system was a mathematical dynamic model for FMD, incorporating vaccination and culling of infectious in animals in Zimbabwe. These mechanisms representing pre- and post-exposure vaccination, culling of symptomatic animals, and culling of infectious non-symptomatic animals. The aim was to investigate using numerical simulations, how these control measures should be implemented to eliminate FMD in the community at a lower cost. Their results from the numerical simulations suggest that vaccination and identifying infectious non-symptomatic animals were the most effective controls during FMD outbreaks. Hence, these two controls must be increased for the whole period of an outbreak. Although in either case they used mathematical models, their conclusions agree with keeling et al. in [10] and Bouma and Orsel in [42] that vaccination is the most effective control when it comes to FMD outbreak. Mushayabasa and Tapedzesa [1] pointed out that the movement of animals, which can be influenced by, among others, seasonal variation, can be considered as a limitation. Another limitation was the assumption that the infection can be transmitted through contact between an infectious and a susceptible animal, while airborne FMDV transmission has been reported as one of the FMDV routes. Therefore, it was recommended that it might be worthwhile to model and analyse mathematically these limitations. Their work provided building blocks to our study as they researched on the control measures that are namely, culling and vaccination. However, it is not clear whether they developed two models for buffaloes and cattle. In their study, though the concept of reducing the intervention and implementation costs were investigated in their study. Our study focused more on the impact of interventions.

Mushayabasa, Bhunu and Dhlamini [36] formulated and analysed a deterministic model to assess the impact of vaccination and culling on controlling FMD in livestock. Their results

demonstrated that vaccination and culling are important on controlling FMD especially if they are both implemented. Furthermore, it was suggested in their work that culling that takes place on infected farms after detection can reduce the disease dynamics fast (as soon as symptoms start to show) [36]. Although it was discussed in [1, 10, 36] that vaccination is one of the FMDV routes, Parida [43] argued that vaccination can prevent clinical disease although the protection is not for a long time. It is only effective six months then re-vaccination of the animals will be required again. Their model focused only on livestock but in our study we would focus on both cattle and buffaloes in the interface.

Ringa and Bauch [6] developed and studied a susceptible-exposed- infectious-recovered-vaccinated-culled (SEIRVC) model as a pair approximation model of FMD transmission in near-endemic populations. Their focus was more on long term dynamics by exploring characteristics of frequent outbreaks of FMD such as loss of natural immunity, dependence on the disease re-importation and vaccine waning. Although these are some characteristics of FMD outbreaks, it was found that the duration of natural immunity is more generally sensitive compared to the duration of vaccine immunity when it comes to the measure and number of FMD outbreaks. However, when it comes to the epidemic, multiple epidemic outbreaks seem to occur if the loss of natural immunity or vaccine waning occur rapidly, and this might make it difficult to eliminate the disease. Ringa and Bauch also touched on the concept of endemicity whereby they tried to give the difference between FMD-endemic settings and FMD free settings. FMD-endemic settings seemed to depend more on factors, such as waning of vaccine immunity, natural immunity and frequent disease re-introduction. Although endemic settings factors such as natural immunity and others are considered to be important, it was argued that they are not commonly used in spatial FMD transmission models. It was from this argument that Ringa and Bauch thought of a pair approximation model to analyse ring and prophylactic vaccination from a SEIRVC model, to explore the impact of the endemic factors using baseline parameters in a fixed population of farms. Their work suggested that optimal long term-term control of FMD by vaccination in near-endemic settings can be attained by using prophylactic vaccines, more especially when

having limited resources. Although Ringa and Bauch in [6] studied more on an epidemic concept, there is a need to fill in the gap of using mathematical models to develop more FMD models in endemic countries to analyse such factors.

In recent years, a mathematical modeling framework was proposed by Mushayabasa, Posny, and Wang [44] to study the intrinsic dynamics of FMD. Biological and ecological factors such as vaccination effects, and seasonal impacts were some of the factors incorporated in their models during the complex interaction among animals. The study conducted both epidemic and endemic analysis, with threshold dynamics characterised by the basic reproduction number. The numerical simulation results were presented to demonstrate the analytic findings. It was found that when the disease transmission rate is very high, vaccination alone may not be sufficient to eradicate FMD in the community. However, the vaccine may have a positive effect on reducing disease risks and lowering progressive FMD cases when an outbreak occurs. Their results suggest that vaccination was still the strong weapon against FMD epidemic even though in some cases it may not be enough and culling might be implemented as authors in [36] suggested. Our study tried to extend their vaccination model by investigating culling as one of the control measures since vaccination alone might not be sufficient to eradicate FMD in the community when the disease transmission rate is very high.

It appears that only a few researchers, for instance, [8], have investigated FMDV using experimental methods. Dekker et al. [45] investigated the rate of FMDV transmission by carriers using published experimental data. Their aim was to quantify the transmission rate of FMDV infection from a carrier to susceptible animals, using a mathematical model. Moreover, the transmission was between buffalo to buffalo, cattle to cattle, buffalo to cattle, cattle to pig as well as sheep to cattle. It was found from their experimental study that the transmission rate of FMDV from carrier to susceptible animals was much lower than the transmission rate estimated during an acute infection of cattle with FMDV. Consequently, the risk of new outbreaks from the introduction of a carrier was clearly much lower than the risk of a new outbreak from the

introduction of an animal after primary infection. Our primary investigation determined whether cattle can be carriers of FMDV and how fast can they spread the virus to other cattle or to buffaloes.

Paton et al. in [8], investigated the transmission of FMDV from experimentally infected Indian buffaloes to in-contact naive and vaccinated cattle and buffaloes. They used the approach of mixing unvaccinated buffaloes and cattle together with buffaloes with FMDV for five days in one place. Susceptible cattle and buffaloes were vaccinated. It took 28 days to see the outcome where animals were divided into six groups with two donor buffaloes with FMDV with four recipient animals, one vaccinated buffalo and one unvaccinated calf. Their results showed that those cattle that were not vaccinated developed clinical signs of FMD compared to those where buffaloes and cattle were vaccinated, because they all became infected with FMDV. In addition, cattle that were vaccinated were protected 100% while buffaloes were only 66.61% protected. Through this experiment, it became known that buffaloes have the potential to spread FMD by direct contact and the best tool to stop this spread is vaccination. These researchers in [8] have done the experiment that most researchers could not do. The number of buffaloes and cattle that were used in their experiment was small. Hence, they could not determine if the differences in clinical protection afforded by vaccination of cattle and buffaloes are significant and warrant a different dose regime.

Akinyemi et al. in [46] built and analysed a deterministic mathematical model for the transmission dynamics of infectious disease with immunity loss and relapse. The threshold $\mathcal{R}_0$ was computed in order to understand the two types of equilibrium namely endemic and disease free equilibrium (DFE). They used Lyapunov function method to analyse the global stability of the two mentioned equilibria namely; the endemic and disease free equilibrium points. The validation of analytical results of the model was done using the stability theory of nonlinear system. Both researchers in [46] and [47] had the same conclusions that, once some infected individuals are introduced into a completely susceptible population and $\mathcal{R}_0 < 1$, then the disease would die out and DFE

is locally asymptotically stable, but if $\mathcal{R}_0 > 1$, then the disease would remain and the epidemic would persist and DFE is unstable [46, 47].

In this chapter, an overview of FMD methods was given, identify the gap and major literature that supports the topic. The next chapter will give some definitions and methods that are needed in conducting this study.

Chapter 3

PRELIMINARIES CONCEPTS

In this chapter, we provide some definitions, examples and theorems about some concepts that are related to mathematical analysis of our models. In this study, dynamical system would be a system of nonlinear ODEs.

Definition 1. *[49] Disease free equilibrium (DFE): is when the disease is not present in the population, and the entire population is susceptible.*

Definition 2. *[49] Endemic equilibrium point: The system has an endemic equilibrium state if there is an existence of the disease in the population without re-introduction of more new infective individuals.*

Definition 3. *[49] Vaccination: Is the administration of generally dead or weakened antigenic material (a vaccine) to produce immunity to a disease.*

Definition 4. *[6] Culling: Slaughtering of the infected animals (farms).*

Definition 5. *[48] Basic reproduction number: the basic reproduction number denoted by $\mathcal{R}_0$ is defined as the expected number of secondary infections produced by one infected individual in a completely susceptible population.*

The basic reproduction number $(\mathcal{R}_0)$ plays the role of a threshold parameter that estimates how severe the epidemic outbreak will be or whether the disease will die out or continue

spreading. If $\mathcal{R}_0 < 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $\mathcal{R}_0 > 1$, then the DFE unstable and invasion is always possible. It is determined by the dominant eigenvalue of the next generation matrix at the DFE.

$\mathcal{R}_0$ helps to determine whether the disease dies out or not. In addition, in order to prevent a disease from becoming endemic it is needed to reduce the basic reproduction number to be below one. The method of $\mathcal{R}_0$ can be used to analyse global stability of equilibrium points of the model. The threshold is often found through the study of the eigenvalues of the Jacobian matrix at the disease equilibrium, hence, this threshold is a reflection of the stability of disease free equilibrium (DFE). Van Driessche and Watmough in [47] studied how to determine the reproduction number $\mathcal{R}_0$ for a general compartmental disease transmission model build on a system of ordinary differential equations. The potential to invade a population by any infectious disease is one of the most concerns in epidemiological models.

Definition 6. *[48] Effective reproductive number $\mathcal{R}_e$ is when transmission occurs in a population that is not entirely susceptible due to implemented control strategies.*

$\mathcal{R}_e$ is determined by the number of susceptible cattle infected by each infected cattle during their infectious period. The aim of control measures is to reduce transmission so that $\mathcal{R}_e$ is reduced below the unity.

Definition 7. *[2] A Jacobian matrix is a matrix of all first order partial derivatives of a vector-valued function.*

In particular, we compute the Jacobian of the system at the disease-free equilibrium, and we pose the condition that all eigenvalues of the corresponding characteristic equation must have negative real parts [49].

For many higher-dimensional models, the Jacobian computed at the disease-free equilibrium cannot be reduced to a $2x2$ matrix. The characteristic polynomial then has degree three or higher. In this case, the reproduction number can be obtained from the constant term. Whether the reproduction number is greater or less than 1 determines the sign of the constant term.

Nonetheless, we still need tools that give necessary and sufficient conditions for the eigenvalues to have negative real parts. These conditions are given by the Routh-Hurwitz criterion, which is stated in the following theorem [49]:

Theorem 1. *Routh-Hurwitz Criteria: Consider the nth-degree polynomial with real constant coefficients.*

Given a polynomial $P(\lambda) = \lambda^n + a_1\lambda^{n-1} + \cdots a_{n-1}\lambda + a_n$.

Define n Hurwitz matrices using the coefficients a_i *of the characteristic polynomial:*

$$H_1 = \begin{pmatrix} a_1 \end{pmatrix}.$$

$$H_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix}.$$

$$H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{pmatrix}.$$

and

$$H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \cdots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & a_n \end{pmatrix}.$$

The Routh-Hurwitz criteria for polynomials are given in the following Table:

Table 3.1: Routh-Hurwitz criteria

n	Coefficient signs	Additional conditions
2	$a_1 > 0, a_2 > 0$	-
3	$a_1 > 0, a_2 > 0, a_3 > 0$	$a_1 a_2 > a_3$
4	$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0$	$a_1 a_2 a_3 > a_3^2 + a_1^2 a_4$
5	$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0,$ $a_5 > 0$	$a_1 a_2 a_3 > a_3^2 + a_1^2 a_4,$ $(a_1 a_4 - a_5)(a_1 a_2 a_3 - a_3^2 - a_1^2 a_4) > a_5 (a_1 a_2 - a_3)^2 + a_1 a_5^2$

The condition for the roots of the polynomial $P(\lambda)$ to be negative or have negative real part is that all the coefficients be strictly positive as stated in the Table 3.1.

[50] Given a dynamical system of the form:

$$\dot{X}(t) = f(X(t), t). \tag{3.1}$$

We then give the definitions to the following concepts as defined by Lungu, Kgosimone and Nyabadza.

Definition 8. *[50] Equilibrium points: A vector $\bar{X}$ is an equilibrium point for a dynamical system* (3.1) *if once the state vector is equal to $\bar{X}$ it remains equal to $\bar{X}$ for all future time. Example; if*

$$\dot{X}(t) = f(X(t), t).$$

then an equilibrium point is a state $\bar{X}$ satisfying $f(\bar{X}, t) = 0$ for all time t.

Suppose $\bar{X}$ is an equilibrium point of a time-invariant system i.e. $\bar{X}$ is an equilibrium point of $\dot{X}(t) = f(X(t))$:

For a precise definition of stability, it is convenient to introduce the notation $S(\bar{X}, R)$ to denote a spherical region in the state space with center at $\bar{X}$ and radius $\mathcal{R}$:

Definition 9. *[50] An equilibrium point $\bar{X}$ is stable if there exists $\mathcal{R}_0 > 0$ for which the following*

is true. For every $\mathcal{R} < \mathcal{R}_0$, there exists r, $0 < r < \mathcal{R}$; such that if $X(0)$ is inside $S(\bar{X}, r)$, then $X(t)$ is inside $S(\bar{X}, \mathcal{R})$ for all $t > 0$.

In simple explanation this means that an equilibrium point is stable whenever the system state is initiated near that point, the state remains near it, perhaps even tending towards the equilibrium point as time increases.

Definition 10. *[50] An equilibrium point $\bar{X}$ is asymptotically stable if whenever it is stable and in addition there exists $\bar{\mathcal{R}}_0 > 0$ such that whenever the state is initiated inside $S(\bar{X}, \bar{\mathcal{R}}_0)$, it tends to $\bar{X}$ as time increases.*

Definition 11. *[50] An equilibrium point $\bar{X}$ is marginally stable if it is stable but not asymptotically stable.*

Definition 12. *[50] An equilibrium point $\bar{X}$ is unstable if it is not stable. Equivalently, $\bar{X}$ is unstable if for some $\mathcal{R} > 0$ and any $r > 0$ there is a point in the spherical region $S(\bar{X}, r)$ such that if initiated there, the system state will eventually move outside of $S(\bar{X}, \mathcal{R})$.*

Definition 13. *[50] An equilibrium point is called globally stable if it is stable for almost all initial conditions, not just those that are close to it.*

[49] An equilibrium point that is locally stable may be globally stable if there are no other locally stable equilibria coexisting with it.

[50] The method of Liapunov functions enables the analysis to be extended beyond only a small region near the equilibrium point (global analysis). The basic idea of this technique for verifying stability is to seek an aggregated summarising function that continually decreases towards a minimum as the system evolves.
Suppose that $\bar{x}$ is an equilibrium point of a given dynamical system. A Liapunov function for the system and the equilibrium point $\bar{x}$ is a real valued function V; which is defined over a region Ω of the state space that contains $\bar{x}$ and satisfies the three requirements:

1. V is continuous.

2. $V(x)$ has a unique minimum at $\bar{x}$ with respect to all other points in Ω.

3. Along any trajectory of the system contained in Ω, the value of V never increases.

Consider the system,

$$\dot{x}(t) = f(x(t)). \tag{3.2}$$

together with a given equilibrium point $\bar{x}$: In the time continuous case the requirement that the value of a Liapunov function never increases along a trajectory is expressed in terms of the time derivative.

Suppose $x(t)$ is a trajectory. Then $V(x(t))$ represents the corresponding value of $V(x(t))$ along the trajectory. In order for V not to increase, we require that $\dot{V}(x(t)) \leq 0$ for all t.

Definition 14. *[50] A function V defined on a region Ω of the state space and containing $\bar{x}$ is a Liapunov function if it satisfies the following three requirements:*
1. V is continuous and has continuous first partial derivatives.
2. $V(x)$ has a unique minimum at $\bar{x}$ with respect to all other points in Ω.
3. The function $\dot{V}(x) = \nabla V(x) f(x)$ satisfies $\dot{V}(x) \leq 0$ for all $x(t)$ in Ω.

Theorem 2. *[50] If there exists a Liapunov function $V(x)$; then the equilibrium point $\bar{x}$ is stable. If, furthermore, the function $\dot{V}(x)$ is strictly negative for every point then the stability is asymptotic.*

Definition 15. *[51] Let I be a set and let $T : I \to I$ be a function that maps I into itself. Such a function is often called an operator, a transformation, or a transform on I. A fixed point of f is an element I_i of I. for which $f(I) = I_i$.*

We use the fixed point theory to obtain the existence of the endemic equilibria. Given that $f(I_i)$ is the disease incidence for stage $i, (i = 1, 2, 3, ..., n)$ then I_i is the infection class. Moreover, the non-linear function $f(I_i)$ $(i = 1, 2, 3, ..., n))$ is assumed to satisfy the following assumptions:

1. $f(0) = 0$,
2. $f'(\mathrm{x}) > 0$,
3. $f''(x) < 0$,
4. $\lim_{x \to +\infty} f(x) = C < +\infty$.

In this case f, is increasing, bounded and convergent with no change of convexity on a finite interval [52].

In this chapter, concepts that will be used in the model analysis have been defined and a detailed explanation on how to calculate them has been provided. We use these theorems and explanation in this chapter as building blocks to the formulation of FMD models in the next chapters.

Chapter 4

Basic model formulation and analysis

We formulate a basic deterministic SEIR model to study the transmission of FMD. This model captures the interaction between infected and susceptible animals. The model is explained with the help of the flow diagrams to understand how FMD progress through various stages. We then analyse the model.

4.1 Basic model formulation

For the basic model, we consider the transmission of FMD between cattle and buffaloes in the absence of vaccination and culling as the control measures. The population size of cattle and buffaloes is assumed to be constant. The model is designed by dividing the total populations of cattle, $N_C(t)$ and buffaloes, $N_B(t)$, at time t, into mutually-exclusive classes of susceptible (S_C, S_B), exposed (E_C, E_B), infected (I_C, I_B) and recovered R_C where the subscripts C and B denote cattle and buffaloes, respectively.

1. The susceptible class is a class of animals who are free of the disease but at risk of catching it.

2. The exposed class is a class of those animals that are infected with the disease but do not show clinical symptoms of the disease yet (infected but not infectious).

3. The infected class is a class of symptomatic infected (and infectious: clinical or sub-clinical) animals.

4. The recovered class is the class of animals that have recovered from the disease with temporary immunity.

4.1.1 Cattle population model

The cattle population is divided into four classes, namely: susceptible, exposed, infectious and recovered which are denoted by S_C, E_C, I_C and R_C, respectively, such that

$$N_C(t) = S_C(t) + E_C(t) + I_C(t) + R_C(t).$$

The population of susceptible cattle is assumed to increase via recruitment at a rate η_C. It is then decreased by infection at a rate β_C via effective contacts with infected cattle and infected buffaloes, respectively. The modification parameter, τ_1 compares the difference in infection from cattle to cattle and from buffaloes to cattle (infected), where τ_1 is a constant taking values in the interval [0,1). If $\tau_1 < 1$ this means that more infections is coming from the cattle than from the buffaloes. Susceptible cattle suffer natural death at a rate μ_1. Similarly, the class also gains from the recovery of the infected cattle at a rate σ_1. Thus, the rate of change of the susceptible population is given by:

$$\frac{dS_C}{dt} = \eta_C - \mu_1 S_C - \beta_C(\tau_1 I_B + I_C)S_C + \sigma_1 R_C. \tag{4.1}$$

Susceptible cattle are infected with FMD through direct contact with infectious animals (cattle and buffaloes) through forces of infection $\beta_C \tau_1 I_B$ and $\beta_C I_C$. This population of exposed cattle is decreased by progression of individuals to the infected class at a rate α_1 and natural death at a rate μ_1, and this gives us the following equation:

$$\frac{dE_C}{dt} = \beta_C(\tau_1 I_B + I_C)S_C - (\mu_1 + \alpha_1)E_C. \tag{4.2}$$

The population of infected cattle is produced when exposed cattle develop symptoms at a rate α_1. This population is assumed to decrease due to cattle recovery at a rate of ϕ_1, natural death at a rate μ_1 and disease-induced death at a rate ρ_1. This gives

$$\frac{dI_C}{dt} = \alpha_1 E_C - (\mu_1 + \rho_1 + \phi_1)I_C. \tag{4.3}$$

The population of recovered cattle decreases by natural death at a rate μ_1 and loss of acquired immunity (recovered cattle become sources again) at the rate σ_1. Here we assume that recovered cattle do not acquire permanent immunity against re-infection. The model equation is given by:

$$\frac{dR_C}{dt} = \phi_1 I_C - (\mu_1 + \sigma_1)R_C. \tag{4.4}$$

Therefore, combining the equations (4.1), (4.2), (4.3), (4.4) yield the following system of nonlinear ODEs (a flow diagram of the model is depicted in Figure 4.1).

$$\begin{aligned}
\frac{dS_C}{dt} &= \eta_C - \mu_1 S_C - \beta_C(\tau_1 I_B + I_C)S_C + \sigma_1 R_C, \\
\frac{dE_C}{dt} &= \beta_C(\tau_1 I_B + I_C)S_C - (\mu_1 + \alpha_1)E_C, \\
\frac{dI_C}{dt} &= \alpha_1 E_C - (\mu_1 + \rho_1 + \phi_1)I_C, \\
\frac{dR_C}{dt} &= \phi_1 I_C - (\mu_1 + \sigma_1)R_C.
\end{aligned} \tag{4.5}$$

The system of non-linear ODEs given by equation (4.5) represents the basic model for the transmission of a FMD infection in the cattle population.

4.1.2 Buffalo population model

The buffaloes population is divided into three classes: susceptible, exposed and infectious, denoted by S_B, E_B and I_B, respectively. Thus, the total population is given by:

$$N_B(t) = S_B(t) + E_B(t) + I_B(t).$$

The population of susceptible buffaloes is assumed to increase via recruitment at a rate η_B. It is decreased by infection at rate β_B via effective contacts with infected cattle and infected buffaloes. It is then decreased by infection at a rate β_B via effective contacts with infected cattle and infected buffaloes, respectively. The modification parameter, τ_2 compares the difference in infection from cattle to buffaloes and from buffaloes to buffaloes (infected), where τ_2 is a constant taking values in the interval [0,1). If $\tau_2 < 1$ this means that more infections is coming from the buffaloes than from the cattle.. Susceptible buffaloes die naturally at a rate μ_2. Hence, the rate of change of the susceptible population is given by:

$$\frac{dS_B}{dt} = \eta_B - \mu_2 S_B - \beta_B(\tau_2 I_C + I_B)S_B. \tag{4.6}$$

Susceptible buffaloes infected by cattle and buffaloes with forces of infection $\beta_B\tau_2 I_C$ and $\beta_B I_B$ becomes sources for exposed buffaloes. The exposed buffaloes population decrease by progressing into the infectious buffalo population at a rate α_2 and by natural death at a rate μ_2, to give:

$$\frac{dE_B}{dt} = \beta_B(\tau_2 I_C + I_B)S_B - (\mu_2 + \alpha_2)E_B. \tag{4.7}$$

The infected buffalo population decreases due to natural death at the rate μ_2. In the buffaloes population, it is assumed that there will be no recovered class since buffaloes are carriers of the disease and can carry the virus over a long period of time [40]. Moreover, no disease related mortality is assumed. Therefore, the rate of change of the infected population is given by:

$$\frac{dI_B}{dt} = \alpha_2 E_B - \mu_2 I_B. \tag{4.8}$$

Hence, equations (4.6), (4.7), (4.8) yield the following deterministic system of ODEs:

$$\begin{aligned}
\frac{dS_B}{dt} &= \eta_B - \mu_2 S_B - \beta_B(\tau_2 I_C + I_B)S_B, \\
\frac{dE_B}{dt} &= \beta_B(\tau_2 I_C + I_B)S_B - (\mu_2 + \alpha_2)E_B, \\
\frac{dI_B}{dt} &= \alpha_2 E_B - \mu_2 I_B.
\end{aligned} \tag{4.9}$$

Now letting the forces of infection be;

$$\lambda_1 = \beta_C(\tau_1 I_B + I_C),$$

$$\lambda_2 = \beta_B(\tau_2 I_C + I_B).$$

The systems of ODEs (4.5) and (4.9) result in the following basic model, describing the FMD dynamics in the two populations (as illustrated in Figure 4.1):

$$\begin{aligned}
\frac{dS_C}{dt} &= \eta_C - (\mu_1 + \lambda_1)S_C + \sigma_1 R_C, \\
\frac{dE_C}{dt} &= \lambda_1 S_C - (\mu_1 + \alpha_1)E_C, \\
\frac{dI_C}{dt} &= \alpha_1 E_C - (\mu_1 + \rho_1 + \phi_1)I_C, \\
\frac{dR_C}{dt} &= \phi_1 I_C - (\mu_1 + \sigma_1)R_C, \\
\frac{dS_B}{dt} &= \eta_B - (\mu_2 + \lambda_2)S_B, \\
\frac{dE_B}{dt} &= \lambda_2 S_B - (\mu_2 + \alpha_2)E_B, \\
\frac{dI_B}{dt} &= \alpha_2 E_B - \mu_2 I_B.
\end{aligned} \tag{4.10}$$

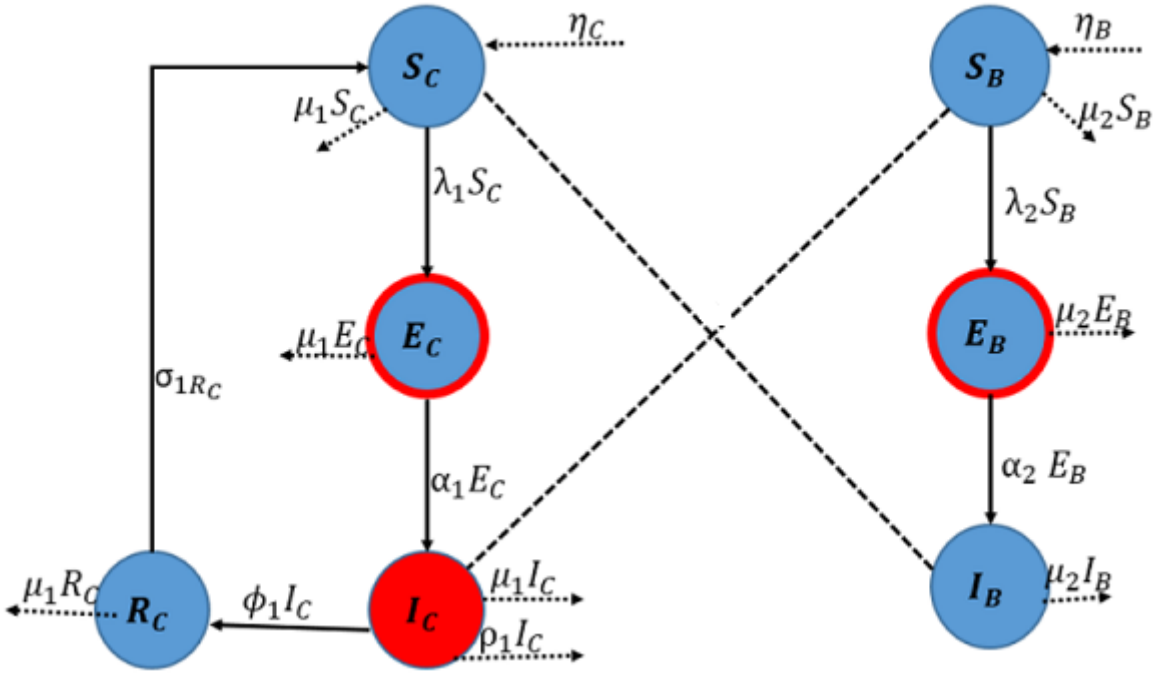

Figure 4.1: Basic model for FMD in cattle and buffaloes populations.

4.2 Analysis of basic model

We first determine the well-posedness of the model by checking the positivity as well as boundedness of the solutions: $S(t)$, $E(t)$, $I(t)$ and $R(t)$. These are necessary for biological meaningfulness of our models.

4.2.1 Positive invariance of the model (4.10)

Since we know that our populations do not need to be negative, it is vital to show that our model is epidemiologically meaningful by establishing that all state variables remain non-negative such that the solutions of the systems of equations with positive initial conditions remain positive for all $t > 0$. The region is called the biologically feasible region where the model will be biologically meaningful. Therefore, we will show this by giving a theorem and the proof of positivity of solutions as given by Mayanja et al. in [53].

