



Bachelor of Veterinary Medicine Research Project 2

Title	Wildlife Defence: A Field Investigation into the conservative control of Wildebeest-associated Malignant Catarrhal Fever (A1HV-1) on a dairy farm in Kenya
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Abstract

Malignant catarrhal fever (MCF) causes a fatal lymphoproliferative disease in cattle, as well as other ungulates. In Africa, the disease is caused by ruminant γ -herpesviruses alcelaphine herpesvirus (A1HV-1), with susceptible wildebeest (*Connochaetes spp.*) as the asymptomatic reservoir host. Although a concerted effort to produce vaccines has been made and multiple vaccine trials have been undertaken, this has so far been unsuccessful (Haig et al. 2008, Russell et al. 2012, Palmeira et al. 2013, Parameswaran et al. 2014). No prophylaxis and no cure mean that control of transmission is essential. This study analyses data from a dairy farm in Kenya with cases of MCF, using rate ratios to investigate disease patterns within the herd and analyse risk factors for disease among cattle to advise the potential effectiveness of control measures. During the 2 year study period, a total of 359 cattle were present on the farm. The overall incidence of MCF was 6.1%, with twenty-two cases of MCF occurring during the study period. In this study, there was no statistical evidence that a particular gender or breed were more susceptible to MCF. Analysis of age provided statistical evidence that animals less than six months old were at increased risk of being a case of MCF compared to other age categories. Finally, analysis of the efficiency of partition fencing erected during the study at reducing MCF, showed no statistical evidence. However, further analysis of using fencing as a control measure is required.

List of Abbreviations

A1HV-1	Alcelaphine Herpesvirus 1
BVD	Bovine Viral Diarrhoea
CBPP	Contagious Bovine Pleural Pneumonia
ECF	East Coast Fever
KWS	Kenya Wildlife Service
MCF	Malignant Catarrhal Fever
OvHV-2	Ovine Herpesvirus 2
WA-MCF	Wildebeest-Associated MCF

1. Introduction

Malignant Catarrhal Fever (MCF) is a disease seen in cattle and other ungulates, including bison and deer, caused by ruminant γ -herpesvirus (Pfitzer *et al.* 2013). The two documented forms of MCF are γ -herpesviruses, alcelaphine herpesvirus 1 (A1HV-1) and ovine herpesvirus 2 (OvHV-2). This paper relates to the A1HV-1 form, which is a particular problem in the South and East of Africa, as well as in some zoos and private wildlife collections internationally (Matzat *et al.* 2015).

MCF is a member of the subfamily *Gammaherpesviridae*, genus *Rhadinovirus*. The genetics have been sequenced and well-studied (Ensser *et al.* 1997, Lankester *et al.* 2015b). The disease is difficult to distinguish clinically from rinderpest and other viral diseases, such as bovine viral diarrhoea (BVD) (Mirangi and Kang'ee 1999). For a definitive diagnosis, postmortem histopathological analysis of tissue samples is recommended (OIE 2013). Multiple ELISA tests have been developed to detect MCF antibodies but this is often inappropriate, as it has been proven that most cattle die before a detectable antibody response is mounted (Wambua *et al.* 2015). PCR-based assays to detect A1HV-1 DNA are the most favoured method of diagnosis, using peripheral blood leukocytes or tissue samples of clinical cases (Li *et al.* 2011).

Transmission of the A1HV-1 virus to cattle occurs from susceptible wildebeest (*Connochaetes spp.*), which are considered an asymptomatic reservoir host. Infected cattle are non-contagious to other cattle. A1HV-1 in a cell-free form has been isolated from wildebeest nasal and lacrimal secretions, as well as blood (Mushi *et al.* 1980a). Transmission of wildebeest-associated MCF (WA-MCF) is thought to be through direct contact and aerosol, between wildebeest and also from wildebeest to cattle. High levels of shedding have been recorded from wildebeest calves less than one year of age (Mushi and Rurangirwa 1981). Indirect transmission has also been observed, although little evidence has been presented (Barnard *et al.* 1989).

In cattle MCF presents as an acute, lymphoproliferative disease. Although recovery from MCF has been reported (Milne and Reid 1990, Penny 1998), MCF has a low morbidity rate (1-3%) and an exceptionally high mortality rate of 90-100% (Swai *et al.* 2013). Clinical signs are caused by acute proliferation of T cells with extensive vasculitis and tissue necrosis due to dysregulated cytotoxic lymphocytes (Schock and Reid 1996). Cattle typically die within 4-7 days after the onset of clinical signs (Plowright *et al.* 1972). In a recent study in which animals were infected either intranasally or intramuscularly with A1HV-1 in experimental conditions, incubation periods ranged between 21 and 68 days (Haig *et al.* 2008).

There is currently no available treatment for MCF. Available control measures include movement of cattle from shared pasture, driving away wildebeest and the construction of boundaries. There is little evidence to

suggest that any of these methods are effective. Conjecture from field experience suggests a vaccine would be the only truly effective control measure for MCF. A concerted effort to produce vaccines has been made and multiple vaccine trials have been undertaken (Haig *et al.* 2008, Russell *et al.* 2012, Palmeira *et al.* 2013, Parameswaran *et al.* 2014). So far these trials have failed to provide evidence of adequate protection of cattle.

MCF has been shown to have profound consequences on pastoralists and farmers in the developing world (Bedelian *et al.* 2007, Lankester *et al.* 2015a). A study conducted in Tanzania identified and ranked MCF as the disease of most concern to pastoralist farmers over more common diseases such as East Coast Fever (ECF) and Contagious Bovine Pleural Pneumonia (CBPP). The threat of the disease forces pastoralists to move cattle to less productive grazing areas to avoid grazing with wildebeest. Losses of cattle due to MCF in areas of co-grazing may reach 7% annually (Reid and Van Vuuren 2004). One study found estimated sale prices per infected animal to be reduced by 50% (Bedelian *et al.* 2007). The impact of WA-MCF on small-holder and large-scale farms has not been described.

Unlike MCF associated with sheep, WA-MCF has been largely neglected in research perhaps due to its geographic location and unknown impact. The objectives of this study are to give a detailed description of the WA-MCF problem within a large dairy herd in Kenya, establish any disease patterns within the herd and analyse risk factors for disease among cattle to advise the potential effectiveness of control measures.

2. Materials and methods

2.1 Farm background

The study farm was situated on the eastern edge of Lake Naivasha located to the west of Nairobi, in the Great Rift Valley of Kenya (Figure 1). Its altitude was approximately 1,892m above sea level. The farm had a total area of 162 hectares, of which an estimated 20% was used for the dairy farm livestock. Crop fields take up a further 20%. The rest was used for residential properties and other businesses unrelated to farming, including commercial horseback riding, dining, boat rides and a campsite. The farm was also a main access point to other properties, both residential and commercial.



Figure 1: Depiction of Kenya with an estimated geographical marker of the area of the study farm. (Unknown 2013)

The study farm employed 73 permanent staff in total. Of these, 31 had direct contact with the livestock and these all lived in on-site accommodation. Horses were stabled within the premises and some co-grazing with the dairy cows occurred. No sheep or livestock other than cattle were kept on the premises. There was a clear perimeter boundary, made up from dense vegetation or fencing, although several sections had been breached sporadically over time. An electrified fence secured areas used for keeping livestock and crops, preventing access to wild herbivores, including wildebeest, which freely-roamed the majority of the property. The fence had been constructed in stages from January to May 2014. Cattle were moved frequently through non-fenced areas and there were no specific biosecurity measures in place. Income was generated through contracted milk purchase and the sale of calves.