Theorem 3. *(Positivity of solutions): Let the initial values of the variables of the system of*

equations (4.10) *be nonnegative, the solutions* $S_C(t)$, $E_C(t)$, $I_C(t)$, $R_C(t)$, $S_B(t)$, $E_B(t)$ *and* $I_B(t)$ *of the system* (4.10) *are nonnegative for* $t \geq 0$.

Proof. We prove that each solution components $S_C(t)$, $E_C(t)$, $I_C(t)$, $R_C(t)$, $S_B(t)$, $E_B(t)$ and $I_B(t)$ remain non-negative. Otherwise, if by contradiction: we assume that there exists a first time t_1 such that $S_C(t_1) = 0$, $S_C'(t_1) < 0$ and $E_C(t) > 0, I_C(t) > 0, R_C(t) > 0, S_B(t) > 0, E_B(t) > 0, I_B(t) > 0$ for $0 < t < t_1$.

From the first equation in (4.1),

$$\frac{dS_C(t_1)}{dt} = \eta_C - \mu_1 S_C(t_1) - \beta_C(\tau_1 I_B + I_C)S_C(t_1) + \sigma_1 R_C(t_1). \tag{4.11}$$

Since $S_C(t_1) = 0$ and $R_C(t) > 0$ from our assumptions then,

$$\frac{dS_C}{dt}(t_1) = \eta_C + \sigma_1 R_C(t_1) > 0.$$

Since we assumed $S_C'(t_1) < 0$ then this is a contradiction and consequently, $S_C(t) \not< 0$.

Therefore, $S_C(t) > 0$.

Suppose that $\exists$ a first time t_2 such that $E_C(t_2) = 0$, $E_C'(t_2) < 0$ and $S_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$. for $0 < t < t_2$.

Now given,

$$\frac{dE_C(t_2)}{dt} = \beta_C(\tau_1 I_B + I_C)S_C(t_2) - (\mu_1 + \alpha_1)E_C(t_2), \tag{4.12}$$

Since from our assumptions $E_C(t_2) = 0$ and $S_C(t), I_C(t), I_B(t) > 0$ then,

$$\frac{dE_C(t_2)}{dt} = \beta_C(\tau_1 I_B + I_C)S_C(t_2) > 0,$$

which is a contradiction and consequently, $E_C(t) \not< 0$.

Therefore, $E_C(t) > 0, \forall\, t \in (0, t_2)$.

Suppose that $\exists$ a first time t_3 such that $I_C(t_3) = 0$, $I_C'(t_3) < 0$ and $S_C(t) > 0$, $E_C(t) > 0$, $R_C(t) > 0$,

$S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$, for $0 < t < t_3$.

Now given,

$$\frac{dI_C(t_3)}{dt} = \alpha_1 E_C(t_3) - (\mu_1 + \rho_1 + \phi_1) I_C(t_3), \tag{4.13}$$

Since from our assumptions $I_C(t) = 0$ and $E_C(t) > 0$ then,

$$\frac{dI_C(t_3)}{dt} = \alpha_1 E_C(t_3) > 0,$$

which is a contradiction, hence $I_C(t) \not< 0$.

Therefore, $I_C(t) > 0$, $\forall\, t \in (0, t_3)$.

Suppose that $\exists$ a first time t_4 such that $R_C(t_4) = 0$, $R_C'(t_4) < 0$ and $S_C(t) > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$, for $0 < t < t_4$.

Now considering,

$$\frac{dR_C}{dt}(t_4) = \phi_1 I_C(t_4) - (\mu_1 + \sigma_1) R_C(t_4), \tag{4.14}$$

Since from our assumptions $R_C(t_4) = 0$ and $I_C(t_4) > 0$ then,

$$\frac{dR_C(t_4)}{dt} = \phi_1 I_C(t_4) > 0,$$

which is a contradiction, hence $R_C(t) \not< 0$.

Therefore, $R_C(t) > 0$, $\forall\, t \in (0, t_4)$.

We assume that there exists a first time t_5 such that $S_B(t_5) = 0$, $S_B'(t_5) < 0$ and $S_C(t) > 0, E_C(t) > 0, I_C(t) > 0, R_C(t) > 0, E_B(t) > 0, I_B(t) > 0$, for $0 < t < t_5$.

Given

$$\frac{dS_B}{dt}(t_5) = \eta_B - \mu_2 S_B(t_5) - \beta_B(\tau_2 I_C + I_B) S_B(t_5), \tag{4.15}$$

Since $S_B(t_5) = 0$ from our assumptions then,

$$\frac{dS_B(t_5)}{dt} = \eta_B > 0.$$

Since we assumed $\frac{dS_B}{dt}(t_5) < 0$ then this contradict to our assumption. Hence $S_B(t) \not< 0$ in $(0, t_5)$. Therefore, $S_B(t) > 0$.

Suppose that $\exists$ a first time t_6 such that $E_B(t_6) = 0$, $E_B'(t_6) < 0$ and $S_C(t) > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $I_B(t) > 0$. for $0 < t < t_6$.

Now given,

$$\frac{dE_B(t_6)}{dt} = \beta_B(\tau_2 I_C + I_B)S_B(t_6) - \mu_2 E_B(t_6) + \alpha_2)E_B(t_6), \tag{4.16}$$

Since from our assumptions $E_B(t_6) = 0$ and $S_B(t), I_C(t), I_B(t) > 0$ then,

$$\frac{dE_B(t_6)}{dt} = \beta_B(\tau_2 I_C)S_B(t_6) > 0,$$

which is a contradiction and consequently, $E_B(t) \not< 0$. Therefore, $E_B(t) > 0$, $\forall\, t \in (0, t_6)$.

Suppose that $\exists$ a first time t_7 such that $I_B(t_7) = 0$, $I_B'(t_7) < 0$ and $S_C(t) > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$. for $0 < t < t_7$.

Now given,

$$\frac{dI_B}{dt}(t_7) = \alpha_2 E_B(t_7) - \mu_2 I_B(t_7), \tag{4.17}$$

Since from our assumptions $I_B(t_7) = 0$ and $E_B(t) > 0$ then,

$$\frac{dI_B}{dt}(t_7) = \alpha_2 E_B(t_7) > 0,$$

which is a contradiction, hence $I_B(t) \not< 0$.

Therefore, $I_B(t)$ remain positive $\forall\, t \in (0, t_7)$. □

Therefore, the solutions of the system are nonnegative whenever $t \geq 0$.

4.2.2 Boundedness of solutions of the model

Lemma 1. *All solutions* $S_C(t), E_C(t), I_C(t), R_C(t), S_B(t), E_B(t), I_B(t) > 0$ *are bounded for all* $t \geq 0$.

Proof. Having assured that we are dealing with positive solutions in Ω, we can now prove that for all $t > 0$, $S_C(t), E_C(t), I_C(t), R_C(t), S_B(t), E_B(t), I_B(t) > 0$ will be bounded above. Model (4.10) is split into two; the cattle population and the buffaloes population dynamics.

Cattle population

Given the total cattle population $N_C = S_C + E_C + I_C + R_C$. By adding the first four equations of the model (4.10) we get,

$$\frac{dN_C}{dt} = \eta_C - \mu_1 N_C - \rho_1 I_C \leq \eta_C - \mu_1 N_C.$$

Now by integrating both sides using the integrating factor,

$$N_C \leq (N_C(0) - \frac{\eta_C}{\mu_1})e^{-\mu_1 t} + \frac{\eta_C}{\mu_1},$$

Taking the limit supremum of N_C as $t \to \infty$

$$\limsup_{t\to\infty} N_C \leq \frac{\eta_C}{\mu_1},$$

We obtain that $S_C(t) \leq \frac{\eta_C}{\mu_1}, E_C(t) \leq \frac{\eta_C}{\mu_1}, I_C(t) \leq \frac{\eta_C}{\mu_1}, R_C(t) \leq \frac{\eta_C}{\mu_1}$.

Since all state variables are positive and bounded above, therefore the feasible region for the cattle dynamics is given by

$$\Omega_C = \{(S_C(t), E_C(t), I_C(t), R_C(t)) \in \mathbb{R}^4_+ | 0 \leq N_C(t) \leq \frac{\eta_C}{\mu_1}\}.'$$

Buffaloes population

Using the same analysis used in the model for cattle population, we have the following results;

Given the total buffaloes population

$$N_B = S_B + E_B + I_B.$$

By adding the right hand side of the last three equations of the model (4.10) such that;

$$\frac{dN_B}{dt} = \eta_B - \mu_2 N_B.$$

Now by integrating both sides using the integrating factor we get,

$$N_B = \left(N_B(0) - \frac{\eta_B}{\mu_2}\right) e^{-\mu_2 t} + \frac{\eta_B}{\mu_2}.$$

as

$$t \to 0, N_B \to N_B(0),$$

as

$$t \to \infty, N_B \to \frac{\eta_B}{\mu_2},$$

Thus,

$$N_B \leq \min\{N_B(0), \frac{\eta_B}{\mu_2}\}.$$

The feasible region in the buffalo population is,

$$\Omega_B = \{(S_B, E_B, I_B), \in \mathbb{R}^3_+; 0 \leq N_B(t) \leq \min\{N_B(0), \frac{\eta_B}{\mu_2}\}.$$

This proves that the set $(S_B(t), E_B(t), I_B(t))$ is bounded above by $\frac{\eta_B}{\mu_2}$ and bounded below by 0.

Therefore, Ω_B is positively invariant and the buffaloes model system.

Thus, $\Omega = \Omega_C \times \Omega_B$ is the feasible region for the combined cattle and buffaloes dynamics. □

4.3 Local and global stability of the disease-free equilibrium

4.3.1 Disease free equilibrium point

Disease free is defined to be when the entire population is susceptible. Disease free equilibrium (DFE) is the equilibrium point which occurs when there are no exposed E_C or E_B, infections I_C or I_B and recovery $\mathbb{R}_C$. We then find the equilibrium point of the system of equations (4.10) as follows:

$$\begin{aligned}
\eta_C - \mu_1 S_C^* - \lambda_1^* S_C^* + \sigma_1 R_C^* &= 0, \\
\lambda_1^* S_C^* - (\mu_1 + \alpha_1) E_C^* &= 0, \\
\alpha_1 E_C^* - (\mu_1 + \rho_1 + \phi_1) I_C^* &= 0, \\
\phi_1 I_C^* - (\mu_1 + \sigma_1) R_C^* &= 0, \\
\eta_B - \mu_2 S_B^* - \lambda_2^* S_B^* &= 0, \\
\lambda_2^* S_B^* - (\mu_2 + \alpha_2) E_B^* &= 0, \\
\alpha_2 E_B^* - \mu_2 I_B^* &= 0.
\end{aligned} \tag{4.18}$$

Considering

$$E_C^* = E_B^* = I_C^* = I_B^* = R_C^* = 0,$$

, at DFE point, $S_C^* = S_C^0$ and $S_B^* = S_B^0$ as follow: DFE point of the system of equations which is denoted by E_0 is given by

$$E_0 = (S_C^*, E_C^*, I_C^*, R_C^*, S_B^*, E_B^*, I_B^*) = (S_C^0, 0, 0, 0, S_B^0, 0, 0),$$

where $S_C^0 = \frac{\eta_C}{\mu_1}$ and $S_B^0 = \frac{\eta_B}{\mu_2}$

The DFE's stability is discussed using the concept of basic reproduction ratio which is defined in terms of spectral radius of the next generation matrix as below:

4.3.2 The basic reproduction number $\mathcal{R}_0$ for the basic model 4.10

One of the most important parameters that discuss the conditions under which the infection is cleared or persists in a population is the basic reproduction number denoted by $\mathcal{R}_0$.

The DFE can be analysed by using the next generation method on the existing system in the form of matrices FV^{-1}, where F (the rate at which infected individuals in compartmental produce new infections) should be non-negative and V (the average length time the individual will spend in the community) non-singular [47, 48, 55].
We consider a matrix $\mathscr{F}_i$, the rate of appearances of new infections in compartment i.

$$\mathscr{F} = \begin{bmatrix} \beta_C(\tau_1 I_B + I_C)S_C \\ 0 \\ \beta_B(\tau_2 I_C + I_B)S_B \\ 0 \end{bmatrix}.$$

The Jacobian matrix of $\mathscr{F}$, for disease free equilibrium is given by,

$$F = \begin{bmatrix} 0 & \beta_C S_C^0 & 0 & \tau_1\beta_C S_C^0 \\ 0 & 0 & 0 & 0 \\ 0 & \tau_2\beta_B S_B^0 & 0 & \beta_B S_B^0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

We consider the transition matrix $\mathscr{V}$,

$$\mathscr{V} = \begin{bmatrix} (\mu_1 + \alpha_1)E_C \\ (\mu_1 + \rho_1 + \phi_1)I_C - \alpha_1 E_C \\ (\mu_2 + \alpha_2)E_B \\ \mu_2 I_B - \alpha_2 E_B \end{bmatrix}.$$

Let $v_1 = \mu_1 + \alpha_1, v_2 = \mu_1 + \rho_1 + \phi_1, v_3 = \mu_2 + \alpha_2$.

The Jacobian matrix of $\mathscr{V}$ evaluated at the disease free equilibrium point E_0 is given by,

$$V = \begin{pmatrix} v_1 & 0 & 0 & 0 \\ -\alpha_1 & v_2 & 0 & 0 \\ 0 & 0 & v_3 & 0 \\ 0 & 0 & -\alpha_2 & \mu_2 \end{pmatrix}.$$

The inverse of V is therefore found as:

$$V^{-1} = \begin{pmatrix} \frac{1}{v_1} & 0 & 0 & 0 \\ \frac{\alpha_1}{v_1 v_2} & \frac{1}{v_2} & 0 & 0 \\ 0 & 0 & \frac{1}{v_3} & 0 \\ 0 & 0 & \frac{\alpha_2}{v_3\mu_2} & \frac{1}{\mu_2} \end{pmatrix}.$$

Hence, the next generation matrix denoted by FV^{-1} is given by:

$$FV^{-1} = \begin{pmatrix} 0 & \beta_C S_C^0 & 0 & \tau_1\beta_C S_C^0 \\ 0 & 0 & 0 & 0 \\ 0 & \tau_2\beta_B S_B^0 & 0 & \beta_B S_B^0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{v_1} & 0 & 0 & 0 \\ \frac{\alpha_1}{v_1 v_2} & \frac{1}{v_2} & 0 & 0 \\ 0 & 0 & \frac{1}{v_3} & 0 \\ 0 & 0 & \frac{\alpha_2}{v_3\mu_2} & \frac{1}{\mu_2} \end{pmatrix},$$

$$= \begin{pmatrix} \frac{\beta_C S_C^0 \alpha_1}{v_1 v_2} & \frac{\beta_C S_C^0}{v_2} & \frac{\tau_1\beta_C S_C^0 \alpha_2}{v_3\mu_2} & \frac{\tau_1\beta_C S_C^0}{\mu_2} \\ 0 & 0 & 0 & 0 \\ \frac{\tau_2\beta_B S_B^0 \alpha_1}{v_1 v_2} & \frac{\tau_2\beta_B S_B^0}{v_2} & \frac{\beta_B S_B^0 \alpha_2}{v_3\mu_2} & \frac{\beta_B S_B^0}{\mu_2} \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

Let

$b_1 = \frac{\beta_C S_C^0 \alpha_1}{v_1 v_2}$, $b_2 = \frac{\beta_C S_C^0}{v_2}$, $b_3 = \frac{\tau_1\beta_C S_C^0 \alpha_2}{v_3\mu_2}$, $b_4 = \frac{\tau_1\beta_C S_C^0}{\mu_2}$,
$b_5 = \frac{\tau_2\beta_B S_B^0 \alpha_1}{v_1 v_2}$, $b_6 = \frac{\tau_2\beta_B S_B^0}{v_2}$, $b_7 = \frac{\beta_B S_B^0 \alpha_2}{v_3\mu_2}$ and $b_8 = \frac{\beta_B S_B^0}{\mu_2}$.

Finding the eigenvalues of the matrix:

$$FV^{-1} - I\lambda = \begin{bmatrix} b_1 - \lambda & b_2 & b_3 & b_4 \\ 0 & 0-\lambda & 0 & 0 \\ b_5 & b_6 & b_7 - \lambda & b_8 \\ 0 & 0 & 0 & 0-\lambda \end{bmatrix}.$$

The characteristic equation is given as:

$$-\lambda^4 + (b_1 + b_7)\lambda^3 - (b_1 b_7 - b_3 b_5)\lambda^2 = 0.$$

The eigenvalues are found as;

$$0, 0, \frac{1}{2}\left(-\sqrt{b_1^2 - 2b_1 b_7 + 4b_3 b_5 + b_7^2} + b_1 + b_7\right), \frac{1}{2}\left(\sqrt{b_1^2 - 2b_1 b_7 + 4b_3 b_5 + b_7^2} + b_1 + b_7\right).$$

$$\implies \lambda_1 = 0, \lambda_2 = 0, \lambda_3 = \frac{1}{2}\left[(b_1 + b_7) + \sqrt{(b_1 + b_7)^2 - 4(b_1 b_7 - b_3 b_5)}\right],$$

$$\lambda_4 = \frac{1}{2}\left[(b_1 + b_7) - \sqrt{(b_1 + b_7)^2 - 4(b_1 b_7 - b_3 b_5)}\right].$$

Therefore, the basic reproduction ratio which is the spectral radius of the next generation matrix, $\mathcal{R}_0 = \rho(FV^{-1})$ is given by:

$$\mathcal{R}_0 = \frac{1}{2}\left[(b_1 + b_7) + \sqrt{(b_1 + b_7)^2 - 4(b_1 b_7 - b_3 b_5)}\right].$$

$$= \frac{1}{2}\left[(b_1 + b_7) + \sqrt{(b_1 - b_7)^2 + 4b_3 b_5)}\right].$$

4.3.3 Local stability analysis

In this section, since we analyse the reproduction number using the next generation matrix method, then the DFE is local asymptotically stable. Hence, the following lemma is ensured.

Lemma 2. *If $\mathcal{R}_0 < 1$, then the disease-free equilibrium E_0 is locally asymptotically stable; if $mathcalR_0 > 1$, then the disease-free equilibrium E_0 is unstable.*

-

4.3.4 The global asymptotic stability of the disease free equilibrium

Using linear stability analysis, we can determine the stability of each equilibrium.

Theorem 4. *The disease free equilibrium E_0 of the system* (4.10) *is globally asymptotically in the positive invariant region Ω if $R_0 \leq 1$. Where Ω is the feasible region.*

Proof. In this proof, we are using the concept of Liapunov function.
Consider a Liapunov function as follows:

$$L_0 = c_1 E_C + c_2 I_C + c_3 R_C + c_4 E_B + c_5 I_B,$$

Where c_1, c_2, c_3, c_4 and c_5 are positive constants to be determined.
Differentiating L_0 with respect to time t along the solutions of the model we get,

$$\dot{L_0} = c_1 \dot{E_C} + c_2 \dot{I_C} + c_3 \dot{R_C} + c_4 \dot{E_B} + c_5 \dot{I_B},$$

Let $u_1 = \mu_1 + \alpha_1, \quad u_2 = \mu_1 + \rho_1 + \phi_1, \quad u_3 = \mu_1 + \sigma_1, \quad$ and $u_4 = \mu_2 + \alpha_2$.
Note that $S_C \leq \frac{\eta_C}{\mu_1}$ and $S_B \leq \frac{\eta_B}{\mu_2}$ at DFE. Now by replacing the derivatives $\dot{E_C}, \dot{I_C}, \dot{R_C}, \dot{E_B}, \dot{I_B}$ into the equation of $\dot{L_0}$ we obtain,

$$\dot{L_0} = c_1[\beta_C(\tau_1 I_B + I_C)S_C - u_1 E_C] + c_2(\alpha_1 E_C - u_2 I_C) + c_3(\phi_1 I_C - u_3 R_C)$$

$$+ c_4[\beta_B(\tau_2 I_C + I_B)S_B - u_4 E_B] + c_5(\alpha_2 E_B - \mu_2 I_B)$$

$$\leq c_1[\beta_C(\tau_1 I_B + I_C) - u_1 E_C] + c_2(\alpha_1 E_C - u_2 I_C) + c_3(\phi_1 I_C - u_3 R_C)$$

$$+ c_4[\beta_B(\tau_2 I_C + I_B) - u_4 E_B] + c_5(\alpha_2 E_B - \mu_2 I_B),$$

$$= c_1\beta_C\tau_1 I_B + c_1\beta_C I_C - u_1 E_C + c_2\alpha_1 E_C - u_2 c_2 I_C + c_3\phi_1 I_C - u_3 c_3 R_C$$

$$+c_4\beta_B\tau_2 I_C + c_4\beta_B I_B - u_4 c_4 E_B + c_5\alpha_2 E_B - c_5\mu_2 I_B,$$

Collecting the linear terms of E_C, I_C, R_C, E_B, I_B and setting the coefficients E_C, I_C, R_C, E_B, I_B to zero we get,

$$= [c_2\alpha_1 - c_1 u_1]E_C + [c_1\beta_C - c_2 u_2 + c_3\phi_1 + c_4\beta_B\tau_2]I_C - c_3 u_3 R_C + [c_5\alpha_2 - c_4 u_4]E_B +$$

$$[c_1\tau_1\beta_C - c_5\mu_2 + c_4\beta_B]I_B = 0.$$

Now solving for c_1, c_2, c_3, c_4 and c_5 we get,

$c_1 = -\frac{c_4(\alpha_2\beta_B - u_4\mu_2)}{\alpha_2\beta_C\tau_1}$, $\quad b_2 = -\frac{c_4 u_1(\alpha_2\beta_B - u_4\mu_2)}{\alpha_1\alpha_2\beta_C\tau_1}$, $\quad c_3 = 0$, $\quad c_5 = \frac{c_4 u_4}{\alpha_2}$,

Let $c_4 = 1$, then this yields,

$$\begin{pmatrix} c_1 \\ c_2 \\ c_3 \\ c_4 \\ c_5 \end{pmatrix} = \begin{pmatrix} -\frac{\alpha_2\beta_B - u_4\mu_2}{\alpha_2\beta_C\tau_1} \\ -\frac{u_1(\alpha_2\beta_B - u_4\mu_2)}{\alpha_1\alpha_2\beta_C\tau_1} \\ 0 \\ 1 \\ \frac{u_4}{\alpha_2} \end{pmatrix},$$

$$= \frac{1}{\alpha_1\alpha_2\beta_C\tau_1} \begin{pmatrix} \alpha_1(u_4\mu_2 - \alpha_2\beta_B) \\ u_1(u_4\mu_2 - \alpha_2\beta_B) \\ 0 \\ \alpha_1\alpha_2\beta_C\tau_1 \\ u_4\alpha_1\beta_C\tau_1 \end{pmatrix},$$

So, $c_1 = \alpha_1(u_4\mu_2 - \alpha_1\beta_B)$, $\quad c_2 = u_1 u_4\mu_2(1 - \alpha_2\beta_B)$, $\quad c_3 = 0$, $\quad c_4 = \alpha_1\beta_C\alpha_2\tau_1$ $\quad$ and $c_5 = u_4\alpha_1\beta_C\tau_1$.

So by factorise c_1 and c_2 we get,

$$c_1 = \alpha_1 u_4 \mu_2 (1 - \mathcal{R}_B),$$

$$c_2 = u_1 u_4 \mu_2 (1 - \mathcal{R}_B)$$

$\mathcal{R}_B < 1$ for c_1 and $c_2 > 0$,

where $\mathcal{R}_B = \frac{\alpha_2 \beta_B}{u_4 \mu_2}$

$\implies \dot{L_0} \leq [c_1 \beta_C + c_4 \tau_2 \beta_B - c_2 u_2] I_C,$

$$= [\alpha_1 u_4 \mu_2 \beta_C (1 - \mathcal{R}_B) + \alpha_1 \beta_C \alpha_2 \tau_1 \beta_B \tau_2 - u_1 u_2 u_4 \mu_2 (1 - \mathcal{R}_B)] I_C,$$

$$= \mu_2 u_4 (1 - \mathcal{R}_B)(\alpha_1 \beta_C - u_1 u_2) + \alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B] I_C,$$

$$= [\mu_2 u_4 u_1 u_2 (1 - \mathcal{R}_B)(\frac{\alpha_1 \beta_C}{u_1 u_2} - 1) + \alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B] I_C,$$

$$= [\alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B - \mu_2 u_4 u_1 u_2 (1 - \mathcal{R}_B)(1 - \mathcal{R}_C)] I_C,$$

where $\mathcal{R}_C = \frac{\alpha_1 \beta_C}{u_1 u_2}$

$$= \alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B \left[1 - \frac{\mu_2 u_1 u_2 u_4 (1 - \mathcal{R}_B)(1 - \mathcal{R}_C)}{\alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B} \right] I_C.$$

Therefore,

$$\dot{L_0} \leq -\alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B (1 - \mathcal{R}_0) I_C.$$

$\implies \dot{L_0} \leq -M(1 - \mathcal{R}_0),$

where $M = \alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B$ is a constant and

$$\mathcal{R}_0 = \frac{\mu_2 u_1 u_2 u_4 (1 - \mathcal{R}_B)(1 - \mathcal{R}_C)}{\alpha_1 \alpha_2 \tau_1 \tau_2 \beta_C \beta_B}.$$

If $\mathcal{R}_0 < 1$, then $\dot{L}_0 < 0$ and $\dot{L}_0 = 0$ when $E_B = I_B = R_C = E_B = I_B = 0$. Therefore, the largest compact invariant set in Ω such that $\dot{L}_0 = 0$ when $\mathcal{R}_0 \leq 1$, is the singleton consisting of E_0. Thus, by LaSalle Invariance Principle [56], E_0 is globally asymptotically stable $\mathcal{R}_0 \leq 1$. This

completes the proof.