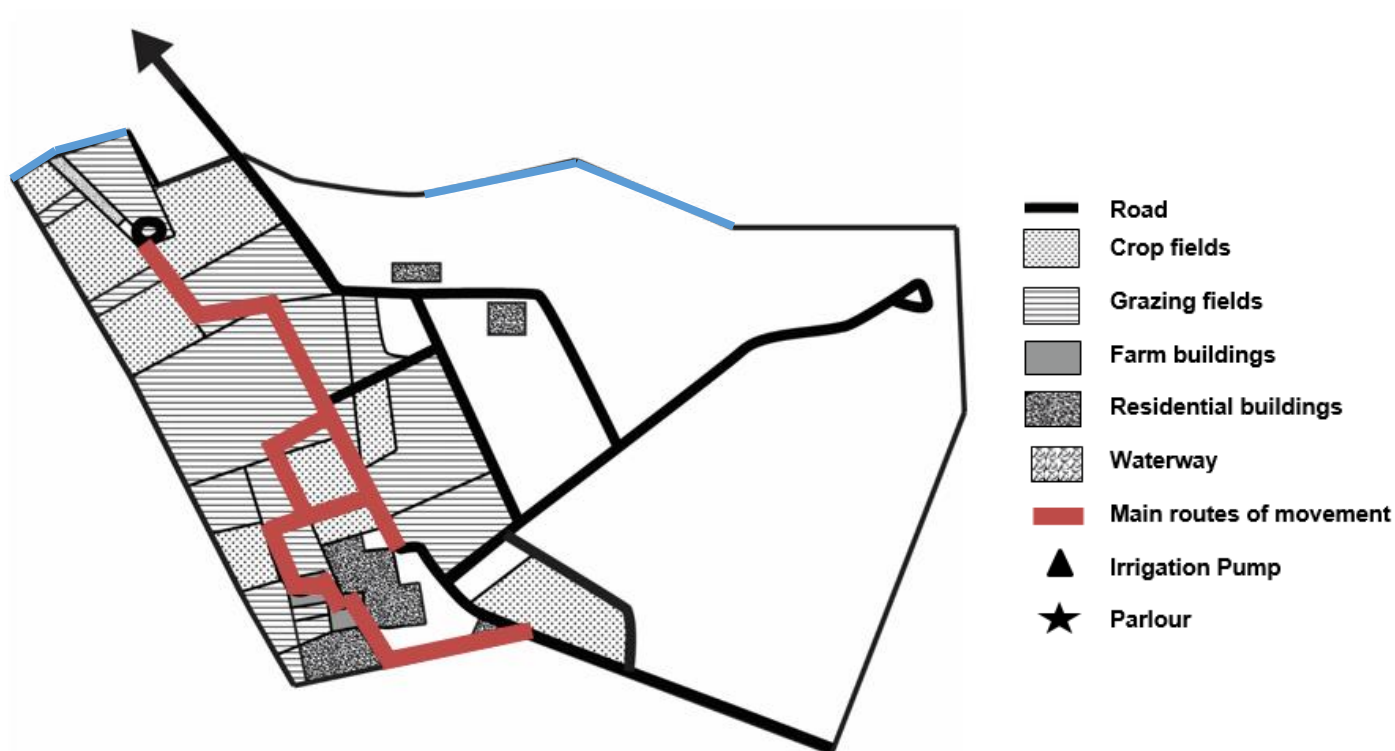
Wildebeest were first introduced into the area in the 1980s. Ten animals were relocated onto the nearby Crescent Island for the shooting of a film in 1984. It was assumed that the lack of predators and readily available resources, along with the high reproductive efficiency associated with wildebeest, led to the large numbers that now move freely around the conservancy. Calving of the wildebeest had been reported to occur mainly from January to March on the study farm.

2.2 Study Population

The number of cattle present on the farm at the time the data was collected was approximately 240. During this period, the population included 61 heifers and 88 lactating cows. Heifers were defined as more than six months of age and having not yet calved. Cattle were managed in 4 groupings, based on age, pregnancy and lactation status. Each group had designated paddocks for grazing (Figure 2); no paddocks were shared between groups. Limited grazing during the dry season meant that paddocks were rotated every 2-7 days.

Movement around the farm, for milking and paddock changes, was on within-farm roads and through paddocks. There was also an isolated maternity paddock, where pregnant cows were kept from a few days before their due date until seven days post-calving, when the calf was removed from the dam. Groups of adult cows were kept outside and were under supervision 24 hours a day by a herdsman for the purpose of security and monitoring health. Herdsmen were designated the same cattle group throughout. Calves were kept close to the milking parlour and farm buildings to allow for close supervision by workers, and separated into two neighbouring paddocks depending on weaning status. Pre-weaning, calves were housed in hutches, holding 3-5 animals, overnight. Weaning occurred at 12 weeks of age.

Figure 2: Overall map of farm with key of significant areas. The red route marks the main path of movement for both cattle and farm vehicles. The farm had both residential and commercial areas. Lake Naivasha directly communicates with the boundaries marked in blue.



Calving occurred all year round and all breeding was through artificial insemination (AI) utilising government-subsidised semen. There were no bulls present on the farm and male calves were sold on to other farmers by six months of age. The breeds were European crossbreeds (Holstein-Friesian, Ayrshire and Brown Swiss) and breed status had been recorded yearly until May 2010 by the Kenyan Livestock Breeders Association. In this case, breed status refers to what percentage of the animals' genetics are pedigree, using a blood sample to determine genotype. Cattle were categorised into four groups according to pedigree status; foundation, intermediate, appendix and purebred. Of the 128 classified cattle that remained within the herd

approximately 20% were foundation, 44% intermediate, 36% appendix, with only one cow classed as purebred. Cows were individually identified with ear tags marked with a unique number.