□

4.3.5 Local stability of the endemic equilibrium

Endemic equilibrium

The endemic equilibrium state denoted by E^* is the state where the disease cannot be totally eradicated but remains in the population. In this case, in order for the disease to persist in the population, $(S_C, E_C, I_C, R_C, S_B, E_B, I_B) \neq (0,0,0,0,0,0,0)$ at the equilibrium state. Let $\lambda_1 = \tau_1 \beta_C I_B + \beta_C I_C$ and $\lambda_2 = \tau_2 \beta_B I_C + \beta_B I_B$ be forces of infection. We calculate the endemic points in terms of the forces of infection by equating the right hand side to zero.

$$\eta_C - (\lambda_1^* + \mu_1) S_C^* + \sigma_1 R_C^* = 0, \tag{4.19}$$

$$\lambda_1^* S_C^* - (\mu_1 + \alpha_1) E_C^* = 0, \tag{4.20}$$

$$\alpha_1 E_C^* - (\mu_1 + \rho_1 + \phi_1) I_C^* = 0, \tag{4.21}$$

$$\phi_1 I_C^* - (\mu_1 + \sigma_1) R_C^* = 0, \tag{4.22}$$

$$\eta_B - (\lambda_2^* + \mu_2) S_B^* = 0, \tag{4.23}$$

$$\lambda_2^* S_B^* - (\mu_2 + \alpha_2) E_B^* = 0, \tag{4.24}$$

$$\alpha_2 E_B^* - \mu_2 I_B^* = 0. \tag{4.25}$$

Let $c_1 = (\mu_1 + \alpha_1), \quad c_2 = (\mu_1 + \rho_1 + \phi_1), \quad c_3 = (\mu_1 + \sigma_1), \quad$ and $c_4 = (\mu_2 + \alpha_2)$.
From equation (4.19)

$$\eta_C + \sigma_1 R_C^* = (\lambda_1^* + \mu_1) S_C^*,$$

$$\implies S_C^* = \frac{\eta_C + \sigma_1 R_C^*}{\lambda_1^* + \mu_1}. \tag{4.26}$$

From equation (4.20)

$$(\mu_1 + \alpha_1)E_C^* = \lambda_1^* S_C^*,$$

$$E_C^* = \frac{\lambda_1^* S_C^*}{c_1}. \tag{4.27}$$

Now substitute S_C^* into (4.27)

$$\implies E_C^* = \frac{\lambda_1^* \left(\eta_C + \sigma_1 R_C^*\right)}{c_1 \left(\lambda_1^* + \mu_1\right)}.$$

From equation (4.21)

$$I_C^* = \frac{\alpha_1 E_C^*}{c_2}, \tag{4.28}$$

Now substitute E_C^* into (4.28)

$$\implies I_C^* = \frac{\alpha_1 \lambda_1^* \left(\eta_C + \sigma_1 R_C^*\right)}{c_1 c_2 \left(\lambda_1^* + \mu_1\right)}.$$

In equation (4.22) substitute I_C^* and solve for R_C^*,

$$R_C^* = \frac{\alpha_1 \lambda_1^* \phi_1 \eta_C}{c_1 c_2 c_3 \left(\lambda_1^* + \mu_1\right) - \alpha_1 \lambda_1^* \sigma_1 \phi_1}. \tag{4.29}$$

Then

$$S_C^* = \frac{a_1 c_2 c_3 \eta_C}{c_1 c_2 c_3 \left(\lambda_1^* + \mu_1\right) - \alpha_1 \lambda_1^* \sigma_1 \phi_1},$$

$$E_C^* = \frac{c_2 c_3 \lambda_1^* \eta_C}{c_1 c_2 c_3 \left(\lambda_1^* + \mu_1\right) - \alpha_1 \lambda_1^* \sigma_1 \phi_1},$$

$$I_C^* = \frac{c_3\alpha_1\lambda_1^*\eta_C}{c_1c_2c_3\left(\lambda_1^*+\mu_1\right)-\alpha_1\lambda_1^*\sigma_1\phi_1}.$$

From equation (4.23)

$$S_B^* = \frac{\eta_B}{\lambda_2^*+\mu_2}. \tag{4.30}$$

From equation (4.24)

$$E_B^* = \frac{\lambda_1^*\eta_B}{c_4\left(\lambda_2^*+\mu_2\right)}. \tag{4.31}$$

From equation (4.25)

$$I_B^* = \frac{\alpha_2\lambda_1^*\eta_B}{c_4\mu_2\left(\lambda_2^*+\mu_2\right)}. \tag{4.32}$$

Let

$$\Psi = c_1c_2c_3\left(\lambda_1^*+\mu_1\right)-\alpha_1\lambda_1^*\sigma_1\phi_1,$$

Therefore, endemic equilibriums:

$$(S_C^*, E_C^*, I_C^*, R_C^*, S_B^*, E_B^*, I_B^*) =$$

$$\left(\frac{\eta_C c_1c_2c_3}{\Psi}, \frac{c_2c_3\lambda_1^*\eta_C}{\Psi}, \frac{c_3\alpha_1\lambda_1^*\eta_C}{\Psi}, \frac{\alpha_1\lambda_1^*\phi_1\eta_C}{\Psi}, \frac{\eta_B}{\lambda_2^*+\mu_2}, \frac{\lambda_1^*\eta_B}{c_4\left(\lambda_2^*+\mu_2\right)}, \frac{\alpha_2\lambda_1^*\eta_B}{c_4\mu_2\left(\lambda_2^*+\mu_2\right)}\right).$$

and

$$\lambda_1^* = \tau_1\beta_C I_B^* + \beta_C I_C^*,$$

$$\lambda_2^* = \beta_B I_B^* + \tau_2\beta_B I_C^*.$$

Substituting expressions $S_C^*, E_C^*, I_C^*, R_C^*, S_B^*, E_B^*$ and I_B^* into the two forces of infection λ_1^* and λ_2^*, we obtain:

$$\lambda_1 = \frac{c\beta_1\alpha_2\lambda_1^*\eta_B}{a_4\mu_2\left(\lambda_2^*+\mu_2\right)} + \frac{c\beta_2a_3\alpha_1\lambda_1^*\eta_C}{a_1a_2a_3\left(\lambda_1^*+\mu_1\right)-\alpha_1\lambda_1^*\sigma_1\phi_1},$$

$$\lambda_2 = \frac{c\beta_3\alpha_2\lambda_1^*\eta_B}{a_4\mu_2\left(\lambda_2^*+\mu_2\right)} + \frac{c\beta_4 a_3\alpha_1\lambda_1^*\eta_C}{a_1a_2a_3\left(\lambda_1^*+\mu_1\right)-\alpha_1\lambda_1^*\sigma_1\phi_1}.$$

Let

$A_1 = \frac{c\beta_1\alpha_2\eta_B}{a_4\mu_2}$, $A_2 = c\beta_2\alpha_1\eta_C a_3$, $A_3 = a_1a_2a_3$, $A_4 = \phi_1\alpha_1\sigma_1$, $A_5 = \frac{c\beta_3\alpha_2\eta_B}{a_4\mu_2}$ and $A_6 = c\beta_4\alpha_1\eta_C a_3$

Then

$$\lambda_1 = \frac{A_1\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{A_2\lambda_1^*}{A_3(\lambda_1^*+\mu_1)-A_4\lambda_1^*}.$$

$$\lambda_2 = \frac{A_5\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{A_6\lambda_1^*}{A_3(\lambda_1^*+\mu_1)-A_4\lambda_1^*}.$$

Case 1: If

$\lambda_1^* = \lambda_2^* = 0$, then

This corresponds to the disease-free equilibrium point where there are no viruses caused by infected buffaloes or infected cattle.

Case 2 : If

$\lambda_1^* \neq 0, \lambda_2^* \neq 0$, then

This is where there exists both viruses from infected cattle and infected buffaloes among the cattle and buffaloes population.

The equilibrium points of the model can be obtained by finding the fixed points of the equations:

$$F(\lambda_1,\lambda_2) = \begin{bmatrix} F_1(\lambda_1,\lambda_2) \\ F_2(\lambda_1,\lambda_2) \end{bmatrix} = \begin{bmatrix} \frac{A_1\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{A_2\lambda_1^*}{A_3(\lambda_1^*+\mu_1)-A_4\lambda_1^*} \\ \frac{A_5\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{A_6\lambda_1^*}{A_3(\lambda_1^*+\mu_1)-A_4\lambda_1^*} \end{bmatrix}.$$

Theorem 5. *There exists a unique fixed point* $(\lambda_1^*,\lambda_2^*), \lambda_1^* > 0, \lambda_2^* > 0$ *satisfying*

$$F(\lambda_1^*,\lambda_2^*) = \begin{bmatrix} \lambda_1^* \\ \lambda_2^* \end{bmatrix};$$

corresponding to the endemic equilibrium point.

Proof. In this case three conditions are considered.

By fixing $\lambda_1 > 0$, and considering the real valued functions depending on λ_1:

$$F_1^{\lambda_1^*}(\lambda_2) = \frac{A_1\lambda_2^*}{\lambda_2^* + \mu_2} + \frac{A_2\lambda_1^*}{A_3(\lambda_1^* + \mu_1) - A_4\lambda_1^*}.$$

So by letting $\lambda_2 = 0$, we get, $\frac{A_2\lambda_1}{A_3(\lambda_1+\mu_1)-A_4\lambda_1} > 0$ if $\frac{A_3(\lambda_1+\mu_1)}{\lambda_1} > A_4$. Taking limit,

$$\lim_{\lambda_2 \to \infty} F_1^{\lambda_1}(\lambda_2) = A_1 + \frac{A_2\lambda_1}{A_3(\lambda_1 + \mu_1) - A_4\lambda_1} < \infty.$$

Hence,

$$0 < F_1^{\lambda_1}(\lambda_2) < \infty,$$

so that the function; $F_1^{\lambda_1}(\lambda_2)$ is bounded for every fixed $\lambda_1 > 0$.

Second condition is to find the first derivative of $F_1^{\lambda_1}(\lambda_2)$ with respect to λ_2.

$$\frac{dF_1^{\lambda_1^*}}{d\lambda_2} = \frac{A_1\mu_1}{(\lambda_1 + \mu_1)^2} > 0.$$

Now finding the third condition which is the second derivative given by;

$$\frac{d^2F_1^{\lambda_1}}{d^2\lambda_2} = -2\left(\frac{A_1\mu_1}{(\lambda_1 + \mu_1)^2}\right) < 0.$$

Since $\frac{dF_1^{\lambda_1}}{d\lambda_2} > 0$ and $\frac{d^2F_1^{\lambda_1}}{d^2\lambda_2} < 0$, then the function $F_1^{\lambda_1}(\lambda_2)$ is an increasing concave down function which is fixed in convexity in the bounded domain.

$\implies$ there exist a unique point $\lambda_2^* > 0$ such that $F_1^{\lambda_1^*}(\lambda_2^*) = \lambda_2^*$

By fixing $\lambda_2 > 0$, and considering the real valued functions depending on λ_2:

$$F_1^{\lambda_1}(\lambda_2) = \frac{A_5\lambda_2^*}{\lambda_2^* + \mu_2} + \frac{A_6\lambda_1^*}{A_3(\lambda_1^* + \mu_1) - A_4\lambda_1^*}.$$

So by letting $\lambda_1 = 0$, then

$$F_2^{\lambda_1^*}(0) = \frac{A_5\lambda_2}{\lambda_2 + \mu_2} > 0.$$

Taking limit,

$$\lim_{\lambda_2 \to \infty} F_2^{\lambda_2}(\lambda_1) = \frac{A_5 \lambda_2^*}{\lambda_2^* + \mu_2} + \frac{A_6}{A_3 - A_4} < \infty.$$

$$\frac{dF_1^{\lambda_2^*}}{d\lambda_1} = \frac{A_3 A_6 \mu_1}{(\lambda_1(A_3 - A_4) + A_3\mu_1)^2} > 0.$$

Now finding the third condition which is the second derivative given by;

$$\frac{\partial^2 F_2^{\lambda_2}}{\partial^2 \lambda_1} = \frac{-2A_3 A_6 \mu_1 (A_3 - A_4)}{(\lambda_1(A_3 - A_4) + A_3\mu_1)^4} < 0.$$

Since $\frac{dF_2^{\lambda_2}}{d\lambda_1} > 0$ and $\frac{d^2F_1^{\lambda_1}}{d^2\lambda_2} < 0$, then the function $F_1^{\lambda_1}(\lambda_2)$ is an increasing concave down function which is fixed in convexity in the bounded domain.

$\implies$ there exists a unique point $\lambda_1^* > 0$ such that $F_2^{\lambda_2}(\lambda_1^*) = \lambda_1^*$

□

4.3.6 Local stability of the endemic Equilibrium point

In this section we need to show the local stability by applying centre manifold theory to determine stability of endemic equilibria near the threshold parameter $\mathcal{R}_0$. With the presence of zero eigenvalues in the stability matrix at the critical point, linear theory fails to provide information on the stability of that point. The main aim of the centre manifold is to reduce the dimension of the system near that point so that the stability of the reduced system can be investigated. In epidemic models, there are two distinct bifurcations at $\mathcal{R}_0 = 1$; namely, forward (supercritical) and backward (sub-critical). A forward bifurcation to occur when R_0 crosses unity from below; a small positive asymptotically stable equilibrium appears and the disease-free equilibrium losses its stability. On the other hand, a backward bifurcation to occur when $\mathcal{R}_0$ is less than unity; a small positive unstable equilibrium appears while the disease-free equilibrium and a larger positive equilibrium are locally asymptotically stable [57]. The theory is applied to examine the existence of backward and forward bifurcation. When the bifurcation is forward, it means that the endemic equilibrium state is locally asymptotically stable for

$\mathcal{R}_0 > 1$ but near one. We prove that there is at least one endemic equilibrium point for all $\mathcal{R}_0 > 1$ by giving a general bifurcation. Since it is not convenient to use $\mathcal{R}_0$ directly as a bifurcation parameter, we define a bifurcation parameter as η_B^*.

Theorem 6. *The model* (4.10) *exhibits a forward bifurcation at* $\mathcal{R}_0 = 1$. *Therefore, the endemic equilibrium point is locally asymptotically stable for* $\mathcal{R}_0 > 1$ *but close to 1.*

We consider system (4.10) and investigate the nature of the bifurcation involving the DFE $E_0(\frac{\eta_C}{\mu_1}, 0, 0, 0, \frac{\eta_B}{\mu_2}, 0, 0)$ for $R_0 = 1$. To be more specific, we look for conditions on the parameter values that cause a forward or backward bifurcation to occur. Hence, we will apply the lemma or results that are summarised in [47, 57] to show that the system may exhibit a forward bifurcation for the endemic equilibria to be stable.

Let $\beta_C = \delta\beta_B$ considering β_B to be proportion to β_C then, let β_B be a bifurcation parameter at $\mathcal{R}_0 = 1$ with the conditions that

If $\beta_B^* > 0$ then $\mathcal{R}_0 > 1$.

If $\beta_B^* < 0$ then $\mathcal{R}_0 < 1$.

If $\beta_B^* = 0$ then $\mathcal{R}_0 = 1$.

So by considering $\beta_B = \beta_B^*$, then we get;

Proof. Let Let $g_1 = \mu_1 + \alpha_1, g_2 = \mu_1 + \phi_1 + \rho_1, g_3 = \mu_2 + \alpha_2$, then

$$\mathcal{R}_0 = \frac{\sqrt{2\alpha_1\alpha_2\mu_2 g_1 g_2 g_3 \beta_B S_B \beta_C S_C (2\tau_1\tau_2 - 1) + \alpha_2^2 g_1^2 g_2^2 \beta_B^2 S_B^2 + \alpha_1^2 \mu_2^2 g_3^2 \beta_C^2 S_C^2} + \alpha_2 g_1 g_2 \beta_B S_B + \alpha_1 \mu_2 g_3 \beta_C S_C}{2\mu_2 g_1 g_2 g_3}.$$

$\mathcal{R}_0 = 1$ is the same as,

$$\beta_B^* = \frac{\sqrt{2\alpha_1\alpha_2\mu_2 g_1 g_2 g_3 S_B S_C \delta (2\tau_1\tau_2 - 1) + \alpha_2^2 g_1^2 g_2^2 S_B^2 + \alpha_1^2 \mu_2^2 g_3^2 \delta^2 S_C^2} + \alpha_2 g_1 g_2 S_B + \alpha_1 \mu_2 g_3 \delta S_C}{2\mu_2 g_1 g_2 g_3}.$$

Where β_B^* is chosen as the bifurcation parameter that occurs at $\mathcal{R}_0 = 1$. Let $J(E_0)$ be a Jacobian matrix of the

model (4.10) at the DFE as:

$$J(E_0) = \begin{bmatrix} -\mu_1 & 0 & \frac{-\beta_C\eta_C}{\mu_1} & \sigma_1 & 0 & 0 & \frac{-\tau_1\beta_C\eta_C}{\mu_1} \\ 0 & -(\mu_1+\alpha_1) & \frac{\beta_C\eta_C}{\mu_1} & 0 & 0 & 0 & \frac{\tau_1\beta_1\eta_C}{\mu_1} \\ 0 & \alpha_1 & -(\mu_1+\rho_1+\phi_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_1 & -(\mu_1+\sigma_1) & 0 & 0 & 0 \\ 0 & 0 & \frac{-\tau_2\beta_B\eta_B}{\mu_2} & 0 & -\mu_2 & 0 & \frac{-\beta_B\eta_B}{\mu_2} \\ 0 & 0 & \frac{\tau_2\beta_B\eta_B}{\mu_2} & 0 & 0 & -(\mu_2+\alpha_2) & \frac{\beta_B\eta_B}{\mu_2} \\ 0 & 0 & 0 & 0 & 0 & \alpha_2 & -\mu_2 \end{bmatrix}.$$

We get one of our eigenvalues as zero which we call a simple eigenvalue of the matrix $J(E_0,\beta_B^*)$ and the other eigenvalues are negative real numbers, therefore we can make use of the center manifold theory. Thus, when $\beta_B = \beta_B^*$, the DFE E_0 is a non-hyperbolic equilibrium as required according to the theorem 4.1 in [57].

Now let $(w_1,w_2,w_3,w_4,w_5,w_6,w_7)^T$ be the right eigenvector corresponding to the zero eigenvalue of the Jacobian matrix $J(E_0,\beta_B^*)$ when $\mathcal{R}_0 = 1$. Now we calculate the right eigenvector w by multiplying $J(E_0)$ with the vector after the transpose we get:

$$J(E_0) = \begin{bmatrix} -\mu_1 & 0 & \frac{-\beta_C\eta_C}{\mu_1} & \sigma_1 & 0 & 0 & \frac{-\tau_1\beta_C\eta_C}{\mu_1} \\ 0 & -(\mu_1+\alpha_1) & \frac{\beta_C\eta_C}{\mu_1} & 0 & 0 & 0 & \frac{\tau_1\beta_1\eta_C}{\mu_1} \\ 0 & \alpha_1 & -(\mu_1+\rho_1+\phi_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_1 & -(\mu_1+\sigma_1) & 0 & 0 & 0 \\ 0 & 0 & \frac{-\tau_2\beta_B\eta_B}{\mu_2} & 0 & -\mu_2 & 0 & \frac{-\beta_B\eta_B}{\mu_2} \\ 0 & 0 & \frac{\tau_2\beta_B\eta_B}{\mu_2} & 0 & 0 & -(\mu_2+\alpha_2) & \frac{\beta_B\eta_B}{\mu_2} \\ 0 & 0 & 0 & 0 & 0 & \alpha_2 & -\mu_2 \end{bmatrix} \times \begin{bmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \\ w_7 \end{bmatrix}.$$

Now let $k_1 = (\mu_1+\alpha_1), k_2 = (\mu_1+\rho_1+\phi_1), k_3 = (\mu_1+\sigma_1), k_4 = (\mu_2+\alpha_2)$.

$$J(E_0) = \begin{bmatrix} -\mu_1 & 0 & -\beta_C S_C & \sigma_1 & 0 & 0 & -\tau_1\beta_C S_C \\ 0 & -k_1 & \beta_C S_C & 0 & 0 & 0 & \tau_1\beta_C S_C \\ 0 & \alpha_1 & -k_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_1 & -k_3 & 0 & 0 & 0 \\ 0 & 0 & -\tau_2\beta_B S_B & 0 & -\mu_2 & 0 & -\beta_B S_B \\ 0 & 0 & \tau_2\beta_B S_B & 0 & 0 & -k_4 & \beta_B S_B \\ 0 & 0 & 0 & 0 & 0 & \alpha_2 & -\mu_2 \end{bmatrix} \times \begin{bmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \\ w_7 \end{bmatrix}.$$

Thus,

$$
\begin{aligned}
-\mu_1 w_1 - \frac{\beta_C \eta_C}{\mu_1} w_3 + \sigma_1 w_4 - \frac{\tau_1 \beta_C^* \eta_C}{\mu_1} w_7 &= 0, \\
-k_1 w_2 + \frac{\beta_C \eta_C}{\mu_1} w_3 + \frac{\tau_1 \beta_C^* \eta_C}{\mu_1} w_7 &= 0, \\
\alpha_1 w_2 - k_2 w_3 &= 0, \\
\phi_1 w_3 + k_3 w_4 &= 0, \\
-\frac{\tau_2 \beta_B^* \eta_B}{\mu_2} w_3 - \mu_{2w_5} - \frac{\beta_B^* \eta_B}{\mu_2} w_7 &= 0, \\
\frac{\tau_2 \beta_B^* \eta_B}{\mu_2} w_3 - k_4 w_6 + \frac{\beta_B^* \eta_B}{\mu_2} w_7 &= 0, \\
\alpha_2 w_6 - \mu_2 w_7 &= 0.
\end{aligned}
$$

Therefore, solving for $w_1, w_2, w_3, w_4, w_5, w_6, w_7$ we get:

$$
\begin{aligned}
w_6 &= \frac{\mu_2 w_7}{\alpha_2}, \\
w_3 &= \frac{-S_B w_7 \alpha_2 \beta_B + k_4 w_7 \mu_2}{\tau_2 S_B \alpha_2 \beta_B}, \\
w_2 &= \frac{-k_2 (S_B w_7 \alpha_2 \beta_B - k_4 w_7 \mu_2)}{\tau_2 S_B \alpha_1 \alpha_2 \beta_B}, \\
w_4 &= \frac{\phi_1 w_7 (\alpha_2 \beta_4 S_b + b_4 \mu_2)}{\alpha_2 b_3 \beta_3 S_b}, \\
w_1 &= \frac{w_7 S_B \alpha_2 \beta_B (\sigma_1 \phi_1 + (1 + \tau_1 \tau_2) k_3 S_C \beta_C + k_4 (\sigma_1 \phi_1 + k_3 S_C \beta_C) \mu_2}{\tau_2 k_3 \alpha_2 \beta_B \mu_1}, \\
w_5 &= \frac{k_4 w_7}{\alpha_2}.
\end{aligned}
$$

The right eigenvector is

$$
W = (w_1, w_2, w_3, w_4, w_5, w_6, 1)^T.
$$

Where $w_7 > 0$ in this case we choose $w_7 = 1$.

Furthermore, the left eigenvector $V = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$ satisfying $V \cdot W = 1$ is given by:

$$
\begin{aligned}
v_1 &= v_4 = v_5 = 0, \\
v_2 &= \frac{v_7 k_4 \alpha_1}{k_1}, \\
v_3 &= \frac{\tau_2 k_1 S_B v_7 \beta_B}{k_1 k_2 - S_C \alpha_1 \beta_C}, \\
v_6 &= \frac{k_4 v_7}{\alpha_2}, \\
v_7 &= \frac{\alpha_2^2 (k_1 k_2 - \alpha_1 \beta_C S_C)}{\left(-\alpha_2^2 k_1 \beta_B S_B - \alpha_2^2 x \beta_B S_B - \alpha_1 \alpha_2^2 \beta_C S_C - \alpha_1 \mu_2 k_4 \beta_C S_C + \alpha_2^2 k_1 k_2 + \mu_2 k_1 k_2 k_4 + \alpha_2 \mu_2 k_1 k_4 + \alpha_2 \mu_2 k_2 k_4\right)}.
\end{aligned}
$$

let $S_C = x_1, E_C = x_2, I_C = x_3, R_C = x_4, S_B = x_5, E_B = x_6, I_B = x_7$,
and $\frac{dS_C}{dt} = f_1, \frac{dE_C}{dt} = f_2, \frac{dI_C}{dt} = f_3, \frac{dR_C}{dt} = f_4, \frac{dS_B}{dt} = f_5, \frac{dE_B}{dt} = f_6, \frac{dI_B}{dt} = f_7$.

Then evaluating the partial derivatives at the disease free equilibrium, we obtain

$$
\begin{aligned}
\frac{\partial^2 f_2}{\partial x_1 \partial x_3} &= \frac{\partial^2 f_2}{\partial x_3 \partial x_1} = \beta_C, \\
\frac{\partial^2 f_2}{\partial x_1 \partial x_7} &= \frac{\partial^2 f_2}{\partial x_7 \partial x_1} = \tau_1 \beta_B, \\
\frac{\partial^2 f_6}{\partial x_5 \partial x_7} &= \frac{\partial^2 f_6}{\partial x_7 \partial x_5} = \beta_B, \\
\frac{\partial^2 f_6}{\partial x_3 \partial x_5} &= \frac{\partial^2 f_6}{\partial x_5 \partial x_3} = \tau_2 \beta_B, \\
\frac{\partial^2 f_2}{\partial x_3 \partial \beta_B} &= \frac{\partial^2 f_2}{\partial \beta_B \partial x_3} = x_1 \delta, \\
\frac{\partial^2 f_2}{\partial x_7 \partial \beta_B} &= \frac{\partial^2 f_2}{\partial \beta_B \partial x_7} = x_1 \tau_1 \delta, \\
\frac{\partial^2 f_6}{\partial x_7 \partial \beta_B} &= \frac{\partial^2 f_2}{\partial \beta_B \partial x_7} = x_5, \\
\frac{\partial^2 f_6}{\partial x_3 \partial \beta_B} &= \frac{\partial^2 f_2}{\partial \beta_B \partial x_3} = x_5 \tau_2.
\end{aligned}
$$

Where in this case x_1 and x_5 are the DFE points $x_1 = \frac{\eta_C}{\mu_1}$ and $x_5 = \frac{\eta_B}{\mu_2}$.
Now by computing the coefficients a and b,

$$a = \sum_{k,i,j=1}^{7} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, c^*),$$
$$b = \sum_{k,i=1}^{7} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial c}(E_0, c^*).$$

$$a =$$

$$b = \frac{\alpha_1\alpha_2\left(\alpha_2\beta_b S_b\left((\tau_1+1)\delta x_1 - \tau_2(\tau_2+1)x_5\right) - (\tau_1+1)\delta\mu_2 x_1 k_4\right)}{\alpha_2^2\left(\beta_B S_B(k_1+k_2) + \alpha_1\beta_C S_C - k_1 k_2\right) - \mu_2 k_4\left(-\alpha_1\beta_C S_C + \alpha_2(k_1+k_2) + k_1 k_2\right)}.$$

$a > 0$ if the following conditions hold:

1. $\tau_1\tau_2 > 1$,

2. $\tau_1\tau_2 < 2$,

3.$\tau_1 < 1$.

$b < 0$ if $\tau_1\tau_2 > 1$ and $\tau_1\tau_2 < 2$. $a > 0, b < 0$. When $\beta_B^* < 0$ with $|\beta_B^*| \leq 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \beta_B^* \leq 1$, 0 is stable, and a positive unstable equilibrium appears. According to the theorem 4.1 in [57] it is sign of the coefficient a which determines the local dynamics around the disease-free equilibrium for $\beta_B = \beta_B^*$. □

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Chapter 5

Vaccination-culling model formulation and analysis

In this chapter, a model namely: Susceptible-Exposed-Infected-Recovery-Vaccinated (SEIRV) model which incorporates vaccination and culling was formulated and analysed. We first determine the positivity as well as boundedness of the solutions: $S(t)$, $E(t)$, $I(t)$, $R(t)$ and $V(t)$ using derivatives and integration techniques. These are necessary conditions for biological meaningfulness of our models.

5.1 Vaccination and culling model formulation

In this section, we will formulate a model where intervention strategies, namely, culling and vaccination, are assumed to be introduced, but only among the cattle population.

5.1.1 Cattle population model

For the vaccination-culling model, the cattle population is subdivided into five classes, namely: susceptible, exposed, infective, recovery, and vaccinated which are denoted by S_C, E_C, I_C, R_C

and V_C respectively, so that the total population

$$N_C(t) = S_C(t) + E_C(t) + I_C(t) + R_C(t) + V_C(t)$$

.

Assumptions of the vaccination and culling model are adopted from the basic model (4.5) above with the modification that, control mechanisms have now been included.

The population of susceptible cattle is assumed to increase via recruitment at a rate η_C. It is then decreased by infection at rates β_C via effective contacts with infected cattle and infected buffaloes, respectively. The relative infectivity of an infected animal is τ_1, where τ_1 is a constant taking values in the interval [0,1). The susceptible cattle are vaccinated at the rate ω, and subsequently move to the vaccinated class where they are protected temporarily from FMD infection. Susceptible cattle suffer natural death at a rate μ_1. Similarly, the class also gains from the recovery of the infected cattle at a rate σ_1. Thus, we get the following differential equation.

$$\frac{dS_C}{dt} = \eta_C - \mu_1 S_C - \beta_C(\tau_1 I_B + I_C)S_C + \sigma_1 R_C - \omega S_C. \tag{5.1}$$

The population of vaccinated cattle is produced from the vaccination of susceptible cattle at a rate ω. Vaccination may provide complete or partial immunity to the disease. This population is assumed to decrease due to natural death at a rate of μ_1. Let ε be the vaccination efficacy. Then the vaccination protection rate is the product of vaccination rate and vaccination efficacy $p = \varepsilon\omega$. Additionally, the proportion of those cattle that are not protected or the vaccination is unsuccessful and can enter the exposed class is $q = 1 - p$. Therefore, the total number of cattle that are vaccinated but not protected from the FMD virus is qV_C. It is assumed that vaccine

does not wane off. Thus, the model equation becomes:

$$\frac{dV_C}{dt} = \omega S_C - \mu_1 V_C - \beta_C q(\tau_1 I_B + I_C)V_C. \tag{5.2}$$

Susceptible and vaccinated cattle enter the exposed compartment at rates $\beta_C S_C I_B$, $\beta_C S_C I_C$, $\tau_1 \beta_C q V_C I_B$ and $\tau_1 \beta_C q V_C I_C$, respectively. This population of exposed cattle is decreased by progression of individuals to infectious class at a rate α_1, and by natural death at a rate μ_1. Thus,

$$\frac{dE_C}{dt} = \beta_C(\tau_1 I_B + I_C)S_C + \beta_C q(\tau_1 I_B + I_C)V_C - (\mu_1 + \alpha_1)E_C. \tag{5.3}$$

The population of infected cattle is produced when exposed cattle develop symptoms at a rate α_1. In order to reduce FMDV, culling is assumed to take place at infective class in this model. Therefore, the population of infective cattle is assumed to decrease via the culling of cattle at a rate θ, cattle recovery at a rate of ϕ_1, natural death at the rate μ_1 and disease-induced death at a rate ρ_1. This gives

$$\frac{dI_C}{dt} = \alpha_1 E_C - (\mu_1 + \rho_1 + \phi_1 + \theta)I_C. \tag{5.4}$$

The population of recovered cattle is decreased by natural death at a rate μ_1 and loss of acquired immunity (recovered cattle become susceptible again) at the rate σ_1. Here we assume that recovered cattle do not acquire permanent immunity against re-infection. The model equation is given by,

$$\frac{dR_C}{dt} = \phi_1 I_C - (\mu_1 + \sigma_1)R_C. \tag{5.5}$$

Therefore, combining the equations (5.1), (5.2), (5.3), (5.4), and (5.5), yields the following

system of nonlinear ODEs (a flow diagram of the model is depicted in Figure 4.1).

$$\begin{aligned}
\frac{dS_C}{dt} &= \eta_C - \mu_1 S_C - \beta_C(\tau_1 I_B + I_C)S_C + \sigma_1 R_C - \omega S_C,\\
\frac{dV_C}{dt} &= \omega S_C - \mu_1 V_C - \beta_C q(\tau_1 I_B + I_C)V_C,\\
\frac{dE_C}{dt} &= \beta_C(\tau_1 I_B + I_C)S_C + \beta_C q(\tau_1 I_B + I_C)V_C - (\mu_1 + \alpha_1)E_C,\\
\frac{dI_C}{dt} &= \alpha_1 E_C - (\mu_1 + \rho_1 + \phi_1 + \theta)I_C,\\
\frac{dR_C}{dt} &= \phi_1 I_C - (\mu_1 + \sigma_1)R_C.
\end{aligned} \tag{5.6}$$

5.1.2 Buffalo population model

It is assumed in this model (vaccination-culling model) that buffaloes are neither culled nor vaccinated. Thus, the model assumptions and the system of nonlinear ODEs for the buffaloes population are the same as described in Section 4.1. Moreover, it is assumed that there is no recovery for buffaloes.

5.1.3 Combined model

The systems of ODEs (4.9) and (5.6) result in the following vaccination-culling model, explaining the FMD dynamics in the combined populations in the presence of control measures (as illustrated

in Figure 5.1):

$$
\begin{aligned}
\frac{dS_C}{dt} &= \eta_C - (\mu_1 + \lambda_1)S_C + \sigma_1 R_C - \omega S_C, \\
\frac{dV_C}{dt} &= \omega S_C - (\mu_1 + q\lambda_1)V_C, \\
\frac{dE_C}{dt} &= \lambda_1 S_C + q\lambda_1 V_C - (\mu_1 + \alpha_1)E_C, \\
\frac{dI_C}{dt} &= \alpha_1 E_C - (\mu_1 + \rho_1 + \phi_1 + \theta)I_C, \\
\frac{dR_C}{dt} &= \phi_1 I_C - (\mu_1 + \sigma_1)R_C, \\
\frac{dS_B}{dt} &= \eta_B - (\mu_2 + \lambda_2)S_B, \\
\frac{dE_B}{dt} &= \lambda_2 S_B - (\mu_2 + \alpha_2)E_B, \\
\frac{dI_B}{dt} &= \alpha_2 E_B - \mu_2 I_B.
\end{aligned}
\tag{5.7}
$$

Based on the assumptions and systems of differential equations above, we get the following compartmental model in Figure 5.1

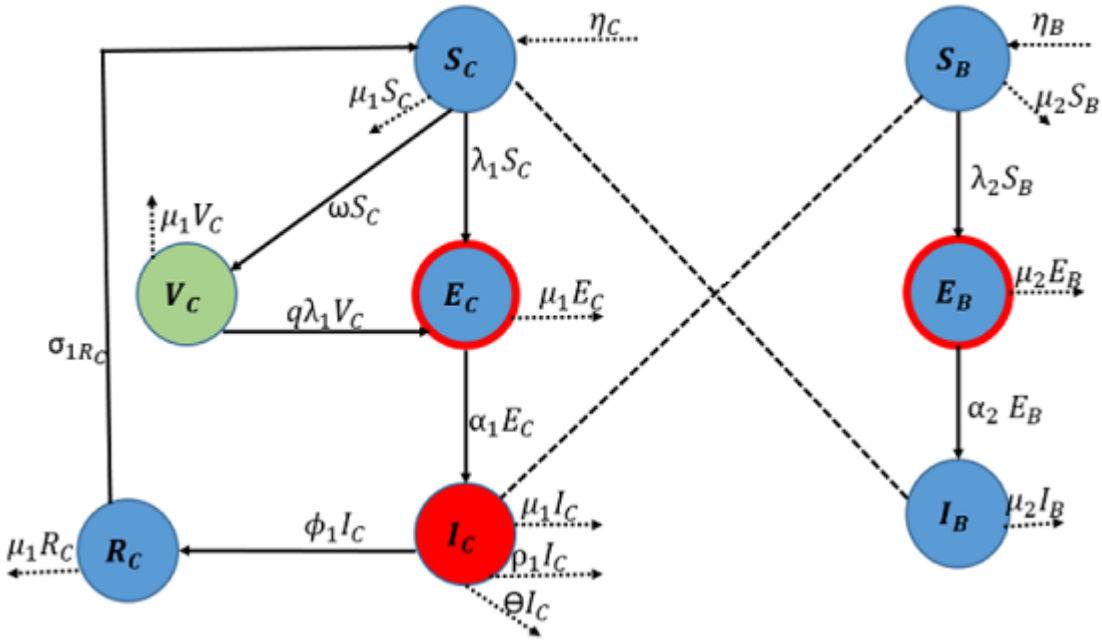

Figure 5.1: Vaccination-culling model for FMD

The above model parameters and variables are further described in Table 5.1

Table 5.1: Variables and parameters of the models

Parameters	Descriptions
$\eta_C \& \eta_B$	Recruitment rates of susceptible cattle and buffaloes into the population
β_C	Transmission rate of susceptible cattle
β_B	Transmission rate of susceptible buffaloes
$\tau_1 \& \tau_2$	Modification parameters (that compare the difference in infections between cattle and buffaloes)
$\mu_1 \& \mu_2$	Natural death rates of cattle and buffaloes
$\alpha_1 \& \alpha_2$	Rates of progression to infectious cattle and buffaloes classes
ρ_1	Disease-induced mortality rate
ϕ_1	Proportion of infective cattle becoming recovered
σ_1	The rate at which recovered cattle become susceptible
ω	The rate at which susceptible cattle population is vaccinated
ε	vaccination efficacy
θ	Proportion of exposed cattle detected and culled

5.2 Analysis of the vaccination-culling model

5.2.1 Positive invariance of the model (4.10)

Proof. We prove that each solution components $S_C(t)$, $V_C(t)$, $E_C(t)$, $I_C(t)$, $R_C(t)$, $S_B(t)$, $E_B(t)$ and $I_B(t)$ remain non-negative. Otherwise, if by contradiction:

Assume that there exists a first time t_1 such that $S_C(t_1) = 0$, $S_C'(t_1) < 0$ and $E_C(t) > 0, I_C(t) > 0, R_C(t) > 0, S_B(t) > 0, E_B(t) > 0, I_B(t) > 0$ for $0 < t < t_1$.

From the first equation in (4.1),

$$\frac{dS_C(t_1)}{dt} = \eta_C - \mu_1 S_C(t_1) - \tau_1 \beta_C I_B(t_1) S_C(t_1) - \beta_C I_C(t_1) S_C(t_1) + \sigma_1 R_C(t_1) - \omega S_C, \tag{5.8}$$

Since $S_C(t_1) = 0$ and $R_C(t) > 0$ from our assumptions then,

$$\frac{dS_C}{dt}(t_1) = \eta_C + \sigma_1 R_C(t_1) > 0.$$

Since we assume $S_C'(t_1) < 0$ then this is a contradiction and consequently, $S_C(t) \not< 0$.

Therefore, $S_C(t) > 0$.

We assume that there exists a first time t_2 such that $V_C(t_2) = 0$, $V_C'(t_2) < 0$ and $S_C(t) > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$ for $0 < t < t_2$.

$$\frac{dV_C(t_2)}{dt} = \omega S_C(t_2) - \mu_1 V_C(t_2) - \tau_1 \beta_C q V_C(t_2) I_B(t_2) - \beta_C q V_C(t_2) I_C(t_2), \tag{5.9}$$

Since $V_C(t_2) = 0$, and $S_C(t) > 0$ from our assumptions then,

$$\frac{dV_C}{dt}(t_2) = \omega S_C(t_2) > 0.$$

Since we assume $V_C'(t_2) < 0$ then this is a contradiction and consequently, $V_C(t) \not< 0$.
Therefore, $V_C(t) > 0$.

Suppose that $\exists$ a first time t_3 such that $E_C(t_3) = 0$, $E_C'(t_3) < 0$ and $S_C(t) > 0$, $V_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$. for $0 < t < t_2$.
Now given,

$$\frac{dE_C(t_3)}{dt} = \tau_1 \beta_C I_B(t_3) S_C(t_3) + \beta_C I_C(t_3) S_C(t_3) + \tau_1 \beta_C q V_C(t_3) I_B(t_3) + \beta_C q V_C(t_3) I_C(t_3) - (\mu_1 + \alpha_1) E_C(t_3), \tag{5.10}$$

Since from our assumptions $E_C(t_3) = 0$ and $S_C(t), V_C(t), I_C(t), I_B(t) > 0$ then,

$$\frac{dE_C(t_3)}{dt} = \tau_1 \beta_C I_B(t_3) S_C(t_3) - \beta_C I_C(t_3) S_C(t_3) +$$

$$\tau_1 q \beta_C I_B(t_3) V_C(t_3) + q \beta_C I_C(t_3) V_C(t_3) > 0,$$

which is a contradiction and consequently, $E_C(t) \not< 0$.
Therefore, $E_C(t) > 0$, $\forall$, $t \in (0, t_3)$.

Suppose that $\exists$ a first time t_4 such that $I_C(t_4) = 0$, $I_C'(t_4) < 0$ and $S_C(t) > 0$, $V_C(t) > 0$, $E_C(t) > 0$,

$R_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$. for $0 < t < t_3$.

Now given,

$$\frac{dI_C(t_4)}{dt} = \alpha_1 E_C(t_4) - (\mu_1 + \rho_1 + \phi_1 + \theta) I_C(t_4), \tag{5.11}$$

Since from our assumptions $I_C(t) = 0$ and $E_C(t) > 0$ then,

$$\frac{dI_C(t_4)}{dt} = \alpha_1 E_C(t_4) > 0,$$

which is a contradiction, hence $I_C(t) \not< 0$.

Therefore, $I_C(t) > 0, \forall\, t \in (0, t_4)$.

Suppose that $\exists$ a first time t_5 such that $R_C(t_5) = 0$, $R'_C(t_5) < 0$ and $S_C(t) > 0$, $V_C(t) > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$. for $0 < t < t_6$.

Now considering,

$$\frac{dR_C}{dt}(t_5) = \phi_1 I_C(t_5) - (\mu_1 + \sigma_1) R_C(t_5), \tag{5.12}$$

Since from our assumptions $R_C(t_5) = 0$ and $I_C(t_5) > 0$ then,

$$\frac{dR_C(t_5)}{dt} = \phi_1 I_C(t_5) > 0,$$

which is a contradiction, hence $R_C(t) \not< 0$.

Therefore, $R_C(t) > 0, \forall\, t \in (0, t_5)$.

We assume that there exists a first time t_6 such that $S_B(t_6) = 0$, $S'_B(t_6) < 0$ and $S_C(t) > 0$, $V_C > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $E_B(t) > 0$, $I_B(t) > 0$ for $0 < t < t_6$.

Given

$$\frac{dS_B}{dt}(t_6) = \eta_B - \mu_2 S_B(t_6) + \tau_2 \beta_B I_C(t_6) S_B(t_6) - \beta_B I_B(t_6) S_B(t_6), \tag{5.13}$$

Since $S_B(t_6) = 0$ from our assumptions then,

$$\frac{dS_B(t_6)}{dt} = \eta_B > 0.$$

Since we assume $\frac{dS_B}{dt}(t_6) < 0$ then this contradict to our assumption. Hence $S_B(t) \not< 0$ in $(0, t_6)$. Therefore, $S_B(t) > 0$.

Suppose that $\exists$ a first time t_7 such that $E_B(t_7) = 0$, $E_B'(t_7) < 0$ and $S_C(t) > 0$, $V_C > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $I_B(t) > 0$. for $0 < t < t_7$.
Now given,

$$\frac{dE_B(t_7)}{dt} = \tau_2\beta_B I_C(t_7)S_B(t_7) - \beta_B I_B(t_7)S_B(t_7) - \mu_2 E_B(t_7) + \alpha_2)E_B(t_7), \tag{5.14}$$

Since from our assumptions $E_B(t_7) = 0$ and $S_B(t), I_C(t), I_B(t) > 0$ then,

$$\frac{dE_B(t_7)}{dt} = \tau_2\beta_B I_C(t_7)S_B(t_7) + \beta_B I_B(t_7)S_B(t_7) > 0,$$

which is a contradiction and consequently, $E_B(t) \not< 0$. Therefore, $E_B(t) > 0$, $\forall$, $t \in (0, t_7)$.

Suppose that $\exists$ a first time t_8 such that $I_B(t_8) = 0$, $I_B'(t_8) < 0$ and $S_C(t) > 0$, $V_C(t) > 0$, $E_C(t) > 0$, $I_C(t) > 0$, $R_C(t) > 0$, $S_B(t) > 0$, $E_B(t) > 0$. for $0 < t < t_8$.
Now given,

$$\frac{dI_B}{dt}(t_7) = \alpha_2 E_B(t_7) - \mu_2 I_B(t_7), \tag{5.15}$$

Since from our assumptions $I_B(t_8) = 0$ and $E_B(t) > 0$ then,

$$\frac{dI_B}{dt}(t_8) = \alpha_2 E_B(t_8) > 0,$$

which is a contradiction, hence $I_B(t) \not< 0$.
Therefore, $I_B(t)$ remain positive $\forall$, $t \in (0, t_8)$. □

Thus, $S_C, V_C, E_C, I_C, R_C, S_B, E_B, I_B > 0$ for all $t > 0$.

5.2.2 Boundedness of solution of the model

Lemma 3. *All solutions* $S_C(t), V_C(t), E_C(t), I_C(t), R_C(t), S_B, E_B, I_B > 0$ *are bounded.*

Proof. To prove boundedness, having assured that we are dealing with positive solutions in positive feasible region. We can now prove that for all $t > 0$, $S_C(t), V_C(t), E_C(t), I_C(t), R_C(t) > 0$ will be bounded above. Model (4.10) is split into two, the cattle population and the buffaloes population but in this section only to show boundedness on cattle population since for buffaloes remain the same as in chapter 4.

Cattle population

Given the total cattle population $N_V = S_C + V_C + E_C + I_C + R_C$.

By adding the right hand side of the first for equations of the model (4.10) such that:

$$\frac{dN_C}{dt} = \eta_C - \mu_1(S_C + V_C + E_C + I_C + R_C) - (\rho_1 + \theta)I_C, \tag{5.16}$$

$$\frac{dN_C}{dt} = \eta_C - \mu_1 N_C - (\rho_1 + \theta)I_C \leq \eta_C - \mu_1 N_C.$$

Now by integrating both sides of the equation using the integrating factor.

$$N_C \leq (N_V(0) - \frac{\eta_C}{\mu_1})e^{-\mu_1 t} + \mu_1 N_C.$$

Taking the limit supremum of N_C as $t \to \infty$

$$\lim_{t\to\infty} \sup N_C \leq \frac{\eta_C}{\mu_1}.$$

Since all state variables are positive and bounded above, therefore the feasible region is given by;

$$\Omega_V = \{(S_C(t), V_C(t), E_C(t), I_C(t), R_C(t)) \in \mathbb{R}^5_+; 0 \leq N_C(t) \leq \frac{\eta_C}{\mu_1 + \omega}\}$$

This proves that $(S_C(t), V_C(t), E_C(t), I_C(t), R_C(t)$ is bounded above. Thus, $\Omega = \Omega_C \times \Omega_V \times \Omega_B$ is the feasible region for the combined cattle, vaccinated cattle and buffaloes dynamics. □

5.2.3 Local and global stability of the DFE

Disease free equilibrium point

First let

$$\lambda_1^* = \tau_1 \beta_C I_B + \beta_C I_C,$$

$$\lambda_2^* = \tau_2 \beta_B I_C + \beta_B I_B.$$

We then find the equilibrium point of the system of equations:

$$\begin{aligned}
\eta_C - \mu_1 S_C^* - \lambda_1^* S_C^* + \sigma_1 R_C^* - \omega S_C^* &= 0, \\
\omega S_C^* - \mu_1 V_C^* - \lambda_1^* q V_C^* &= 0, \\
\lambda_1^* S_C^* + \lambda_1 q^* V_C^* - (\mu_1 + \alpha_1) E_C^* &= 0, \\
\alpha_1 E_C^* - (\mu_1 + \rho_1 + \phi_1 + \theta) I_C^* &= 0, \\
\phi_1 I_C^* - (\mu_1 + \sigma_1) R_C^* &= 0, \\
\eta_B - \mu_2 S_B^* - \lambda_2^* S_B^* &= 0, \\
\lambda_2^* S_B^* - (\mu_2 + \alpha_2) E_B^* &= 0, \\
\alpha_2 E_B^* - \mu_2 I_B^* &= 0.
\end{aligned} \tag{5.17}$$

Given the DFE point as E_1, where

$E_1 = (S_C^*, V_C^*, E_C^*, I_C^*, R_C^*, S_B^*, E_B^*, I_B^*) = (S_C^0, V_C^0, 0, 0, 0, S_B^0, 0, 0)$, where $S_C^0 = \frac{\eta_C}{\mu_1 + \omega}$, $V_C^0 = \frac{\omega S_C^0}{\mu_1}$ and $S_B^0 = \frac{\eta_B}{\mu_2}$.

5.2.4 The effective reproduction number ($\mathcal{R}_e$)

The effective model reproduction rate for the model (5.17) is given by $\mathcal{R}_e$. Using the technique by van den Driessche and Watmough, $\mathcal{R}_e$ is calculated as follows:

We consider a matrix $\mathscr{F}_i$, the rate of appearances of new infections in compartment i,

$$\mathscr{F} = \begin{bmatrix} \tau_1\beta_C S_C I_B + \beta_C S_C I_C + \tau_1\beta_C q V_C I_B + \beta_C q V_C I_C \\ 0 \\ \tau_2\beta_B S_B I_C + \beta_B S_B I_B \\ 0 \end{bmatrix}.$$

The Jacobian matrix of $\mathscr{F}$, for disease free equilibrium is given by,

$$F = \begin{bmatrix} 0 & \tau_1\beta_C(S_C^0 + qV_C^0) & 0 & \beta_C(S_C^0 + qV_C^0) \\ 0 & 0 & 0 & 0 \\ 0 & \tau_2\beta_B S_B^0 & 0 & \beta_B S_B^0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

We consider the transition matrix $\mathscr{V}$ as:

$$\mathscr{V} = \begin{bmatrix} (\mu_1 + \alpha_1)E_C \\ (\mu_1 + \rho_1 + \phi_1 + \theta)I_C - \alpha_1 E_C \\ (\mu_2 + \alpha_2)E_B \\ \mu_2 I_B - \alpha_2 E_B \end{bmatrix}.$$

Let $v_1 = (\mu_1 + \alpha_1), v_2 = (\mu_1 + \rho_1 + \phi_1 + \theta), v_3 = (\mu_2 + \alpha_2)$. The Jacobian matrix of $\mathscr{V}$ evaluated at the disease free equilibrium point E_1 is given by:

$$V = \begin{pmatrix} \nu_1 & 0 & 0 & 0 \\ -\alpha_1 & \nu_2 & 0 & 0 \\ 0 & 0 & \nu_3 & 0 \\ 0 & 0 & -\alpha_2 & \mu_2 \end{pmatrix}.$$

The inverse of V is therefore found as:

$$V^{-1} = \begin{pmatrix} \frac{1}{\nu_1} & 0 & 0 & 0 \\ \frac{\alpha_1}{\nu_1\nu_2} & \frac{1}{\nu_2} & 0 & 0 \\ 0 & 0 & \frac{1}{\nu_3} & 0 \\ 0 & 0 & \frac{\alpha_2}{\mu_2\nu_3} & \frac{1}{\mu_2} \end{pmatrix}.$$

Hence, the next generation matrix denoted by FV^{-1} is given by:

$$FV^{-1} = \begin{pmatrix} 0 & \tau_1\beta_C(S_C^0+qV_C^0) & 0 & \beta_C(S_C^0+qV_C^0) \\ 0 & 0 & 0 & 0 \\ 0 & \tau_2\beta_B S_B^0 & 0 & \beta_B S_B^0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\nu_1} & 0 & 0 & 0 \\ \frac{\alpha_1}{\nu_1\nu_2} & \frac{1}{\nu_2} & 0 & 0 \\ 0 & 0 & \frac{1}{\nu_3} & 0 \\ 0 & 0 & \frac{\alpha_2}{\mu_2\nu_3} & \frac{1}{\mu_2} \end{pmatrix},$$

$$= \begin{pmatrix} \frac{\tau_1\beta_C(S_C^0+qV_C^0)}{\nu_1\nu_2} & \frac{\beta_C(S_C^0+qV_C^0\alpha_1)}{\nu_2} & \frac{\tau_1\beta_C(S_C^0+qV_C^0\alpha_2)}{\mu_2\nu_3} & \frac{\beta_C(S_C^0+qV_C^0\alpha_1)}{\mu_2} \\ 0 & 0 & 0 & 0 \\ \frac{\tau_2\beta_B S_B^0\alpha_1}{\nu_1\nu_2} & \frac{\tau_2\beta_B S_B^0}{\nu_2} & \frac{\beta_B S_B^0\alpha_2}{\mu_2\nu_3} & \frac{\beta_B S_B^0}{\mu_2} \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

Let $d_1 = \frac{\tau_1\beta_C(S_C^0+qV_C^0)}{\nu_1\nu_2}$, $d_2 = \frac{\beta_C(S_C^0+qV_C^0\alpha_1)}{\nu_2}$, $d_3 = \frac{\tau_1\beta_C(S_C^0+qV_C^0\alpha_2)}{\mu_2\nu_3}$, $d_4 = \frac{\beta_C(S_C^0+qV_C^0\alpha_1)}{\mu_2}$, $d_5 = \frac{\tau_2\beta_B S_B^0\alpha_1}{\nu_1\nu_2}$, $d_6 = \frac{\tau_2\beta_B S_B^0}{\nu_2}, d_7 = \frac{\beta_B S_B^0\alpha_2}{\mu_2\nu_3}$, $d_8 = \frac{\beta_B S_B^0}{\mu_2}$.

Then,

$$\mathcal{R}_e = \frac{1}{2}\left[(d_1+d_7) + \sqrt{(d_1-d_7)^2+4d_3d_5}\right].$$

If the virus is to persist in the vaccinated population. i.e $I_C > 0$ and $I_B > 0$ which occurs when $\mathcal{R}_e > 1$. It follows that disease eradication will eventually occur when $\mathcal{R}_e < 1$. In this case $\mathcal{R}_0 > \mathcal{R}_e$ since $0 < \omega < 1$.

$\mathcal{R}_0 = \pi\mathcal{R}_e$, where π is constant parameter.

This shows that FMD intervention strategies have positive impact on controlling FMD in the community.

5.2.5 Local stability analysis

In this part, to show stability it is omitted here since global stability implies local stability.

5.2.6 The global asymptotic stability of the disease free equilibrium

Using linear stability analysis, we can determine the local stability of each equilibrium. However, even for simple systems, this does not always give us a clear biological interpretation on when disease will spread or die out globally.

Theorem 7. *The disease free equilibrium E_1 of the system is globally asymptotically stable in the positive invariant region Ω if $\mathcal{R}_e \leq 1$.*

Proof. In this proof, we are using the concept of Liapunov function.

Let us define a function

Consider a Liapunov function as follows:

$$V_0 = d_1E_C + d_2I_C + d_3R_C + d_4E_B + d_5I_B,$$

By differentiating L_0 with respect to time t along the solutions of the model we get,

$$\dot{V_0} = d_1\dot{E_C} + d_2\dot{I_C} + d_3\dot{d_C} + d_4\dot{E_B} + d_5\dot{I_B},$$

Let $w_1 = \mu_1 + \alpha_1, \quad w_2 = \mu_1 + \rho_1 + \phi_1 + \theta, \quad w_3 = \mu_1 + \sigma_1, \quad$ and $w_4 = \mu_2 + \alpha_2$.

Note that $S_C \leq \frac{\eta_C}{\mu_1+\omega}$ $\quad ,V_C \leq \frac{\omega S_C^0}{\mu_1}$ and $S_B \leq \frac{\eta_B}{\mu_2}$ at DFE. Now by replacing the derivatives $\dot{E}_C, \dot{I}_C, \dot{R}_C, \dot{E}_B, \dot{I}_B$ into the equation of $\dot{V}_0$ we obtain,

$$\dot{V}_0 = d_1[\beta_C(\tau_1 I_B + I_C)S_C + \beta_C q(\tau_1 I_B + I_C)V_C - w_1 E_C] + d_2(\alpha_1 E_C - w_2 I_C) + d_3(\phi_1 I_C - w_3 R_C)$$

$$+d_4[\beta_B(\tau_2 I_C + I_B)S_B - w_4 E_B] + d_5(\alpha_2 E_B - \mu_2 I_B)$$

$$\leq d_1[\beta_C(\tau_1 I_B + I_C) + \beta_C q(\tau_1 I_B + I_C) - w_1 E_C] + d_2(\alpha_1 E_C - w_2 I_C) + d_3(\phi_1 I_C - w_3 R_C)$$

$$+d_4[\beta_B(\tau_2 I_C + I_B) - w_4 E_B] + d_5(\alpha_2 E_B - \mu_2 I_B),$$

$$= d_1\beta_C\tau_1 I_B + d_1\beta_C I_C + d_1\beta_C q\tau_1 I_B + d_1\beta_C q I_C - w_1 E_C + d_2\alpha_1 E_C - w_2 d_2 I_C + d_3\phi_1 I_C - d_3 b_3 R_C$$

$$+d_4\beta_B\tau_2 I_C + d_4\beta_B I_B - w_4 b_4 E_B + d_5\alpha_2 E_B - d_5\mu_2 I_B,$$

Collecting the linear terms of E_C, I_C, R_C, E_B, I_B and setting the coefficients E_C, I_C, R_C, E_B, I_B to zero we get,

$$= [d_2\alpha_1 - d_1 w_1]E_C + [d_1\beta_C - d_2 w_2 + d_3\phi_1 + d_1 q\beta_C + d_4\beta_B\tau_2]I_C - d_3 w_3 R_C + [d_5\alpha_2 - d_4 w_4]E_B +$$

$$[d_1\beta_C\tau_1 - d_5\mu_2 + d_1 q\tau_1\beta_C]I_B = 0.$$

Now solving for d_1, d_2, d_3, d_4 and d_5 we get,

$d_1 = -\frac{d_4(\alpha_2\beta_B - w_4\mu_2)}{(1+q)\alpha_2\beta_C\tau_1}$, $\quad d_2 = -\frac{d_4 w_1(\alpha_2\beta_B - w_4\mu_2)}{(1+q)\alpha_1\alpha_2\beta_C\tau_1}$, $\quad d_3 = 0$, $\quad d_5 = \frac{d_4 w_4}{\alpha_2}$,

Let $d_4 = 1$, then this yields,

$$\begin{pmatrix} d_1 \\ d_2 \\ d_3 \\ d_4 \\ d_5 \end{pmatrix} = \begin{pmatrix} -\frac{(\alpha_2\beta_B - w_4\mu_2)}{(1+q)\alpha_2\beta_C\tau_1} \\ -\frac{w_1(\alpha_2\beta_B - w_4\mu_2)}{(1+q)\alpha_1\alpha_2\beta_C\tau_1} \\ 0 \\ 1 \\ \frac{w_4}{\alpha_2} \end{pmatrix},$$

$$= \frac{1}{\alpha_1\alpha_2\beta_C\tau_1(1+q)} \begin{pmatrix} \alpha_1(w_4\mu_2 - \alpha_2\beta_B) \\ w_1(w_4\mu_2 - \alpha_2\beta_B) \\ 0 \\ \alpha_1\alpha_2\beta_C\tau_1(1+q) \\ w_4\alpha_1\beta_C\tau_1(1+q) \end{pmatrix},$$

So, $d_1 = \alpha_1(w_4\mu_2 - \alpha_2\beta_B)$, $\quad d_2 = w_1(w_4\mu_2 - \alpha_2\beta_B)$, $\quad d_3 = 0$, $\quad d_4 = \alpha_1\alpha_2\beta_C\tau_1(1+q)$ $\quad$ and $d_5 = w_4\alpha_1\beta_C\tau_1(1+q)$.