For most of the year, cows were hand-milked twice daily in a fixed parlour. When grazing conditions were at their peak, a group of 32 "elite" cows (defined by producing >16 litres/day) were milked three times daily. Milk not suitable for collection because the cow had a disease such as mastitis and/or was receiving medication such as antibiotics, was fed to the calves. During milking, cows were fed a mixed ration of dairy meal, sunflower seeds and alfalfa from a common feed trough. The "elite" cows received further nutrition once daily in the form of crushed maize, which was grown and processed on site. Dry cows and heifers were given maize stalks twice daily to subsidise the poor grazing available during the dry season.

The cattle over three months old were dipped weekly with Tratix® to protect against tick borne diseases, specifically ECF, Babesiosis and Anaplasmosis, which are prominent in the area. Cattle were also vaccinated against Foot and Mouth disease every four months. For disposal, carcasses were put into large purpose-dug pits on property with soda lime.

2.3 Data collection

Most data pertaining to the study was collected using paper records obtained from the farm site during a visit approved by the Kenyan Department of Veterinary Services. Data collection occurred between 9th and 20th of March 2015. A manual approach was taken to consolidate these records and extract the required information. Types of data collected included individual animal details (age, breed, gender and reason for leaving herd), milk records and rainfall figures. Digital records had also been kept through the study period, although there were fluctuations in what was recorded and how regularly. Again a manual search and extraction technique was used for these records. A database was then compiled using Microsoft Excel.

A map of the farm was created by identifying boundaries and generating polygons using Google Earth® 7.1 in consultation with the farm manager and property owner. This was then imported into Photoshop CC® and graphically modified, before converting to a JPEG image.

A census was conducted annually until 2014 by Kenya Wildlife Service (KWS), Hell's Gate National Park, of all wildlife species within Naivasha. Each landowner received an overview of the collected data, which was divided into animals counted per property. A copy was obtained with permission of the land owner for use in this study.

Rainfall measurements were collected via a standard rain gauge, which was calibrated so that rain can be measured in millilitres. Readings were taken daily and recorded manually on paper records.

Data on temperature was collected using Weather Underground® (www.wunderground.com), an online resource which uses BestForecast™ software, collecting data from 6,000 automated weather stations operating internationally. The data utilised in this study was a monthly average of the average maximum temperatures of each day.

MCF cases were defined by farm staff based on the observed criteria of: inappetence & weight loss, black scour, corneal opacity, nasal and lacrimal discharge, and buccal ulceration (Figure 3 & 4). This classification is in line with the case definitions by Kalunda *et al.* (1981), Barnard *et al.* (1994) and others. Other criteria used included rapid progression of signs, a reduced milk yield and being unresponsive to broad spectrum antibiotics. It should be noted that not all criteria were fulfilled by every case and no minimum was specified.



Figure 3: Dairy cow showing ocular opacity and mucopurulent nasal discharge. (Image curtesy of Sanctuary Farm, Kenya)



Figure 4: Dairy cow showing severe, acute weight loss and black scours. (Image curtesy of Sanctuary Farm, Kenya)

2.4 Data Analysis

A historical cohort study approach was utilised to produce a descriptive analysis using time-to-event data. This approach was used to make use of all available data from the study period and allowed for censoring to incorporate all animals at risk during the study period, including those that did not develop disease. Two different time scales were used including age and calendar time. The study period was defined as 1st March 2013 to the 28th February 2015. Statistical software, Stata® 13.1, was used for the analysis. Kaplan-Meier curves were created and Mantel-Haenzel rate ratios were generated to quantify the associations between putative risk factors and the primary outcome (i.e. being a case of MCF). To examine age as a risk factor, Lexis expansion by age categories was applied first to obtain age-at-risk bands. Then Poisson Regression was used to analyse rate ratios, examining the association between each age category and the baseline.

One bull calf was excluded from the analysis due to a lack of records. The date of birth for seven bull calves had been lost from the records. For this analysis these animals were assigned estimated dates of birth; as each calf was numbered chronologically, the time between the last and next recorded birth was split to give an estimated birth date. Some animals had lost their original tags and been retagged with a new number. In these cases efforts were made by the farmer to cross-reference records although some potential inaccuracies were acknowledged.