So by factorise d_1 and d_2 we get,

$$d_1 = \alpha_1 w_4\mu_2(1 - \mathcal{R}_B),$$

$$d_2 = w_1 w_4\mu_2(1 - \mathcal{R}_B),$$

$\mathcal{R}_B < 1$ for d_1 and $d_2 > 0$,

where $\mathcal{R}_B = \frac{\alpha_2\beta_B}{w_4\mu_2}$

$\implies \dot{V}_0 \leq [d_1\beta_C - d_2 w_2 + d_3\phi_1 + d_1 q\beta_C + d_4\beta_B\tau_2]I_C,$

$$= [\alpha_1 w_4\mu_2\beta_C(1 - \mathcal{R}_B) + \alpha_1 w_4\mu_2\beta_C q(1 - \mathcal{R}_B) + \alpha_1\beta_C\alpha_2\tau_1\beta_B\tau_2(1+q) - w_1 w_2 w_4\mu_2(1 - \mathcal{R}_B)]I_C,$$

$$= \mu_2 w_4(1 - \mathcal{R}_B)(\alpha_1\beta_C + \alpha_1\beta_C q - w_1 w_2) + \alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1+q)]I_C,$$

$$= [\mu_2 w_4 w_1 w_2(1 - \mathcal{R}_B)\left(\frac{\alpha_1\beta_C(1+q)}{w_1 w_2} - 1\right) + \alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1+q)]I_C,$$

$$= [\alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1+q) - \mu_2 w_4 w_1 w_2(1 - \mathcal{R}_B)(1 - \mathcal{R}_C)]I_C,$$

where $\mathcal{R}_C = \frac{\alpha_1\beta_C(1+q)}{w_1w_2}$

$$= \alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1+q)\left[1 - \frac{\mu_2 w_1 w_2 w_4(1-\mathcal{R}_B)(1-\mathcal{R}_C)}{\alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1+q)}\right] I_C.$$

Therefore,

$$\dot{V}_0 \leq \alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1-\mathcal{R}_e)I_C.$$

$$= -\alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1-\mathcal{R}_e)I_C$$

$\implies \dot{V}_0 \leq -M(1-\mathcal{R}_e)$,

where $M = \alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B$ is a constant and

$$\mathcal{R}_e = \frac{\mu_2 w_1 w_2 w_4(1-\mathcal{R}_B)(1-\mathcal{R}_C)}{\alpha_1\alpha_2\tau_1\tau_2\beta_C\beta_B(1+q)}.$$

Thus, $\dot{V}_0 \leq 0$ as long as $\mathcal{R}_e \leq 1$. When $\mathcal{R}_e < 1$, $\dot{V}_0 = 0$ yields $I_C = 0$. Then, it can be observed from the system that when $t \to \infty$, $E_C \to 0$, $R_C \to 0$, $E_B \to 0$, $I_B \to 0$ and $I_B \to 0$ and $S_C \to S_C^0$, $V_C \to V_C^0$, $S_B \to S_B^0$. Therefore, the largest compact invariant set such that $\dot{V}_0 = 0$ when $\mathcal{R}_e \leq 1$, is the singleton $E_1 = (S_C^0, V_C^0, 0, 0, 0, S_B^0, 0, 0)$. Thus, by LaSalle Invariance Principle [56], E_1 is globally asymptotically stable when $\mathcal{R}_e \leq 1$. This completes the proof.

□

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Local stability of endemic equilibrium

We first find the unique endemic equilibrium point then show that it is locally and globally stable. Let $f_1 = \mu_1 + \omega, f_2 = \alpha_1 + \mu_1, f_3 = \mu_1 + \rho_1 + \phi_1 + \theta, f_4 = \mu_1 + \sigma_1$ and $f_5 = \mu_2 + \alpha_2$.

$$\eta_C - (f_1 + \lambda_1) S_C^{**} + \eta_C + \sigma_1 R_C^{**} = 0,$$

$$\implies S_C^{**} = \frac{\eta_C + \sigma_1 R_C^{**}}{f_1 + \lambda_1^{**}}.$$

$$\omega S_C^{**} - V_C^{**}\left(q\lambda_1^{**} + \mu_1\right) = 0,$$

$$\implies V_C^{**} = \frac{\omega\left(\eta_C + \sigma_1 R_C^{**}\right)}{\left(f_1 + \lambda_1^{**}\right)\left(\mu_1 + \mathrm{q}\lambda_1^{**}\right)}.$$

$$E_C^{**} = \lambda_1^{**} S_C^{**} + q\lambda_1^{**} V_C^{**} - f_2 E_C^{**} = 0,$$

$$\implies E_C^{**} = \frac{\left(\eta_c + \sigma_1 R_C^{**}\right)\left(\lambda_1^{**}\mu_1 + \mathrm{q}\lambda_1^{**}\left(\lambda_1^{**} + \omega\right)\right)}{f_2\left(f_1 + \lambda_1^{**}\right)\left(\mu_1 + \mathrm{q}\lambda_1^{**}\right)}.$$

$$\alpha_1 E_C^{**} - f_3 I_C^{**} = 0,$$

$$\implies I_C^{**} = \frac{\alpha_1\left(\eta_c + \sigma_1 R_C^{**}\right)\left(\lambda_1^{**}\mu_1 + \mathrm{q}\lambda_1^{**}\left(\lambda_1^{**} + \omega\right)\right)}{f_2 f_3\left(f_1 + \lambda_1^{**}\right)\left(\mu_1 + \mathrm{q}\lambda_1^{**}\right)}.$$

$$\phi_1 I_C^{**} - f_4 R_C^{**} = 0,$$

$$\implies R_C^{**} = -\frac{\alpha_1\phi_1\eta_c\left(\lambda_1^{**}\mu_1 + \mathrm{q}\lambda_1^{**}\left(\lambda_1^{**} + \omega\right)\right)}{\alpha_1\sigma_1\phi_1\left(\lambda_1^{**}\mu_1 + \mathrm{q}\lambda_1^{**}\left(\lambda_1^{**} + \omega\right)\right) - f_2 f_3 f_4\left(a_1 + \lambda_1^{**}\right)\left(\mu_1 + \mathrm{q}\lambda_1^{**}\right)}.$$

For buffaloes $S_B^{**} = \frac{\eta_b}{\lambda_2+\mu_2}, E_B^{**} = \frac{\lambda_2\eta_b}{f_5(\lambda_2+\mu_2)}, I_B^{**} = \frac{\alpha_2\lambda_2\eta_b}{f_5\mu_2(\lambda_2+\mu_2)}$.

Then by substitute R_C we have,

$$S_C^{**} = \frac{f_2 f_3 f_4 \eta_C(\mu_1 + q\lambda_1^{**})}{f_2 f_3 f_4\lambda_1^{**}(\mu_1 + q\lambda_1^{**}) + f_1 f_2 f_3 f_4(\mu_1 + q\lambda_1^{**}) - \alpha_1\sigma_1\phi_1(\lambda_1^{**}\mu_1 + q\lambda_1^{**}(\lambda_1^{**} + \omega))},$$

$$V_C^{**} = -\frac{f_2 f_3 f_4 \omega\eta_C}{-f_2 f_3 f_4\lambda_1^{**}(\mu_1 + q\lambda_1^{**}) - f_1 f_2 f_3 f_4(\mu_1 + q\lambda_1^{**}) + \alpha_1\sigma_1\phi_1(\lambda_1^{**}\mu_1 + q\lambda_1^{**}(\lambda_1^{**} + \omega))},$$

$$E_C^{**} = \frac{f_3 f_4 \eta_C(\lambda_1^{**}\mu_1 + q\lambda_1^{**}(\lambda_1^{**} + \omega))}{f_2 f_3 f_4\lambda_1^{**}(\mu_1 + q\lambda_1^{**}) + f_1 f_2 f_3 f_4(\mu_1 + q\lambda_1^{**}) - \alpha_1\sigma_1\phi_1(\lambda_1^{**}\mu_1 + q\lambda_1^{**}(\lambda_1^{**} + \omega))},$$

$$I_C^{**} = \frac{f_4\alpha_1 \eta_C(\lambda_1^{**}\mu_1 + q\lambda_1^{**}(\lambda_1^{**} + \omega))}{f_2 f_3 f_4\lambda_1^{**}(\mu_1 + q\lambda_1^{**}) + f_1 f_2 f_3 f_4(\mu_1 + q\lambda_1^{**}) - \alpha_1\sigma_1\phi_1(\lambda_1^{**}\mu_1 + q\lambda_1^{**}(\lambda_1^{**} + \omega))}.$$

Now finding the two forces of infection

$\lambda_1^* = \tau_1\beta_C I_B + \beta_C I_C,$

$$\implies \lambda_1^* = \frac{\alpha_2\tau_1\beta_C\lambda_2^*\eta_B}{f_5\mu_2\left(\lambda_2^*+\mu_2\right)} + \frac{f_4\alpha_1\beta_C\eta_C\left(\lambda_1^*\mu_1 + q\lambda_1\left(\lambda_1^*+\omega\right)\right)}{f_2f_3f_4\left(f_1+\lambda_1^*\right)\left(\mu_1+q\lambda_1^*\right) - \alpha_1\sigma_1\phi_1\left(\lambda_1^*\mu_1 + q\lambda_1^*\left(\lambda_1+\omega\right)\right)}.$$

$\lambda_2^* = \beta_B I_B + \tau_2\beta_B I_C,$

$$\implies \lambda_2^* = \frac{\alpha_2 c\beta_B\lambda_2^*\eta_B}{f_5\mu_2\left(\lambda_2^*+\mu_2\right)} + \frac{f_4\alpha_1 c\beta_B\eta_B\left(\lambda_1^*\mu_1 + q\lambda_1^*\left(\lambda_1^*+\omega\right)\right)}{f_2f_3f_4\left(f_1+\lambda_1^*\right)\left(\mu_1+q\lambda_1^*\right) - \alpha_1\sigma_1\phi_1\left(\lambda_1^*\mu_1 + q\lambda_1^*\left(\lambda_1^*+\omega\right)\right)}.$$

Let

$B_1 = \frac{c\beta_1\alpha_2\eta_B}{f_5\mu_2}, \quad B_2 = \tau_1\beta_C\alpha_1\eta_C f_4, \quad B_3 = f_2f_3f_4, \quad B_4 = \phi_1\alpha_1\sigma_1, \quad B_5 = \frac{\tau_2\beta_B\alpha_2\eta_B}{f_5\mu_2}$ and $B_6 = \beta_B\alpha_1\eta_C f_4$

Then

$$\lambda_1 = \frac{B_1\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{B_2q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1}{B_3(f_1+\lambda_1^*)(q\lambda_1^*+\mu_1) - B_4(q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1)},$$

$$\lambda_2 = \frac{B_5\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{B_6q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1}{B_3(f_1+\lambda_1^*)(q\lambda_1^*+\mu_1) - B_4(q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1)}.$$

Case 1: if

$\lambda_1^* = \lambda_2^* = 0$ then,

This corresponds to the disease-free equilibrium point where there are no viruses caused by infected buffaloes or infected cattle.

Case 2 : if

$\lambda_1^* \neq 0, \lambda_2^* \neq 0$ then,

This is where there exists both viruses from infected cattle and infected buffaloes among the cattle and buffaloes population.

The equilibrium points of the model can be obtained by finding the fixed points of the equations

$$\kappa(\lambda_1,\lambda_2) = \begin{bmatrix}\kappa_1(\lambda_1,\lambda_2)\\ \kappa_2(\lambda_1,\lambda_2)\end{bmatrix} = \begin{bmatrix}\frac{B_1\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{B_2q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1}{B_3(f_1+\lambda_1^*)(q\lambda_1^*+\mu_1)-B_4(q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1)}\\ \frac{B_5\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{B_6q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1}{B_3(f_1+\lambda_1^*)(q\lambda_1^*+\mu_1)-B_4(q\lambda_1^*(\omega+\lambda_1^*)+\lambda_1^*\mu_1)}\end{bmatrix}.$$

Theorem 8. *There exists a unique fixed point* $(\lambda_1^*, \lambda_2^*), \lambda_1^* > 0, \lambda_2^* > 0$ *satisfying*

$$\kappa(\lambda_1^*, \lambda_2^*) = \begin{bmatrix} \lambda_1^* \\ \lambda_2^* \end{bmatrix}$$

corresponding to the endemic equilibrium point.

Proof. In this case three conditions are considered.

By fixing $\lambda_1 > 0$, and considering the real valued functions depending on λ_1:

$$\kappa_1^{\lambda_1^*}(\lambda_2) = \frac{B_1\lambda_2^*}{\lambda_2^* + \mu_2} + \frac{B_2 q\lambda_1^*(\omega + \lambda_1^*) + \lambda_1^*\mu_1}{B_3(f_1 + \lambda_1^*)(q\lambda_1^* + \mu_1) - B_4(q\lambda_1^*(\omega + \lambda_1^*) + \lambda_1^*\mu_1)} = 0,$$

then

$$\frac{B_5\lambda_2^*}{\lambda_2^* + \mu_2} + \frac{B_6 q\lambda_1^*(\omega + \lambda_1^*) + \lambda_1^*\mu_1}{B_3(f_1 + \lambda_1^*)(q\lambda_1^* + \mu_1) - B_4(q\lambda_1^*(\omega + \lambda_1^*) + \lambda_1^*\mu_1)} > 0,$$

if

$$\frac{B_3(f_1 + \lambda_1^*)(q\lambda_1^* + \mu_1)}{(q\lambda_1^*(\omega + \lambda_1^*) + \lambda_1^*\mu_1)} > B_4.$$

Taking limit,

$$\lim_{\lambda_2 \to \infty} \kappa_1^{\lambda_1}(\lambda_2) = A_1 + \frac{A_2\lambda_1}{A_3(\lambda_1 + \mu_1) - A_4\lambda_1} < \infty.$$

Hence, $0 < \kappa_1^{\lambda_1}(\lambda_2) < \infty$, so that the function $\kappa_1^{\lambda_1}(\lambda_2)$ is bounded for every fixed $\lambda_1 > 0$.

Second condition is to find the first derivative of $\kappa_1^{\lambda_1}(\lambda_2)$ with respect to λ_2.

$$\frac{d\kappa_1^{\lambda_1^*}}{d\lambda_2} = \frac{A_1\mu_1}{(\lambda_1 + \mu_1)^2} > 0.$$

Now finding the third condition which is the second derivative given by;

$$\frac{d^2\kappa_1^{\lambda_1}}{d^2\lambda_2} = -2\left(\frac{A_1\mu_1}{(\lambda_1 + \mu_1)^2}\right) < 0.$$

Since $\frac{d\kappa_1^{\lambda_1}}{d\lambda_2} > 0$ and $\frac{d^2\kappa_1^{\lambda_1}}{d^2\lambda_2} < 0$, then the function $\kappa_1^{\lambda_1}(\lambda_2)$ is an increasing concave down function which is fixed in convexity in the bounded domain.

$\Longrightarrow$ there exist a unique point $\lambda_2^* > 0$ such that $\kappa_1^{\lambda_1^*}(\lambda_2^*) = \lambda_2^*$

By fixing $\lambda_2 > 0$, and considering the real valued functions depending on λ_2:

$\frac{A_5\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{A_6\lambda_1^*}{A_3(\lambda_1^*+\mu_1)-A_4\lambda_1^*}$

So by letting $\lambda_1 = 0$, we get,

$$F_2^{\lambda_1^*}(0) = \frac{A_5\lambda_2}{\lambda_2+\mu_2} > 0$$

Taking limit,

$$\lim_{\lambda_2\to\infty} F_2^{\lambda_2}(\lambda_1) = \frac{A_5\lambda_2^*}{\lambda_2^*+\mu_2} + \frac{A_6}{A_3-A_4} < \infty$$

Hence, $0 < F_1^{\lambda_1}(\lambda_2 < \infty)$ so that the function $F_1^{\lambda_1}(\lambda_2)$ is bounded for every fixed $\lambda_1 > 0$.

Second condition is to find the first derivative of $F_2^{\lambda_2}(\lambda_1)$ with respect to λ_1

$$\frac{dF_1^{\lambda_2^*}}{d\lambda_1} = \frac{A_3A_6\mu_1}{(\lambda_1(A_3-A_4)+A_3\mu_1)^2} > 0$$

Now finding the third condition which is the second derivative given by;

$$\frac{\partial^2 F_2^{\lambda_2}}{\partial^2\lambda_1} = \frac{-2A_3A_6\mu_1(A_3-A_4)}{(\lambda_1(A_3-A_4)+A_3\mu_1)^4} < 0.$$

Since $\frac{dF_2^{\lambda_2}}{d\lambda_1} > 0$ and $\frac{d^2F_1^{\lambda_1}}{d^2\lambda_2} < 0$, then the function $F_1^{\lambda_1}(\lambda_2)$ is an increasing concave down function which is fixed in convexity in the bounded domain.

$\Longrightarrow$ there exist a unique point $\lambda_1^* > 0$ such that $F_2^{\lambda_2}(\lambda_1^*) = \lambda_1^*$ $\square$

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Chapter 6

Numerical simulations

6.1 Introduction

This chapter presents numerical simulations to enhance the understanding of the predictions of the analytical results for the vaccination-culling model. We note that if we switch off the vaccination and culling processes, the model reduces to the basic model and hence, using the vaccination-culling model is sufficient to gain insights into scenarios without control. The data was obtained from published literature and also using Namibian data. We illustrate the simulation results using graphs plotted from *MATLAB* software.

6.2 Data fitting and parameter estimation

6.2.1 Data

We used the data for FMD in both cattle and buffaloes from the Ministry of Agriculture, Water and Forestry; Directorate of Veterinary Services in Namibia. Data was obtained as per region in Namibia where there were FMD outbreaks. Data were collected on sick animals, dead, those at risk and the animal testing results whether positive or negative. Therefore, in our study we used yearly cumulative data.

Figure 6.1: **Cumulative cases based on Namibia available data**

Years	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010
Data	1771	1778	1928	2111	2114	2185	2296	3102	3134	3203
Years	2011	2012	2013	2014	2015	2016	2017	2018	2019	
Data	3218	3541	3600	4000	4200	4500	4800	5200	5500	

6.2.2 Model fit with vaccination and parameter estimation values with initial conditions

In order to be able to perform numerical simulations, model parameter values were estimated by fitting the model to the cumulative cases of FMD as given in Table 5.1:

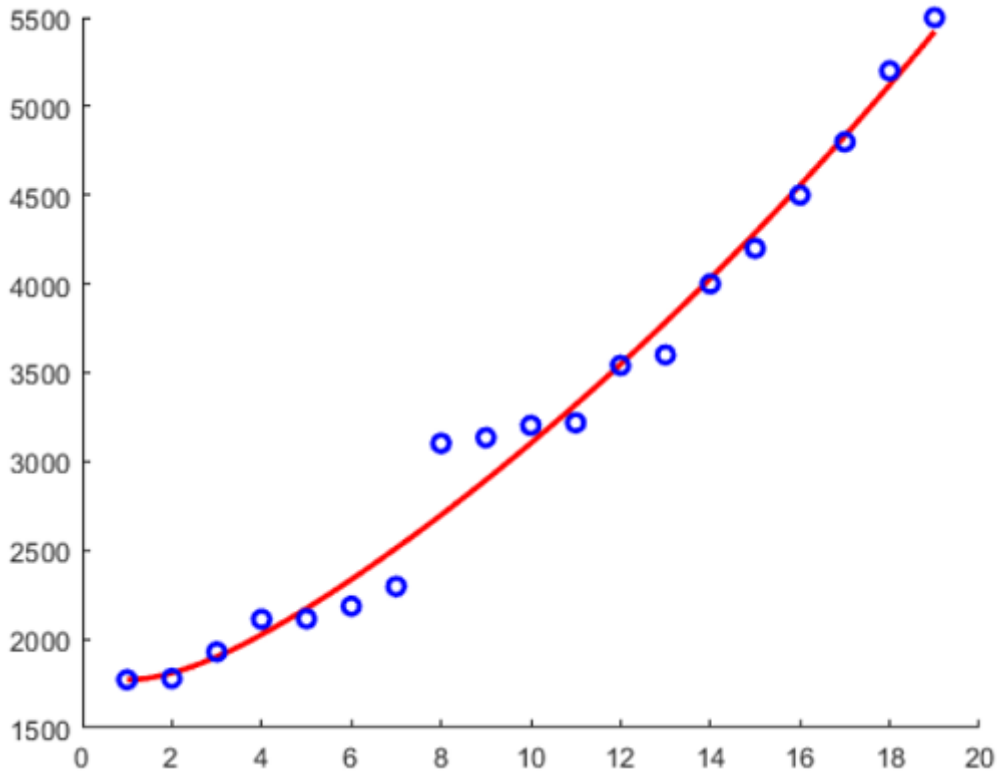

Figure 6.2: Model fit to data for FMD on cattle and buffaloes in Namibia using parameter values from published literature.

We used the method of least square curve fitting to fit model (5.7). The method is beneficial in obtaining the parameter values that are then used in the numerical simulations. Figure 6.2 shows that model (5.7) fits well to the FMD yearly cumulative cases of Namibian as from 2001 up to 2019, with data obtained from the published

literature $\mu_1 = 0.001$, $\alpha_1 = 0.26$, $\phi_1 = 0.143$ and $\rho_1 = 0.0056$ [3, 6, 18, 36, 58, 59, 60]. In the simulations, the initial conditions $(S_C(0), V_C(0), E_C(0), I_C(0), R_C(0), S_B(0), E_B(0), I_B(0)) = (20000, 0, 0, 1771, 0, 200, 0, 2)$ are used from Namibian cumulative cases. The curve fitting process generates the following parameters given in Table 6.1:

Table 6.1: Parameter estimates of FMD SEIRV model for Namibia and their values.

Symbol	**Parameter description**	**Value per year**	**Source**
η_C	cattle recruitment rate	0.2700	Fitted
τ_1&τ2	modification parameters	0.1002 & 0.9800	Fitted
β_C	buffaloes to cattle rate or cattle to cattle transmission rate	6.0000e-05	Fitted
μ_1	natural death rate	6.8000e-06	Fitted
σ_1	rate at which recovery cattle become susceptible	0.2800	Fitted
α_1	rate at which exposed cattle become infectious	0.4900	Fitted
ρ_1	disease induced death rate	2.5000e-05	Fitted
ϕ_1	rate at which infectious cattle recover	0.0361	Fitted
η_B	buffaloes recruitment rate	0.0120	Fitted
β_B	cattle to buffaloes or buffaloes to buffaloes transmission rate	1.7000e-05	Fitted
μ_2	buffaloes natural death rate	5.3000e-06	Fitted
α_2	rate at which exposed buffaloes become infectious	0.0021	Fitted
ε	vaccination efficacy	0.7375	Fitted
ω	vaccination rate	0.8248	Fitted
θ	culling rate	0	Fitted

For the model, the initial population were estimated using the data, number of cattle and buffaloes at risk of FMD as susceptible cattle and buffaloes and infected cattle and buffaloes as the first yearly cumulative sick cattle and buffaloes. We consider the cattle population size from herds to reach a steady-state $\frac{\eta_C}{\mu_1} = 20000$. The average cattle lifespan in Namibia is about 20 years, hence the natural death $\mu_1 = \frac{1}{20x365}$ $(yearly)$. For computational sake, we assume the year to have 365 days. Dividing the total population by natural death, this yields a recruitment rate of $\frac{200}{73}$ $(yearly)$. Cattle are highly susceptible to FMD and this enables the virus to spread rapidly to the entire herd [6]. Therefore, when susceptible cattle are infected, cattle to cattle transmission is expected to have a greater rate compared to cattle to buffalo transmission since cattle interact more with other cattle, than with buffaloes. In addition, when susceptible buffaloes are infected, buffalo to buffalo transmission is expected to have a greater rate compared to buffalo to cattle transmission since buffaloes interact more with each other, than with cattle.

6.3 Numerical simulations results

In order to illustrate the results of the foregoing analysis, we simulated a model system 5.7 using the MATLAB ODE solver, ode45 and parameter values in Table 6.1. The ode45 Runge-Kutta method is the most popular

method for solving ODEs by means of numerical approximations. We present the model simulations with these best fit parameter values as shown in Figure 6.3.

Figures 6.3 and 6.4 represent the population of cattle and buffaloes in the presence of vaccination and absence of culling. Figures 6.5 and 6.6 show the effects on FMD when vaccination efficacy dropped to zero and then increased. Moreover, Figures 6.7 and 6.8 depict the impact of vaccination rate on FMD when there is no vaccination and when vaccination rate has increased. Figures 6.9 and 6.10 represent the impact of culling on FMD when increased from zero to 20%. The rest of the figures show the impact of vaccination rate on all the four transmission rates.

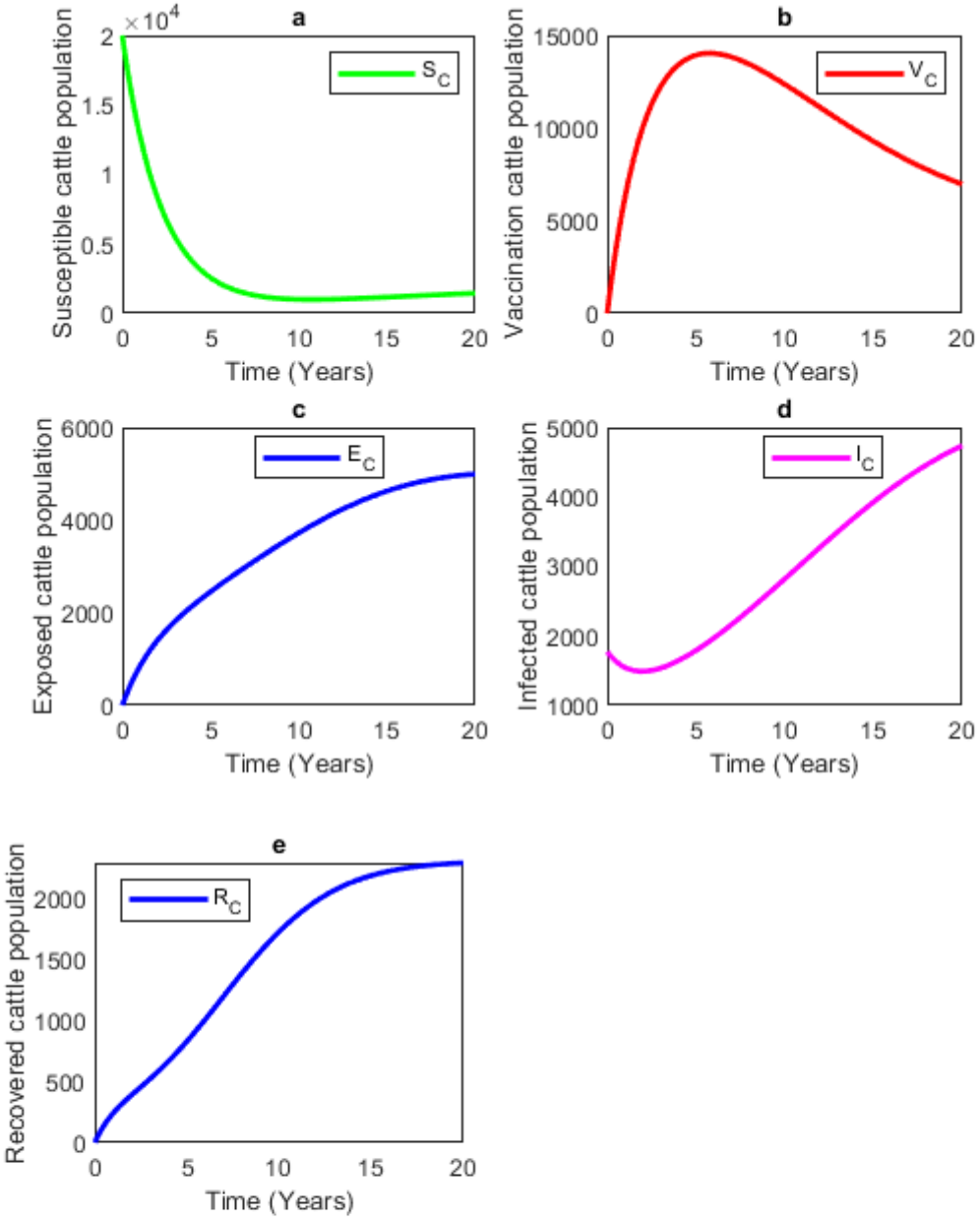

Figure 6.3: Simulations of model (4.18) showing the effects FMD transmission dynamics among cattle population demonstrated over a period of time (in years).

Figure 6.3 shows that if susceptible cattle interact with infected cattle or buffaloes, then the susceptible cattle population will decrease whilst vaccinated, exposed, infected and recovered populations increase. This suggests that vaccination is needed as is a potential to protect or reduce FMD infection among the cattle population.

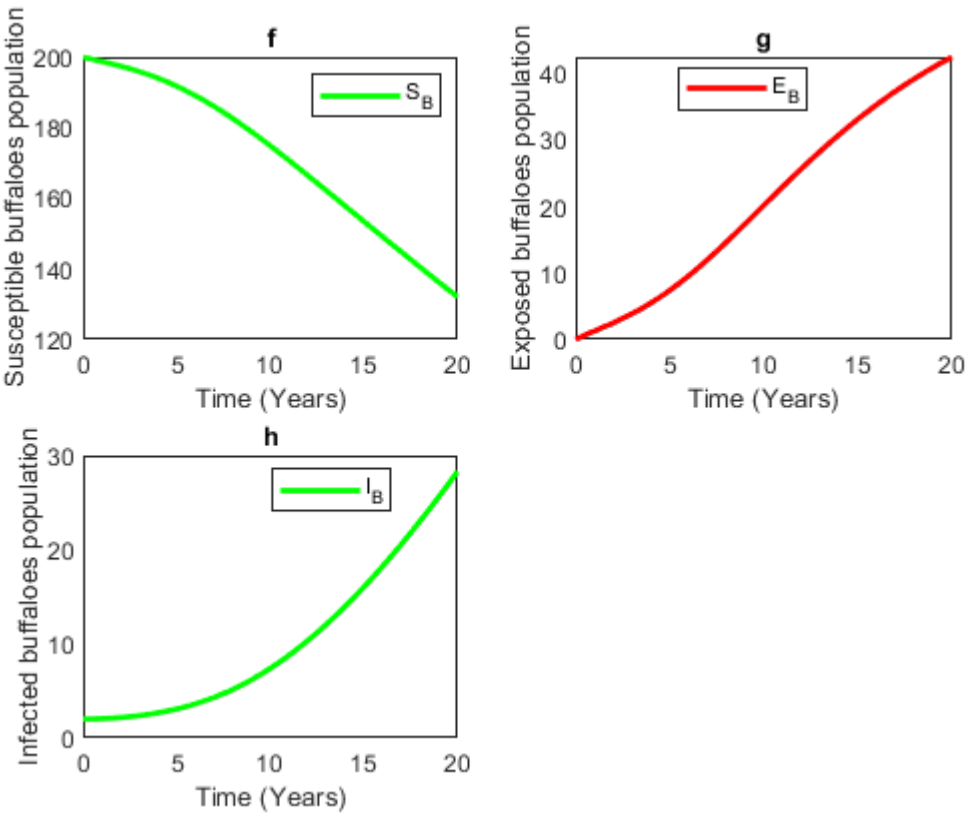

Figure 6.4: Simulations of model (4.18) showing FMD transmission dynamics among buffaloes population demonstrated over a period of time (in years)

Figure 6.4 simulations show that when susceptible cattle receive vaccination, then there will be a slight decrease in susceptible buffaloes and increase in exposed and infected buffaloes although not much as in cattle population. This suggests that vaccination of cattle may not completely protect the cattle population since buffaloes can still infect the cattle when they interact.

6.3.1 Vaccination numerical results

In this section, we explore the role of vaccination of cattle on minimizing cumulative FMD infections in cattle and buffaloes.

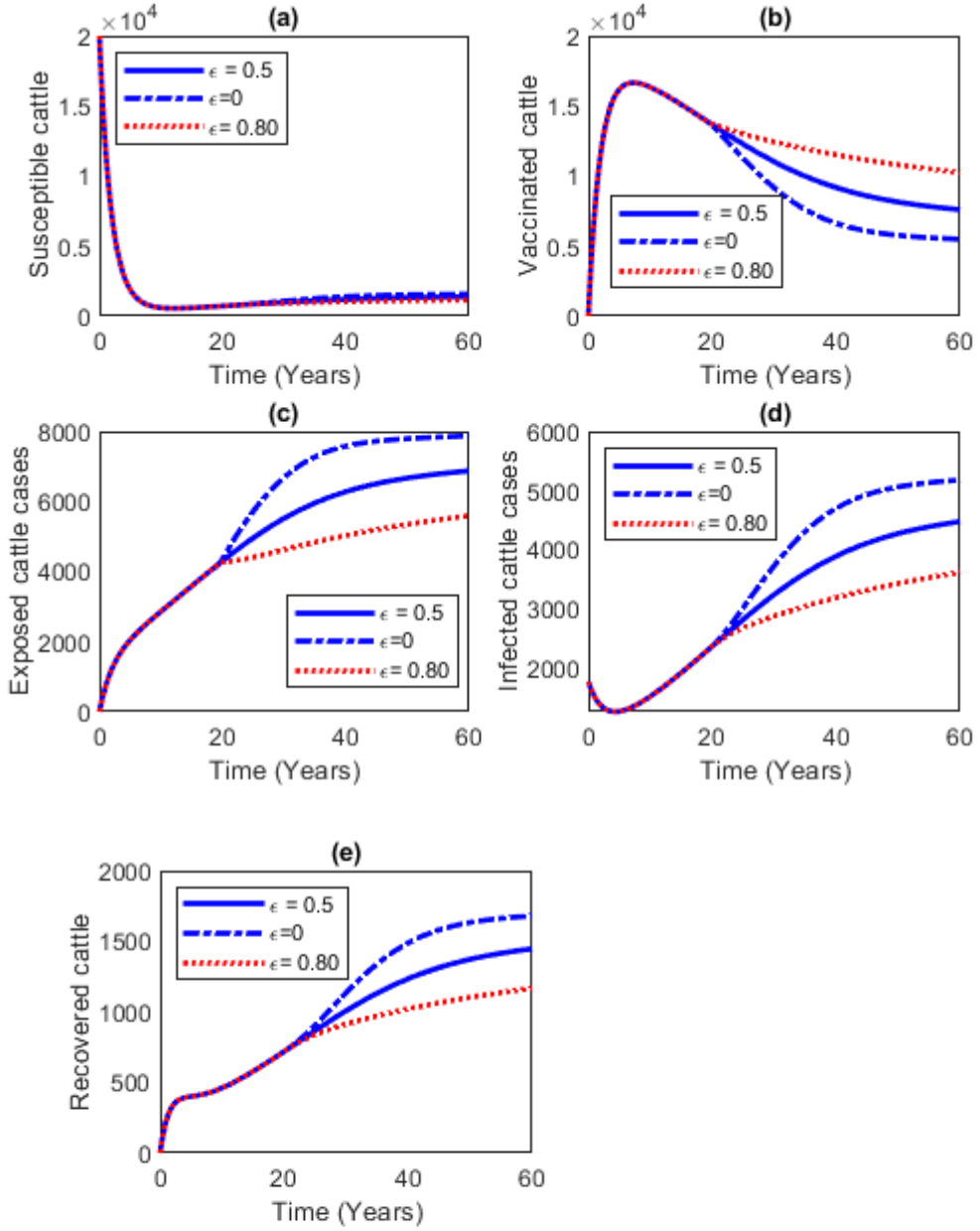

Figure 6.5: Simulations describe that $\varepsilon = 0.5$ is the fitted value for vaccination efficacy. From year 0 to year 19, $\varepsilon = 0.5$ after year 19, there are two addition scenarios: (i) the efficacy drop to zero or (ii) the efficacy increase to 0.8.

Figure 6.5 shows that if the vaccine loses its efficacy from 0.5 to 0, then recovered cattle, exposed cattle and infected cattle populations will increase but if the efficacy increases from 0.5 to 0.8, then the recovered cattle, exposed cattle and infected cattle populations will decrease. This suggests that the increase in the vaccination efficacy has potential to protect the cattle from FMD infection.

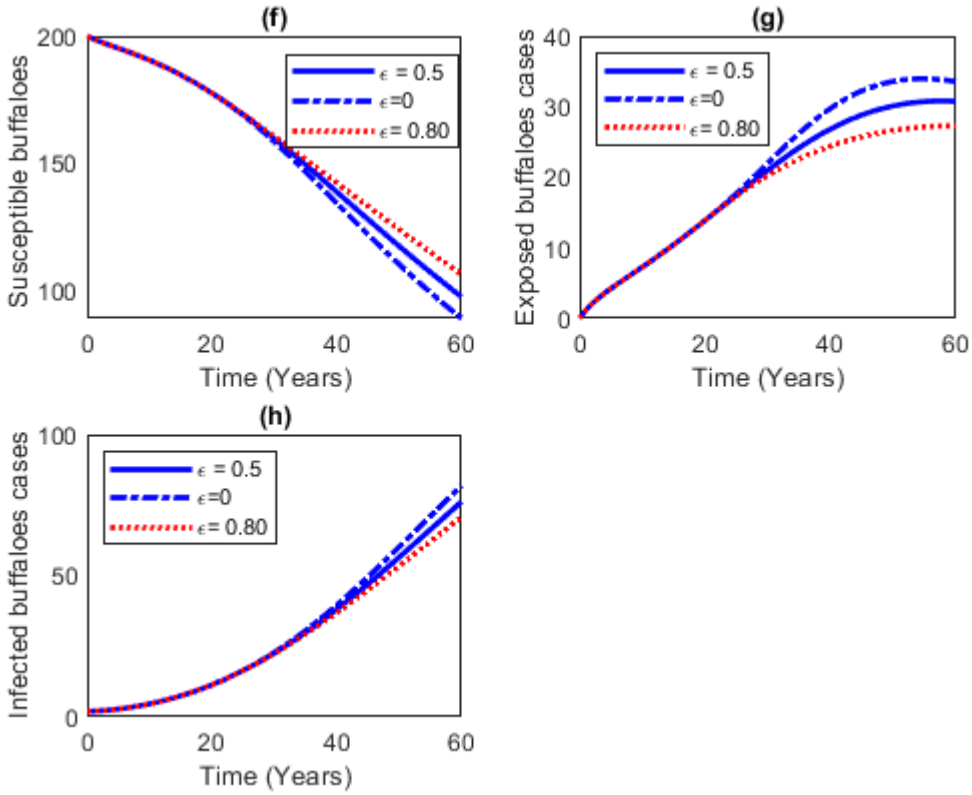

Figure 6.6: $\varepsilon = 0.5$ is the fitted value for vaccination efficacy. From year 0 to year 19, $\varepsilon = 0.8$. After year 19, there are two addition scenarios: (i) the efficacy drop to zero or (ii) the efficacy increase to 0.8.

Figure 6.6 shows that if the vaccine loses its efficacy from 0.5 to 0, then susceptible buffaloes will decrease whilst exposed buffaloes and infected buffaloes will increase but if the efficacy increases from 0.5 to 0.8, then the susceptible buffaloes increase but exposed buffaloes and infectious buffaloes decrease. This suggests that the increase in the vaccination efficacy on cattle populations has potential to reduce FMD infection in cattle and buffaloes populations but more significant in cattle.

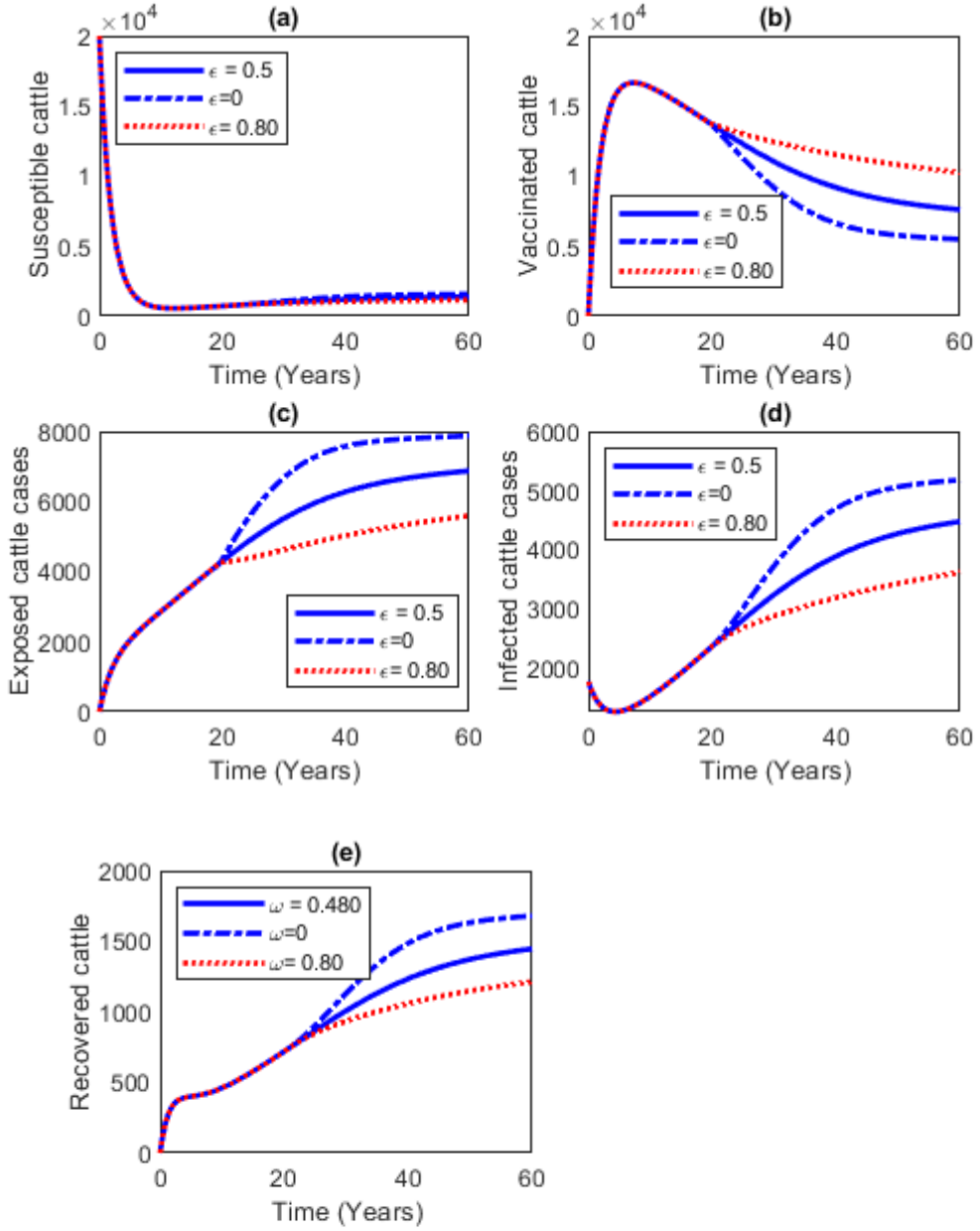

Figure 6.7: Effects of vaccination rate on FMD. $\omega = 0.480$ is the fitted value for vaccination rate from year 0 to year 19 as in table 6.1. After year 19, there are two scenarios (i) the vaccination rate drop to zero, (ii) the vaccination rate increases to 0.80

Figure 6.7 shows that if the vaccination stopped completely from 0.8248 to 0, then the exposed, infected and recovered cattle populations will increase, but if the vaccination rate increases from 0.480 to 0.80 then the exposed, infected and recovered cattle populations decrease. This suggests that the increase of vaccination rate has a positive impact on controlling FMD.

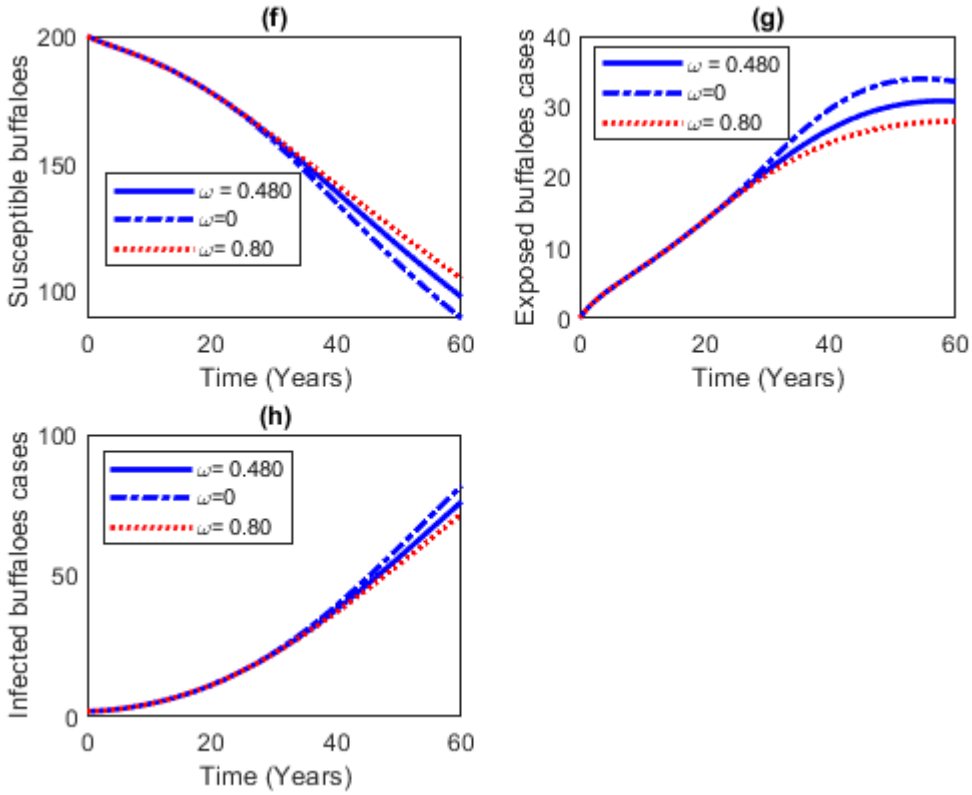

Figure 6.8: Effects of vaccination rate on FMD. Parameter value used is: $\omega = 0.480$ and the rest of the parameter values are fixed as in table 6.1

Figure 6.8 shows that if the vaccination is completely stopped from 0.480 to 0, then susceptible buffaloes will decrease whilst exposed and infected buffaloes will increase, but if the vaccination rate increases from 0.480 to 0.80 then susceptible buffaloes will increase but exposed and infected buffaloes decrease. This suggests that the increase of vaccination on cattle population has a potential to protect the cattle from FMD infection but according to the numerical results, it shows a slightly small difference. Therefore buffaloes are not really protected when the cattle are vaccinated. This shows that there is still a risk of FMD infection if buffaloes and cattle interact.

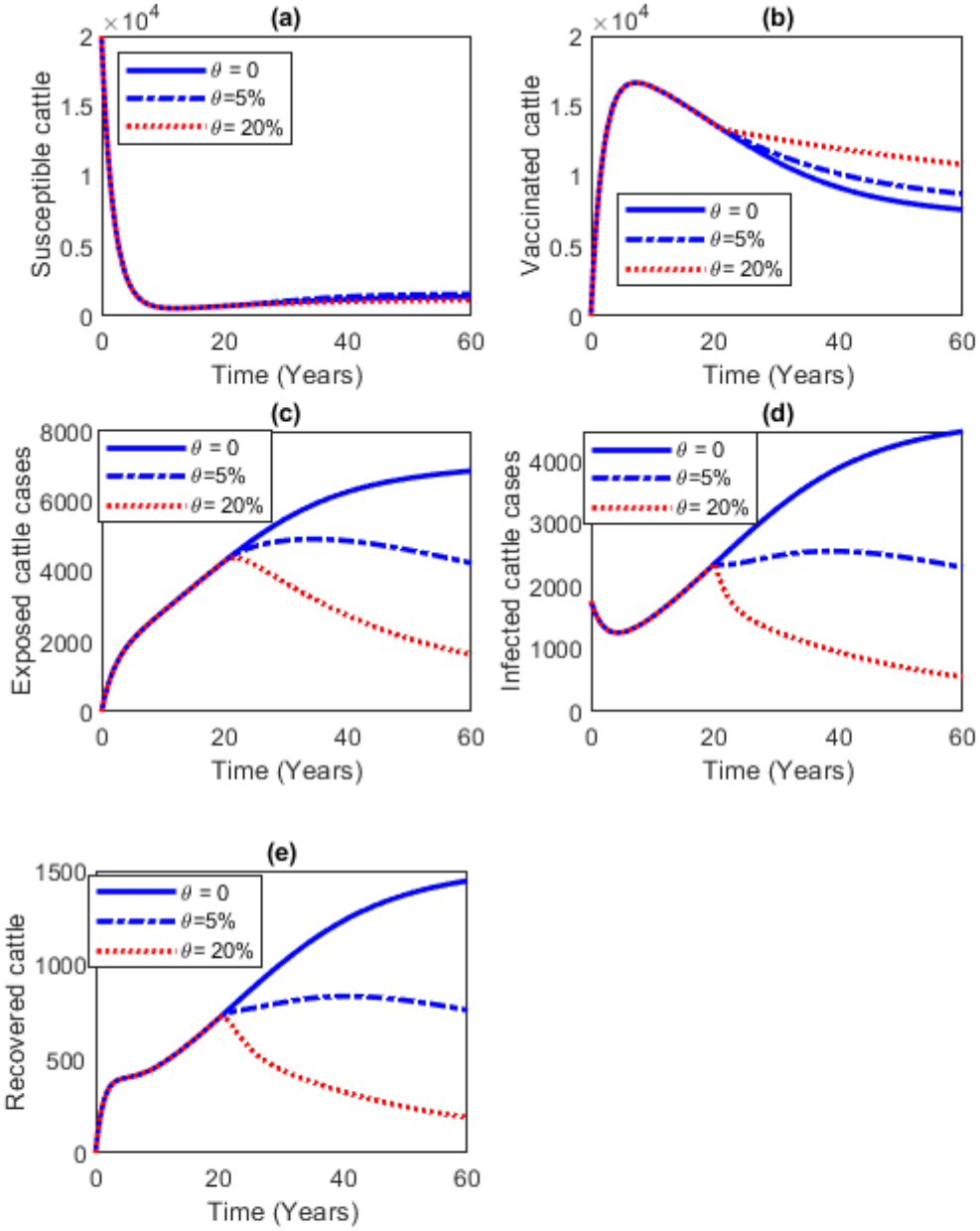

Figure 6.9: Simulations describe that $\theta = 0$ is the fitted value for culling. From year 0 to year 19, $\theta = 0$ after year 19, there are two addition scenarios: (i) the culling increase from zero to 5% or (ii) the culling increase from 0% to 20%.

Figure 6.9 simulations show that when culling rate increase then the vaccinated cattle will increase whilst recovered cattle, exposed cattle and infected cattle decreases significantly. Results demonstrated that culling has a positive impact on controlling FMD among cattle population. Although, vaccination will work better than culling.

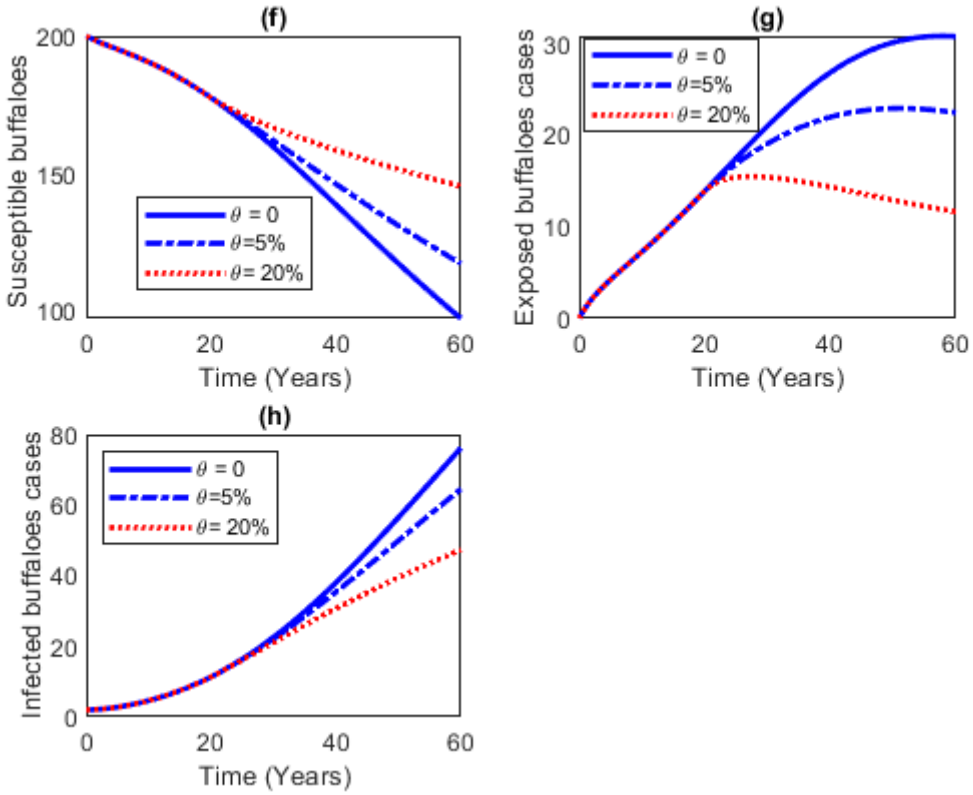

Figure 6.10: Effects of culling on FMD for buffaloes. Parameter value used is: $\theta = 0$ and the rest of the parameter values are fixed as in table 6.1

Figure 6.10 simulations show that when culling rate increases then the susceptible buffaloes increase whilst exposed buffaloes and infectious buffaloes decrease although not significantly compared to the cattle population. Results demonstrated that culling also has an impact on controlling FMD among buffaloes population.