3. Results

3.1 Demographic data

During the study period, a total of 359 cattle were present on the farm. Two hundred and nine animals were born and 53 animals died or were culled. No animals entered the herd except through birth. One hundred and twenty-nine animals exited for reasons other than death. Most cows were female (73.8%). Due to the complexities of the livestock's genetics, breeds were classified using their majority purebred status where available. If this had not been recorded, an observational definition was made using breed standard characteristics where possible. Some subjects were unable to be classified as they died before an observational definition could be made by the author and had no existing record of breed. Friesian was the most common breed classification of all cows (57.4%). The overall incidence of MCF was 6.1%, with twenty-two cases of MCF occurring during the study period. This information is summarised in Table 1. The KWS census estimated that in 2014 the total number of wild herbivores was 497, including 211 wildebeest (mean 1.3 wildebeest per hectare).

Table 1: Descriptive data of study demography and the representation of MCF cases seen with column percentages.

Variable	Category	Number	Percentage (%)	MCF cases	MCF (%)
Gender	Female	265	73.8	19	86.4
	Male	94	26.2	3	13.6
Breed	Ayrshire	45	12.5	5	22.7
	Friesian	206	57.4	14	63.6
	Brown Swiss	2	0.6	0	0.0
	Unknown	106	29.5	3	13.7

3.2 Risk Analysis

To assess primary risk factors for becoming a case of MCF, univariable associations between these factors and the disease were analysed. Of the data collected, it was possible to analyse the effects of sex, breed, and age. Univariable analysis of sex via the Mantel-Haenszel method produced a rate ratio of 1.5 when comparing risk of females having MCF to males although there was no statistical evidence to support and association (Table 2). There were only two Brown Swiss cows in this study. These were therefore excluded from the breed analysis as numbers were insufficient to produce interpretable results. Animals with unknown breed status could not be meaningfully classified in the analysis, so were also excluded. A rate ratio comparing Friesian with Ayrshire cattle gave no evidence that Friesian cattle were at increased risk, (Table 2).

Table 2: Mantel-Haenszel rate ratios showing risk between categories of the variables of gender and breed. For gender, the rate ratio compares females to males. For breed, the rate ratio compares Friesian to Ayrshire.

Variable	Category	Number	Rate Ratio	P Value (95% CI)
Gender	Female	265	1.5	0.52 (0.44, 5.0)
	Male	94		
Breed	Friesian	206	1.5	0.42 (0.55, 4.2)
	Ayrshire	45		

Results on age analysis provided statistical evidence that animals less than six months of age were at increased risk of being a case of MCF compared to other age categories by having risk ratios less than one and low P-values (all less than 0.1) (Table 3). Further analysis of this risk factor was demonstrated using a

Kaplan-Meier survival curve (Figure 5), which showed the percentage of cumulative survival, specifically animals not probable to have MCF, decreasing with age.

Table 3: Rate ratios for age categories compared to a baseline (<6 months of age).

Age Group (months)	Number	Rate Ratios	P Value (95% CI)
0 to ≤ 6	-	Baseline	-
6 to ≤ 12	144	0.27	0.091 (0.058, 1.2)
12 to ≤ 36	147	0.27	0.005 (0.023, 0.51)
36 to ≤ 60	87	0.27	0.050 (0.073, 1.0)
60 to ≤ 84	68	0.11	0.033 (0.013, 0.83)
> 84	67	0.40	0.097 (0.13, 1.2)