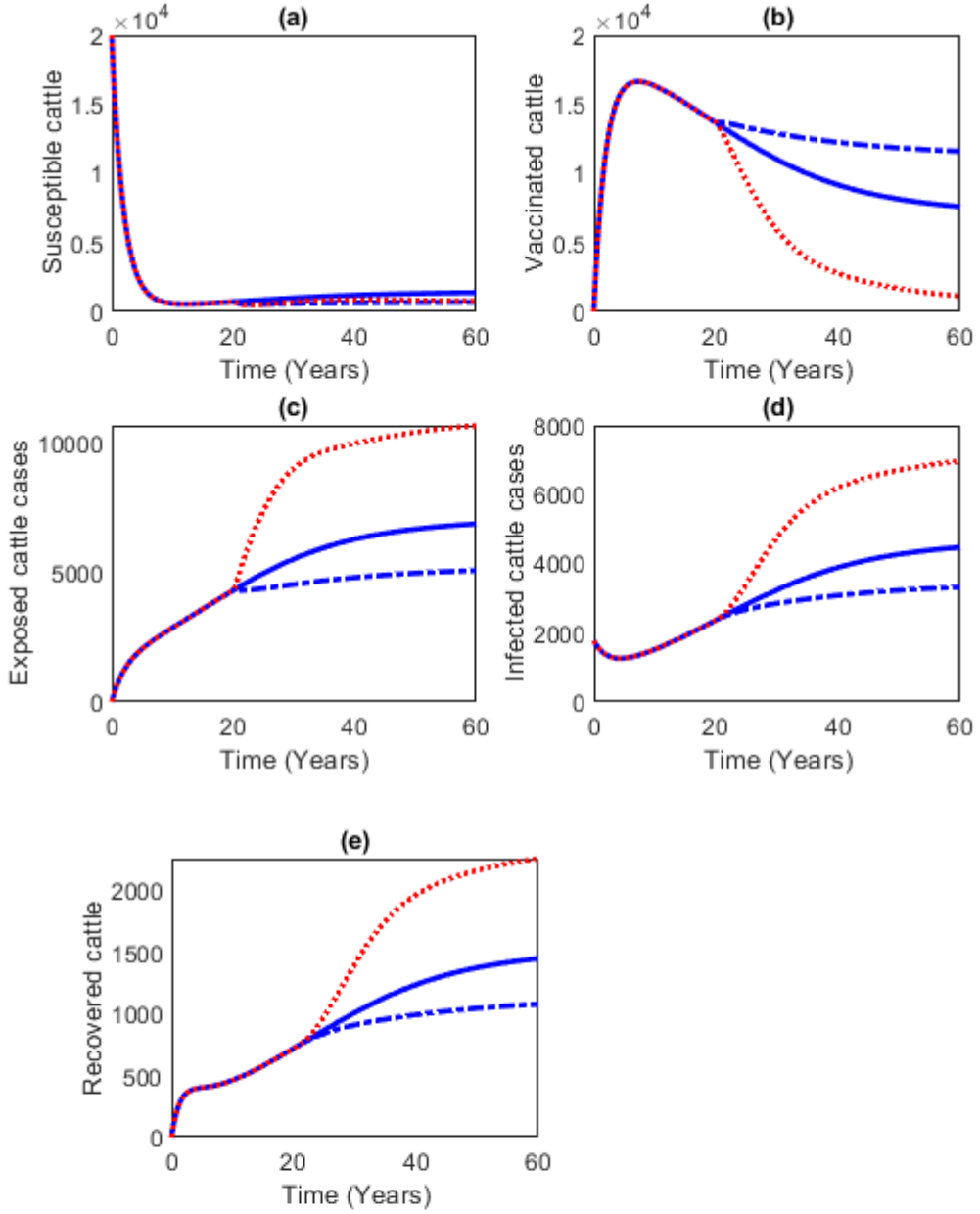

Figure 6.11: Given the cumulative $\omega = 0.480$ and $\beta_C = 6.000e-05$ represent by bold blue curve. The impact of vaccination rate on the transmission rate (FMD infection from buffaloes to susceptible cattle) on cattle population demonstrated over a period of 60 years:
(i) $\omega = 0.80$, $\beta_C = 0.0000008$ as vaccination increases and transmission rate decreases, represent by dashed blue curve
(ii)$\omega = 0.80$, $\beta_C = 0.04$ as both vaccination rate and transmission rate increase, represented by the red dotted curve. The rest of the parameters are fixed on table 6.1 .

Figure 6.11 simulations show that when vaccination rate increases and the transmission rate decreases from infected buffaloes to susceptible cattle, then there is a slight decrease in exposed and infected cattle populations.

Moreover, when the vaccination and transmission rates increase from infected buffaloes to susceptible cattle, then vaccinated cattle will decrease whilst recovered, exposed and infected cattle increase significantly. Results demonstrated that increasing of vaccination has a positive impact on reducing the transmission of FMD among the cattle population.

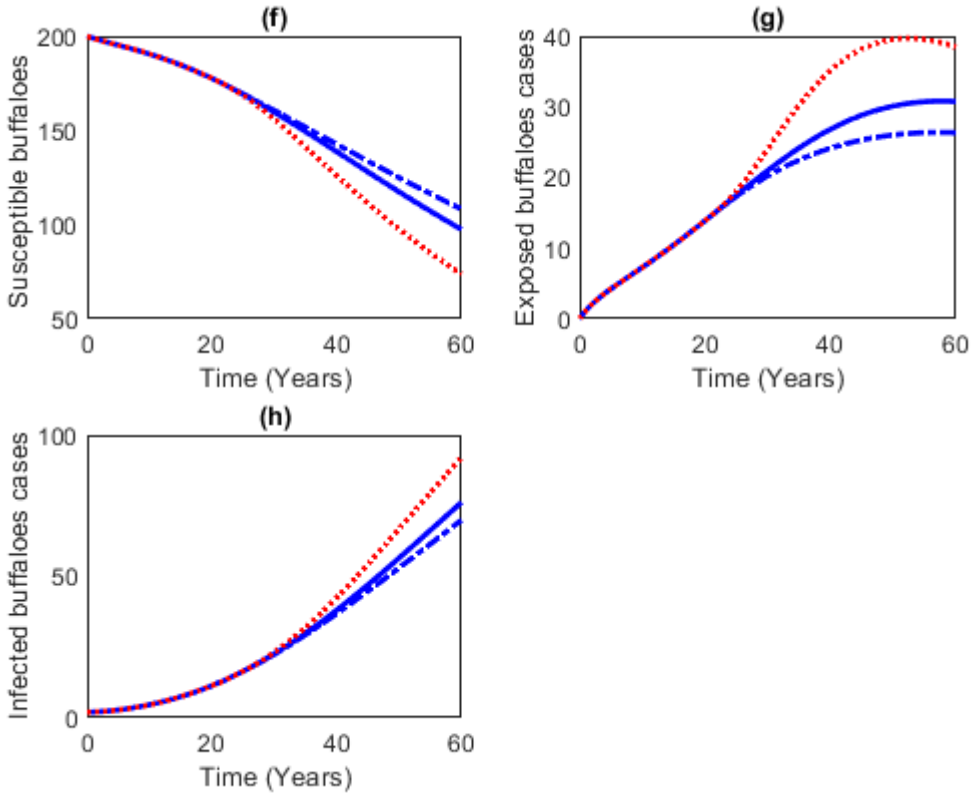

Figure 6.12: Given the cumulative $\omega = 0.480$ and $\beta_C = 6.000e-05$ (bold blue curve). The impact of vaccination rate on the transmission rate (FMD infection from buffaloes to susceptible cattle) on buffaloes population demonstrated over a period of 60 years:
(i) $\omega = 0.80$, $\beta_C = 0.04$ as vaccination increases and transmission rate decreases (blue dashed curve)
(ii)$\omega = 0.80$, $\beta_C = 0.04$ as both vaccination rate and transmission rate increase(dotted red curve).

Figure 6.12 simulations show that when vaccination rate increases and the transmission rate decreases from infected buffaloes to susceptible cattle, then there is a slight increase in the susceptible buffaloes population, whilst exposed and infected buffaloes population decrease. In addition, when vaccination and transmission rates increase then the susceptible buffaloes population decreases whilst exposed and infected buffaloes increase. This suggests that the increase of vaccination rate will reduce the risk of FMD infection.

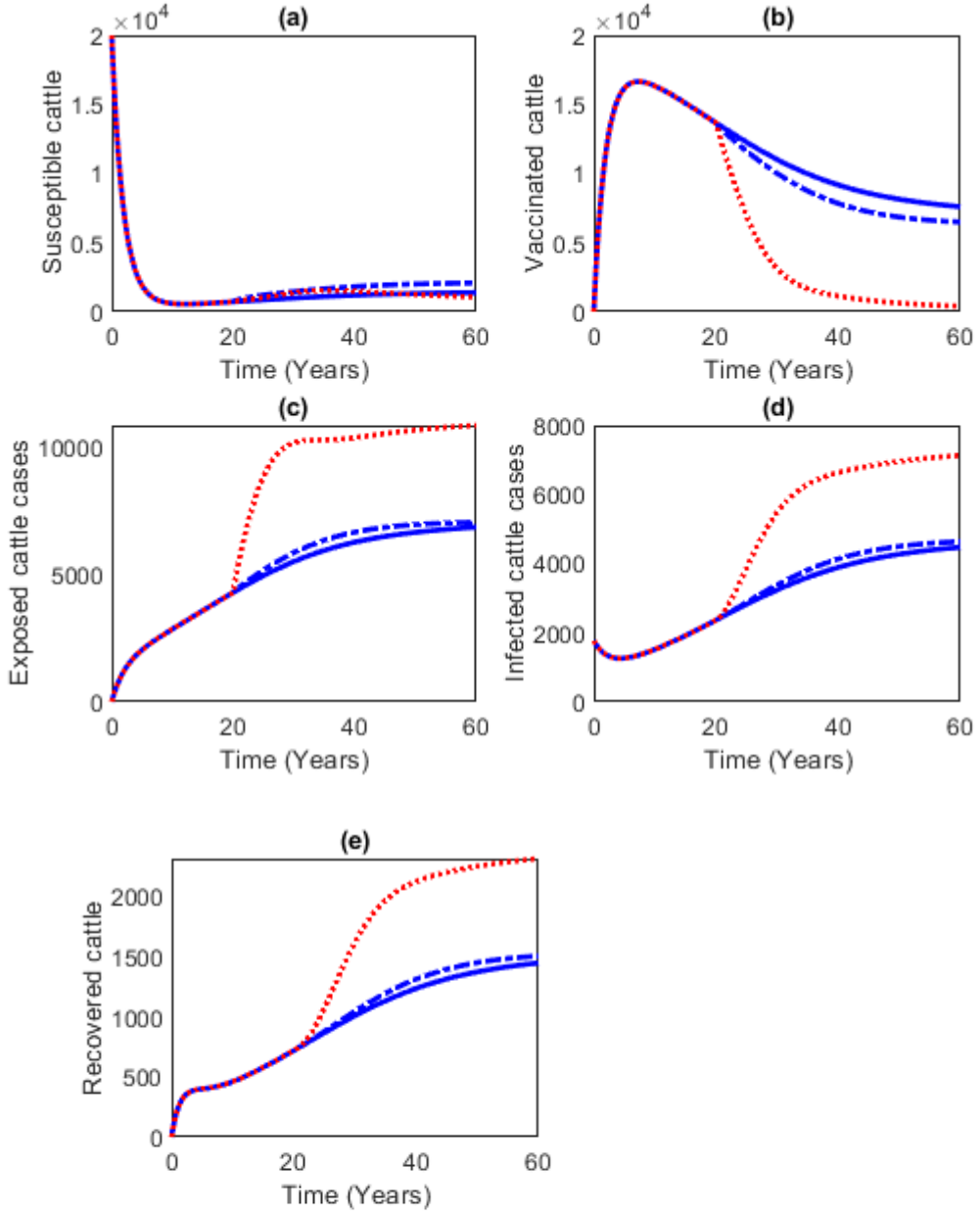

Figure 6.13: Given the cumulative $\omega = 0.480$ and $\beta_C = 6.000e-05$ (bold blue curve). The impact of vaccination rate on the transmission rate (FMD infection from buffaloes to susceptible cattle) on cattle population:
(i) $\omega = 0.35$, $\beta_C = 0.0000008$ when vaccination and transmission rate both decrease (dashed blue curve)
(ii)$\omega = 0.35$, $\beta_C = 0.04$ when vaccination rate decreases and transmission rate increases (dotted red curve). The rest of the parameters are fixed on table 6.1

Figure 6.13 simulations show that when both vaccination and transmission rates from infected buffaloes to susceptible cattle decrease, then exposed, infected and recovered cattle populations increase. In addition, when vaccination

rate decreases and transmission rate increases, then exposed, infected and recovered cattle populations increase. This suggests that the decrease of vaccination rate will put the cattle at risk of FMD infection.

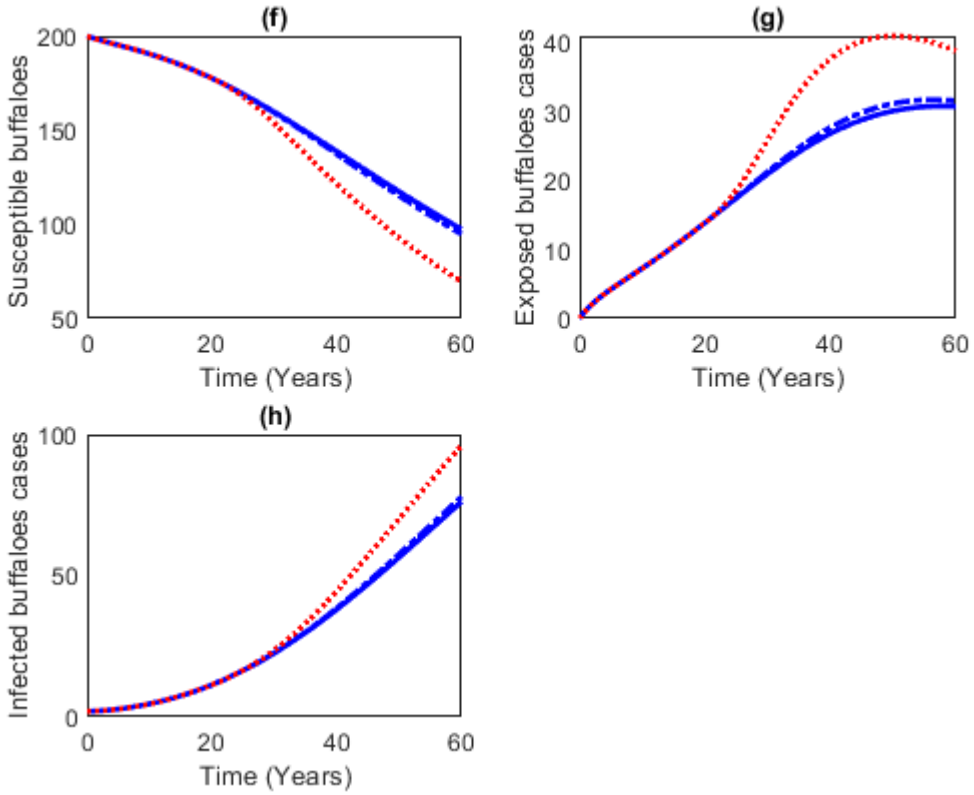

Figure 6.14: Given the cumulative $\omega = 0.480$ and $\beta_C = 6.000e-05$ (bold blue curve). The impact of vaccination rate on the transmission rate (FMD infection from buffaloes to susceptible cattle) on buffaloes population:
(i) $\omega = 0.35$, $\beta_C = 0.0000008$ when vaccination and transmission rate both decrease (dashed blue curve)
(ii)$\omega = 0.35$, $\beta_C = 0.04$ when vaccination rate decreases and transmission rate increases (dotted red curve).

Figure 6.14 simulations show that when both vaccination and transmission rates from infected buffaloes to susceptible cattle decrease, then the exposed and infected buffaloes populations increase slightly. In addition, when vaccination rate decreases and transmission rate increases, then susceptible buffaloes population decreases whilst exposed and infected buffaloes populations increase. This suggests that the decrease of vaccination rate on cattle will put the buffaloes at risk of FMD infection and transmitting it to the susceptible cattle.

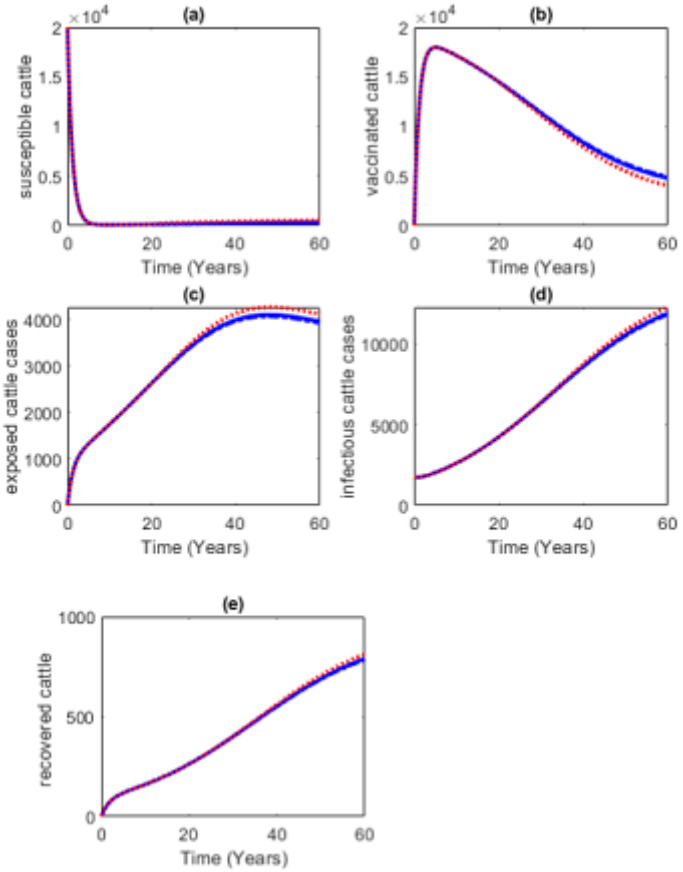

Figure 6.15: Given the cumulative $\omega = 0.480$ and $\beta_B = 1.1976e-04$ represent by bold blue curve. The impact of vaccination rate on the transmission rate (FMD infection from buffaloes to susceptible buffaloes) on cattle population demonstrated over a period of time:
(i) $\omega = 0.80$, $\beta_B = 0.000005$ as vaccination increases and transmission rate decreases, represent by dashed blue curve
(ii)$\omega = 0.35$, $\beta_B = 0.008$ as vaccination rate decreases and transmission rate increases, represented by the red dotted curve. The rest of the parameters are fixed on table 6.1

Figure 6.15 simulations show that when vaccination rate increases and the transmission rate decreases from infected cattle to susceptible buffaloes, then there is a slight decrease in exposed and infected buffaloes populations. However, when vaccination rate decreases and the transmission rate increases from infected cattle to susceptible buffaloes, then exposed and infectious buffaloes populations increase. Results demonstrated that if cattle are not protected then they can transmit FMD to susceptible buffaloes once they get into contact. Therefore, it is necessary to vaccinate more cattle in order to protect both cattle and buffaloes from FMD.

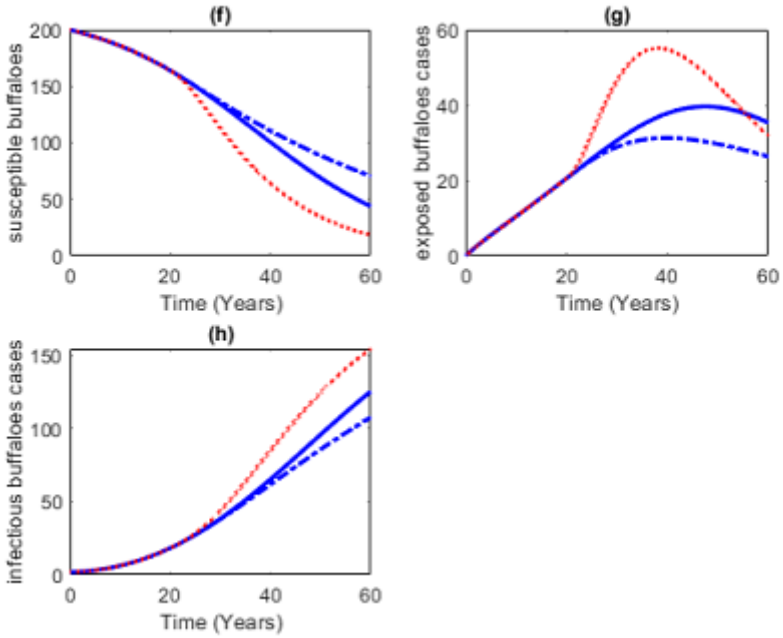

Figure 6.16: Given the cumulative $\omega = 0.480$ and $\beta_B = 1.1976e-04$ represent by bold blue curve. The impact of vaccination rate on the transmission rate (FMD infection from cattle to susceptible cattle) on buffaloes population demonstrated over a period of time:
(i) $\omega = 0.80$, $\beta_B = 0.000005$ as vaccination increases and transmission rate decreases, represent by dashed blue curve
(ii)$\omega = 0.35$, $\beta_B = 0.008$ as vaccination rate decreases and transmission rate increases, represented by the red dotted curve.

Figure 6.16 simulations show that when vaccination rate increases and the transmission rate decreases from infected cattle to susceptible cattle, then there is a slight increase in susceptible buffaloes population and a decrease in exposed and infected buffaloes populations. However, when vaccination rate decreases and the transmission rate increases from infected cattle to susceptible cattle, then exposed buffaloes and infected buffaloes populations increase whilst there is a slightly decrease in susceptible buffaloes. Results demonstrated that infected cattle seemed to have a great impact on transmitting FMD to susceptible buffaloes, therefore, increasing of vaccination rate is necessary in order to reduce the transmission of FMD from infected cattle to susceptible buffaloes population.

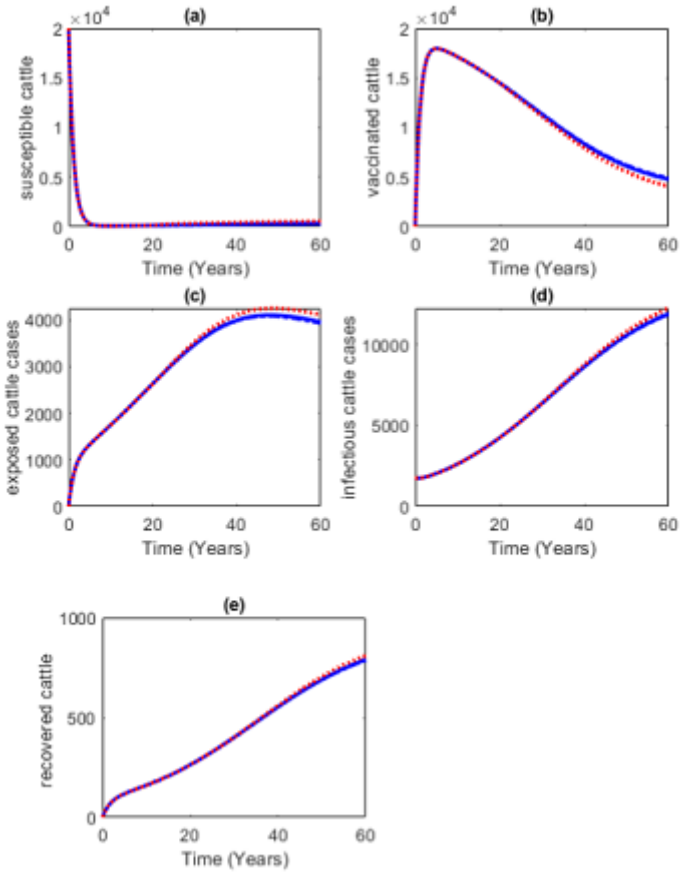

Figure 6.17: Given the cumulative $\omega = 0.8248$ and $\beta_B = 1.1976e-04$ (bold blue curve). The impact of vaccination rate on the transmission rate (FMD infection from cattle to susceptible cattle) on cattle population:
(i) $\omega = 0.80$, $\beta_B = 0.008$ when vaccination and transmission rate both increase (dashed blue curve)
(ii)$\omega = 0.35$, $\beta_B = 0.000005$ when vaccination rate and transmission rate both decrease (dotted red curve). The rest of the parameters are fixed on table 6.1

Figure 6.17 simulations show that when both vaccination and transmission rates from infected cattle to susceptible cattle increase, then exposed, infected and recovered cattle populations increase. In addition, when both vaccination and transmission rates decrease, then exposed, infected and recovered cattle populations decrease significantly. This suggests that the decrease of vaccination rate will put the cattle at risk of FMD infection.

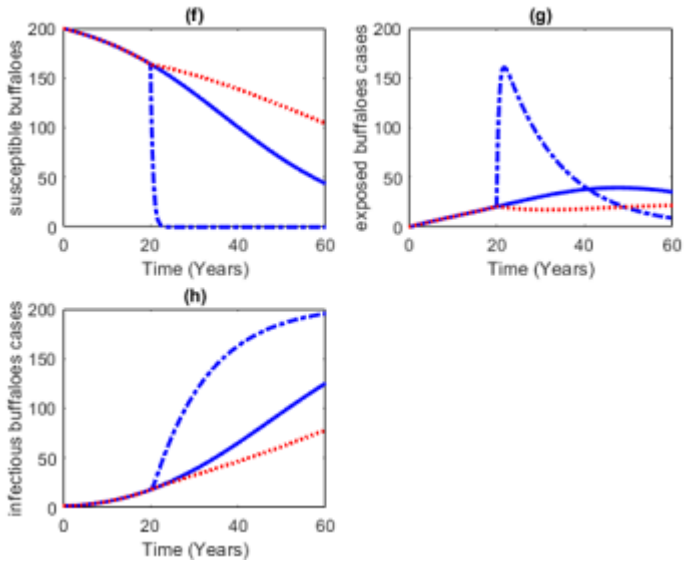

Figure 6.18: Given the cumulative $\omega = 0.480$ and $\beta_B = 1.1976e-04$ (bold blue curve). The impact of vaccination rate on the transmission rate (FMD infection from cattle to susceptible cattle) on cattle population:
(i) $\omega = 0.80$, $\beta_B = 0.008$ when vaccination and transmission rate both increase (dashed blue curve)
(ii)$\omega = 0.35$, $\beta_B = 0.00005$ when vaccination rate and transmission rat both decrease (dotted red curve). The rest of the parameters are fixed on table 6.1

Figure 6.18 simulations show that when both vaccination and transmission rates from infected cattle to susceptible cattle increase, then exposed and infected buffaloes population increase whilst susceptible buffaloes population decreases. However, when both vaccination and transmission rates decrease, then exposed and infected buffaloes slightly decrease whilst susceptible buffaloes increase. This suggests that the decrease of vaccination rate on cattle population will put the buffaloes at risk of FMD infection.

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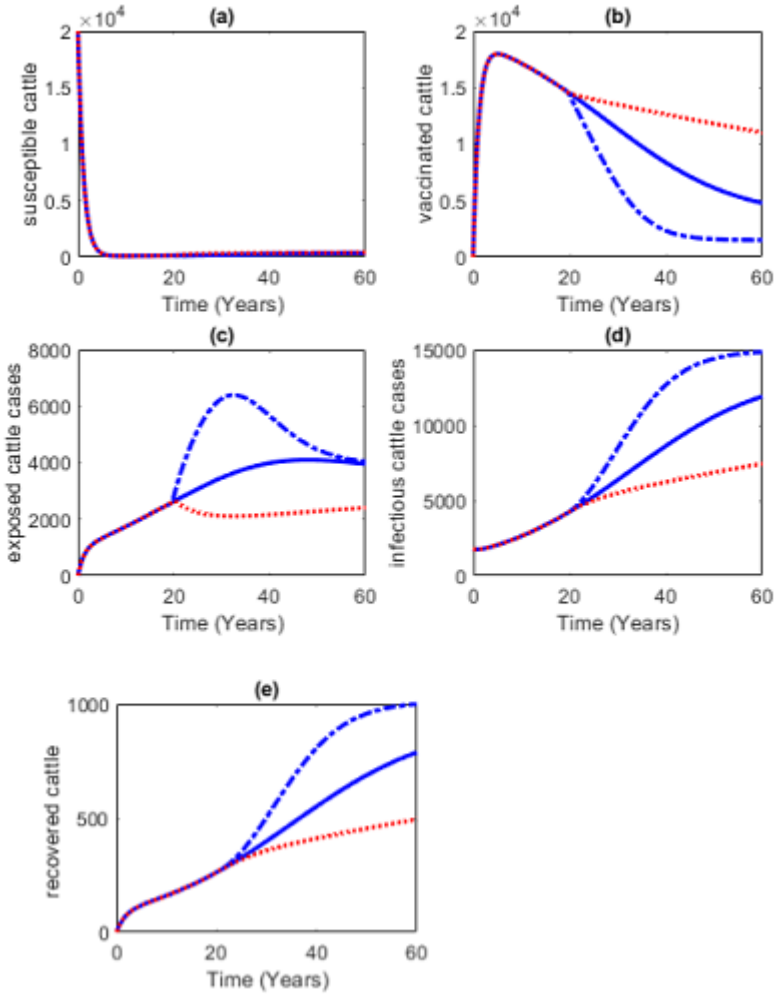

Figure 6.19: bold blue curve represent cumulative $\omega = 0.480$ and $\varepsilon = 0.5$. on cattle population. (i)Dashed blue curve $\omega = 0.80$, $\varepsilon = 0.4$ vaccination increases and vaccination efficacy decreases.
(ii) $\omega = 0.35$, $\varepsilon = 0.8$ vaccination rate decreases and efficacy increases, represented by the red dotted curve.

Figure 6.19 simulations show that when vaccination rate increases and the efficacy decreases, then there is a great increase in exposed, recovered and infected cattle populations. Moreover, when vaccination rate decreases and efficacy increases, then exposed, recovered and infected cattle populations decrease. This demonstrated that when vaccination efficacy increases, then the vaccination is very effective and cattle population will be protected.

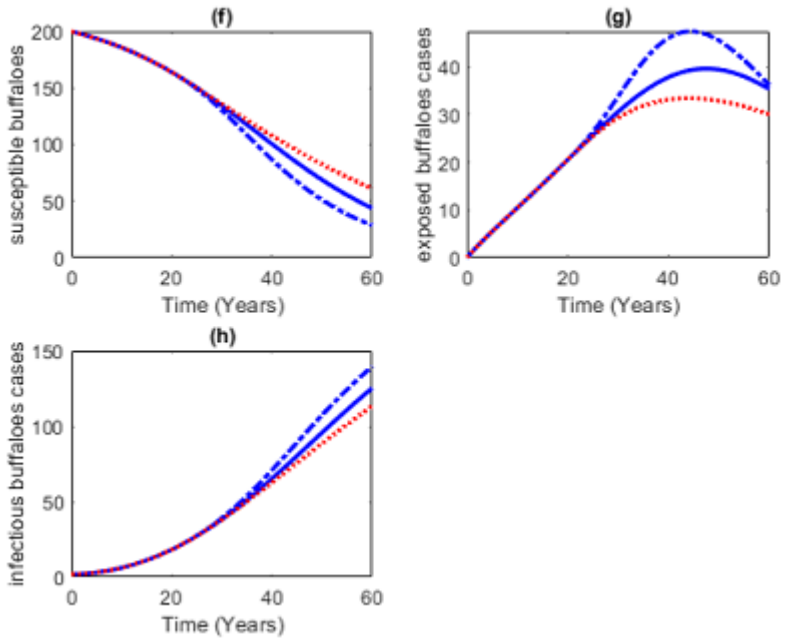

Figure 6.20: Bold blue curve represent cumulative $\omega = 0.480$ and $\varepsilon = 0.5$. on cattle population. (i)Dashed blue curve $\omega = 0.80$, $\varepsilon = 0.4$ vaccination increases and vaccination efficacy decreases.
(ii) $\omega = 0.35$, $\varepsilon = 0.75$ vaccination rate decreases and efficacy increases, represented by the red dotted curve.

Figure 6.20 simulations show that when vaccination rate increases and the efficacy decreases, then there is a great increase in exposed and infected buffaloes populations whilst susceptible buffaloes population decreases. Moreover, when vaccination rate decreases and efficacy increases, then exposed and infected buffaloes populations decrease whilst susceptible buffaloes population increases. This demonstrated that when vaccination efficacy increases, then the vaccination is very effective to protect both the cattle and buffaloes populations.

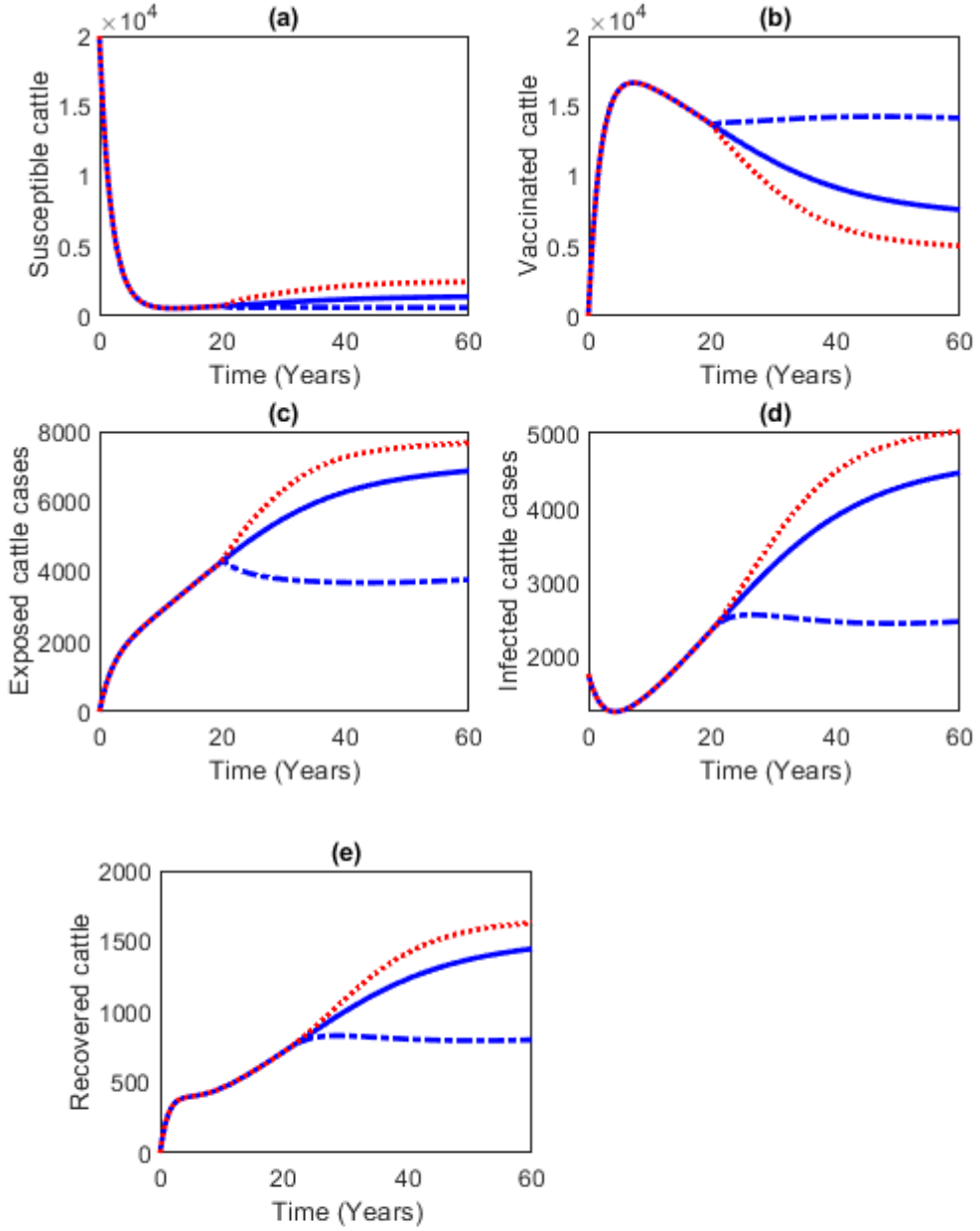

Figure 6.21: Bold blue curve represent cumulative $\omega = 0.480$ and $\varepsilon = 0.5$.
(i) $\omega = 0.98$, $\varepsilon = 0.8$ when vaccination and vaccination efficacy both increase (dashed blue curve). (ii)$\omega = 0.35$, $\varepsilon = 0.4$ when vaccination rate and efficacy both decrease (dotted red curve).

Figure 6.21 simulations show that when both vaccination rate and vaccination efficacy increase, then exposed, recovered and infected cattle populations greatly decrease. In addition, when vaccination rate and efficacy both decrease, then exposed, recovered and infected cattle populations increase. This proved that it is necessary for both vaccination rate and efficacy to be high in order to reduce FMD.

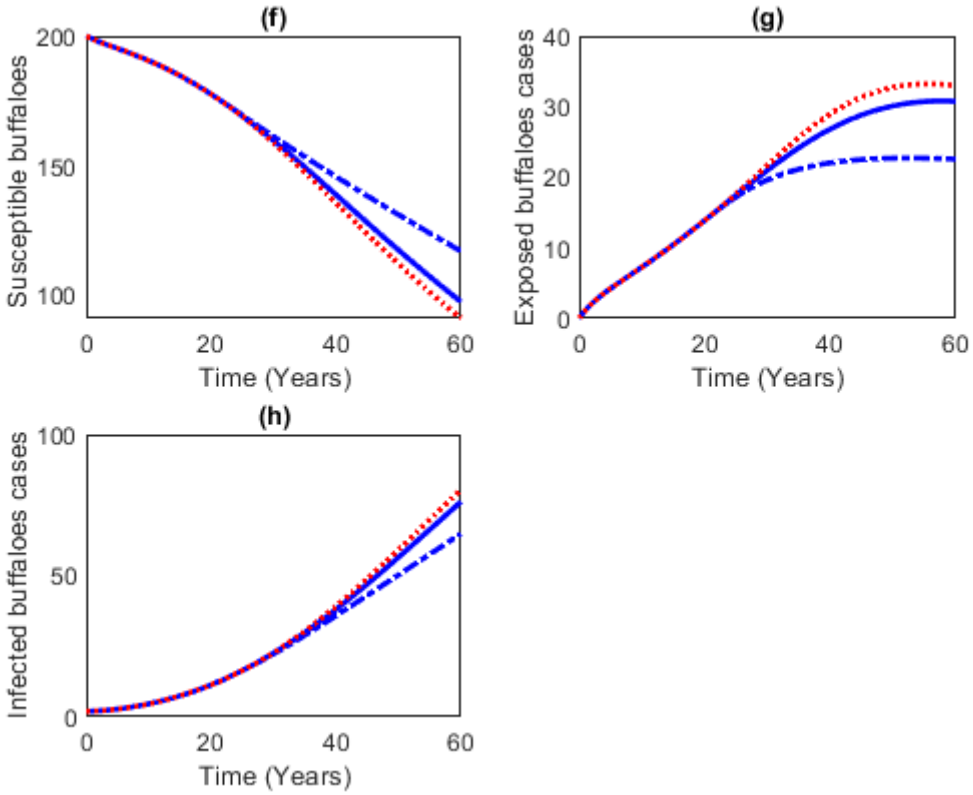

Figure 6.22: Bold blue curve represent cumulative $\omega = 0.480$ and $\varepsilon = 0.5$.
(i) $\omega = 0.98$, $\varepsilon = 0.80$ when vaccination and vaccination efficacy both increase (dashed blue curve). (ii)$\omega = 0.35$, $\varepsilon = 0.4$ when vaccination rate and efficacy both decrease (dotted red curve).

Figure 6.22 simulations show that when both vaccination rate and vaccination efficacy increase, then exposed and infected buffaloes populations decrease whilst susceptible buffaloes population increases. In addition, when vaccination rate and efficacy both decrease, then exposed and infected buffaloes populations increase. This proved that it is necessary for both vaccination rate and efficacy to be high in order to reduce FMD.

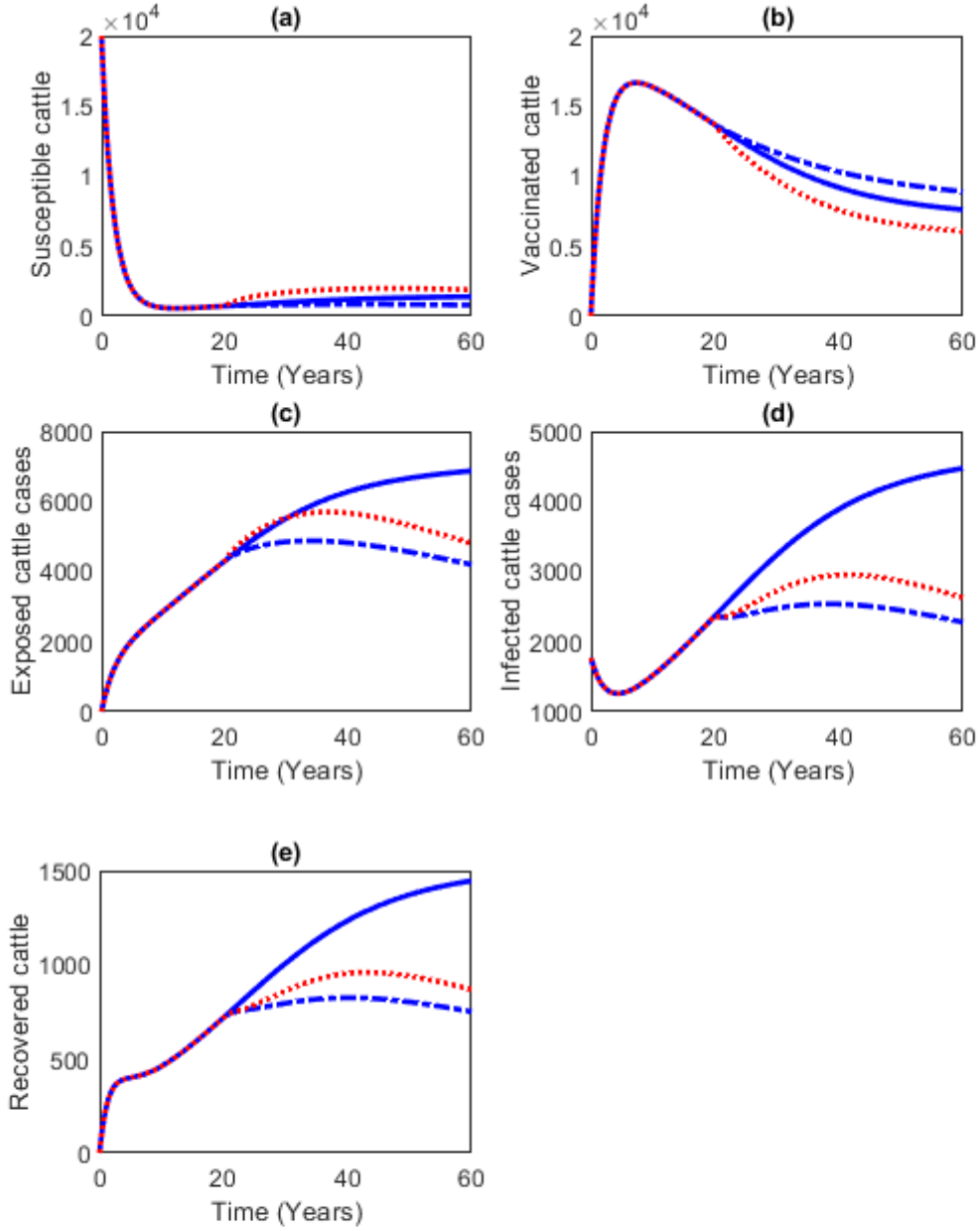

Figure 6.23: Bold blue curve represent cumulative $\omega = 0.480$ and $\theta = 0$. The impact of vaccination rate and culling on FMD when both implemented.
(i) Dashed blue curve $\omega = 0.80$, $\theta = 0.5$ when both vaccination rate and Culling increases.
(ii) $\omega = 0.35$, $\theta = 0.5$ vaccination rate decreases and culling increases, represented by the red dotted curve.

Figure 6.23 simulations show that when both vaccination and culling rates increase then exposed, recovered and infected cattle populations decrease. Moreover, when vaccination rate decreases and culling increases, then exposed, recovered and infected cattle populations increase slightly. Results demonstrated that culling rate has an impact on reducing FMD on cattle population but greatly if both vaccination and culling implemented.

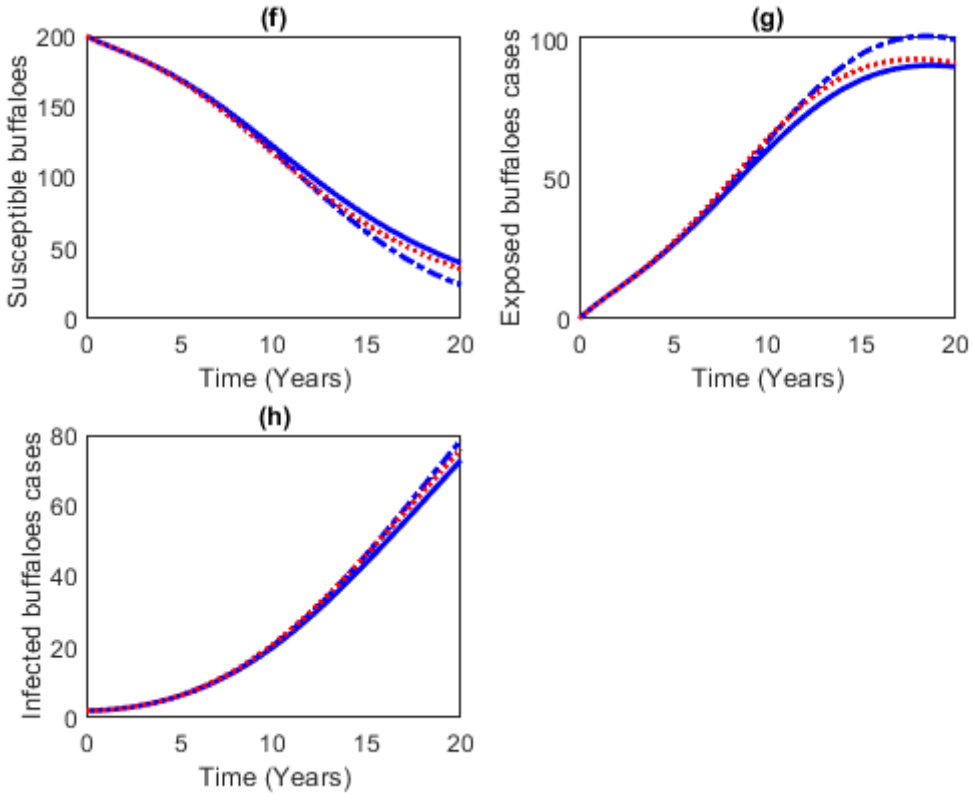

Figure 6.24: Bold blue curve represent cumulative $\omega = 0.480$ and $\theta = 0$. The impact of vaccination rate and culling on FMD when both implemented.
(i) Dashed blue curve $\omega = 0.80$, $\theta = 0.5$ when both vaccination rate and Culling increases.
(ii) $\omega = 0.35$, $\theta = 0.5$ vaccination rate decreases and culling increases, represented by the red dotted curve.

Figure 6.24 simulations show that when both vaccination and culling rates increase then exposed and infected buffaloes populations decrease but not much compared to cattle population. Results demonstrated that culling has an impact on reducing FMD on cattle population but not in buffaloes population. Therefore, it is advisable to practice culling more on cattle population to reduce FMD since culling of buffaloes is not practiced.

Chapter 7

Discussion of results, conclusions and recommendations

7.1 Discussion

We studied the transmission dynamics of FMD in cattle and buffaloes by presenting two mathematical models for foot and mouth disease with and without vaccination and culling. Mathematical analysis was carried out on two models and appropriate theories were used to establish fundamental results on stability of equilibrium points as well as compute important thresholds. All state of variables were proved to be non-negative and bounded in the positively invariant feasible region. The models had two forces of infection. One showed the rate at which infected (cattle and buffaloes) infected the susceptible cattle. The other force of infection showed the rate at which susceptible buffaloes get infected by the infected cattle and infected buffaloes.

The existence and stability of the equilibrium points depending on the values of $\mathcal{R}_0$ were carried out. Two types of equilibria were established; namely the disease free equilibrium and the endemic equilibrium associated with the infection from infected cattle and infected buffaloes. It was established that DFE was locally and asymptotically stable whenever $\mathcal{R}_0 < 1$ and endemic equilibrium is locally and asymptotically stable whenever $\mathcal{R}_0 > 1$. Applying standard mathematical techniques we established global stability of the disease free equilibrium.

Mathematical analysis revealed the effects of the control reproduction numbers $\mathcal{R}_e$ which is an effective reproduction number for vaccination model and $\mathcal{R}_0$ for reproduction number for basic model. The disease free equilibrium was locally asymptotically stable when $\mathcal{R}_0 < 1$ it means DFE for FMD is stable. This tells us that FMD can be

eliminated from the population while when $\mathcal{R}_0 > 1$ DFE is unstable and it implies that FMD infection continues in cattle and buffaloes population and cannot be fully eliminated from the population. The disease will spread when there is an outbreak. In this case, $\mathcal{R}_0 > \mathcal{R}_e$.

Numerical simulations were conducted on predictions of theoretical results. Through numerical analysis we observed that applying vaccination without culling shows the decrease in susceptible cattle and an increase in the rest of the classes but a slight difference in buffaloes population. This shows that vaccination may have a great impact on reducing cumulative new FMD infection cases all the time. Furthermore, we observed that high vaccination rate with high vaccination efficacy has a great impact when it comes to reducing FMD infection. Therefore, this shows that vaccination is very effective and is protecting the cattle population. We also observed that by increasing vaccination on cattle population, it has a slight benefit on buffaloes population although not much of the difference compare to the cattle population. Therefore, an intervention which ensures higher rate might be crucial in order to control FMD. However, since there is not much protection given to the buffaloes, this shows that vaccination is more effective in protecting cattle more than buffaloes. Since buffaloes are not well protected and they are still interacting with cattle, in the long run the benefits of vaccination might wane out because the buffaloes are not protected. On the other hand, even if the vaccination is effective, infected buffaloes still increase in numbers. One could argue that this is because buffaloes are not protected even though cattle are well protected so new cases will still arise.

Another promising finding was that after vaccinating the cattle and still new cases arose, then this means that there is still a route of infection that is not blocked which is the infection from buffaloes to cattle population. Moreover, when it comes to vaccines, some vaccines protect the animals from falling sick but not from getting infected but they can still infect other animals. From the results, we have seen that vaccination is not sufficient to protect both the cattle and buffaloes. This means extra measures are needed to prevent buffaloes from spreading the infection. Our findings are similar to those reported by Mushayabasa and Tapedzesa [1], Bouma [42] also Orsel and kneeling et al. [10] but our study revealed the consequences on the buffaloes and the potential threat that the buffaloes may pose to the control effects population.

Our study also focused on the potential effects of introducing and increasing culling into the cattle population; a control measure that Namibia is not currently using. We showed that when culling of cattle is practiced and increased, the FMD cases decrease over time. Even though from the available data Namibia never practiced culling in the cattle population, our results suggested that culling has a positive impact on controlling or reducing FMD among the cattle population. This control measure has a consequence of reducing exposed and infected buffaloes populations since the number of infected cattle available to interact with buffaloes has reduced due to the culling

of infected cattle. Better results were observed when both vaccination and culling were used simultaneously. Mushayabasa, Bhunu and Dhlamini [36], concluded that vaccination and culling reduce the disease dynamics fast when both are implemented and when culling is taking place on the infected farms after detection. Our study has found similar results with focus on all the state variables of cattle and buffaloes on the population on interface setting, Mushayabasa, Bhunu and Dhlamini only focused on impact on exposed and infection classes of cattle while our study expanded to all the seven classes.

We investigated the effects of vaccination and the FMD transmission rates. High vaccination rate and low transmission rate from infected cattle to susceptible cattle was the best strategy that reduced the FMD burden, followed by high vaccination and low transmission rate from infected buffaloes to susceptible buffaloes. We observed that low vaccination rate and high transmission rate especially from infected cattle to susceptible cattle and infected buffaloes to susceptible buffaloes was a great threat when it comes to FMD. Furthermore, low vaccination rate and low transmission rate was the least strategy to protect cattle and buffaloes from FMD. Low vaccination and high transmission rate was the worst strategy for protecting cattle and buffaloes from FMD since in this strategy the flow of cattle or buffaloes is high into the unprotected route of infection compared to the flow into the vaccination route where infection is low. Although, our study is similar to others such as [36, 44], our study has touched on the concept of vaccination efficacy and different routes of FMD transmission that others did not look at.

7.2 Conclusions

We designed and analysed a mathematical deterministic model FMD dynamics. Numerical simulations of the model were performed using the Matlab ODE45. The effect of vaccination and culling were performed on the cumulative exposed, infection and recovery on both cattle and buffaloes. We observed that vaccination and culling have a positive impact on eradicating FMD in cattle and buffaloes populations. The results on the numerical simulations suggest that culling used together with vaccination produce better results in reducing the FMD cases in both cattle and buffaloes compared to vaccination alone.

7.3 Recommendations

The results from our study revealed several benefits for the current vaccination programs in Namibia and also exposed the dangers of relaxing the current strategies. From our results we recommend the following:

i. Vaccination rate to be increased with corresponding increase in vaccination efficacy.

ii. Vaccination program to be used optimally together with culling.

iii. It will be a good idea for the authorities to look for ways to protect the buffaloes from interacting with cattle otherwise all the benefits of vaccination will wane out. Since it is not practical to vaccinate the buffaloes because there is no vaccine meant to protect the buffaloes in Namibia, one aspect that could be considered is to put up measures on the borders areas. The authority may consider electric fencing as a barrier for animals crossing over.

iv. Cattle along the park boundaries can be put in paddocks to avoid contact with buffaloes.

7.4 Weakness and future work

The findings in this work have important implications and several limitations, which should be acknowledged. We did not distinguish our model by age of animals or risk (i.e high or low risk) for the significant risk of FMD transmission. Hence, for effective control of FMD during an outbreak, these two controls should be maximised for the entire period. The models presented in this thesis were able to capture and predict scenarios on FMD based on the set of assumptions some of which are restrictive. We have assumed that infections can be transmitted through contact between an infectious and a susceptible animal, although airborne foot-and-mouth disease virus transmission has been documented. We did not investigate an optimal strategy that can be put in place when culling and vaccination are used together. Associated with the optimal strategy, cost benefits in analysis could be incorporated. Investigation is required on the mode of transmission to determine which is more dangerous; whether it is the infection that comes from buffaloes or infection that starts from cattle or the combination of these two forms of transmissions. All these can be used to improve the outcomes of this thesis and we shall explore some of these in our future work.

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Appendix

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ETHICAL CLEARANCE CERTIFICATE

Ethical Clearance Reference Number: FOS /418/2018 **Date: 1 October, 2018**

This Ethical Clearance Certificate is issued by the University of Namibia Research Ethics Committee (UREC) in accordance with the University of Namibia's Research Ethics Policy and Guidelines. Ethical approval is given in respect of undertakings contained in the Research Project outlined below. This Certificate is issued on the recommendations of the ethical evaluation done by the Faculty/Centre/Campus Research & Publications Committee sitting with the Postgraduate Studies Committee.

Title of Project: Developing Mathematical Models On The Dynamics Of Foot And Mouth Disease (Fmd) In Cattle And Buffalo Using Vaccination And Culling: A Namibian Perspective

Researcher: EVALTHINE SHIKUMWIFA

Student number: 200941135

Supervisor(s): Dr E. Koga (Main) University Of Namibia (UNAM); Dr D. Iiyambo, University Of Namibia (UNAM)Dr F. Chirove, University Of Kwazulu-Natal (UKZN)(Co)

Faculty: Faculty of Science

Take note of the following:

(a) Any significant changes in the conditions or undertakings outlined in the approved Proposal must be communicated to the UREC. An application to make amendments may be necessary.
(b) Any breaches of ethical undertakings or practices that have an impact on ethical conduct of the research must be reported to the UREC.
(c) The Principal Researcher must report issues of ethical compliance to the UREC (through the Chairperson of the Faculty/Centre/Campus Research & Publications Committee) at the end of the Project or as may be requested by UREC.
(d) The UREC retains the right to:
(i) Withdraw or amend this Ethical Clearance if any unethical practices (as outlined in the Research Ethics Policy) have been detected or suspected,
(ii) Request for an ethical compliance report at any point during the course of the research.

UREC wishes you the best in your research.

Dr. J.E. de Villiers: UREC Chairperson

Ms. P. Claassen: UREC Secretary