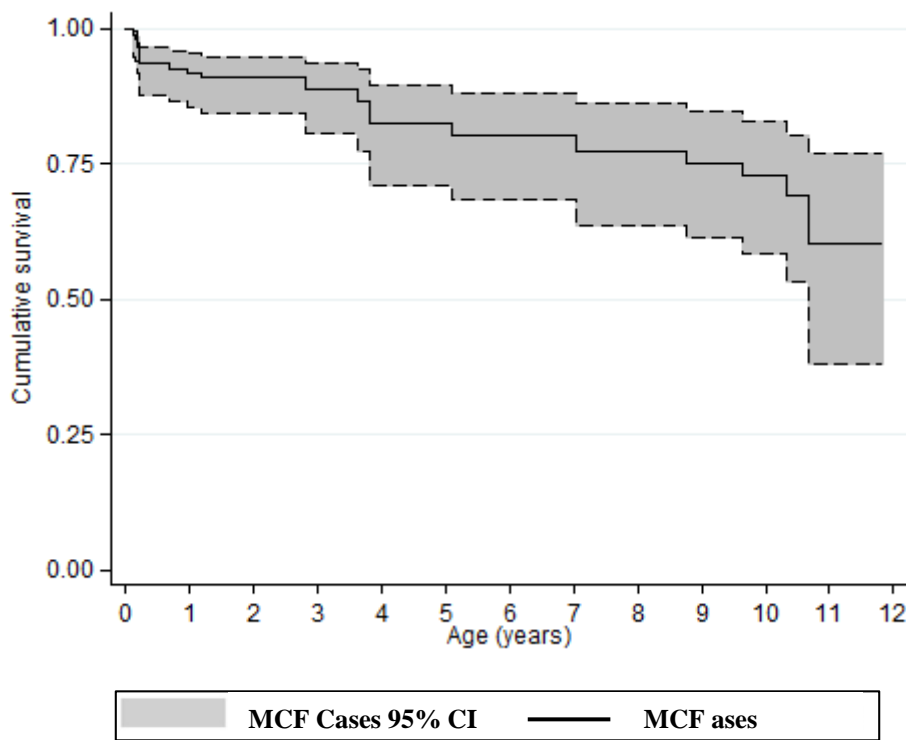


Figure 5: Unadjusted Kaplan-Meier survival curves for MCF cases in relation to age of animal. All animals were included in the analysis if present on the farm during the study period. MCF cases were defined as death from disease. The Y-axis represents the cumulative probability of death from MCF.

In the final univariable analysis, the significance of the erection of the boundary fencing was analysed. Lexis expansion was applied based on the time the fence was erected, using the midpoint between the start and end of construction. The vertical line on the Kaplan-Meier survival graph highlights this point (Figure 6). The rate ratio of 0.77 shows the rate of MCF decreased after the fence was erected but the P-value was 0.56 (95% CI, 0.32-1.84), suggesting no statistical evidence of an effect. A trend of MCF cases were seen at

similar times of the year both before and after the fence. Figure 7 depicts data collected on rainfall and temperature during the study period. There was no observable relationship between these factors and recorded MCF cases. Fluctuations in either direction of either rainfall or temperature did not consistently fit with an increased rate of MCF.

Figure 6: Unadjusted Kaplan-Meier survival curves for MCF cases in relation to calendar month. All animals were included in the analysis if present on the farm during the study period. MCF cases were defined as death from disease. The Y-axis represents the cumulative probability of death from MCF. The vertical line indicates the midpoint of the fence construction.

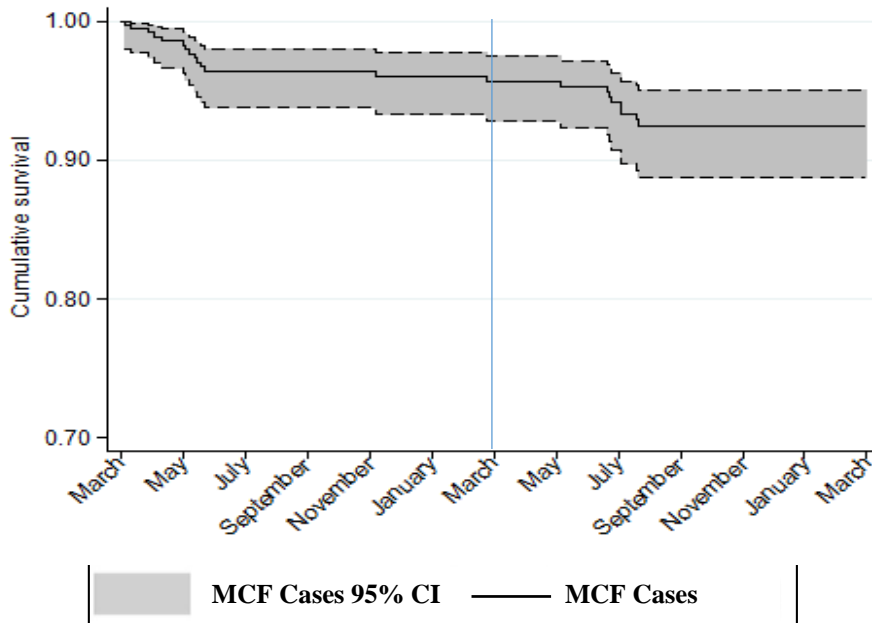
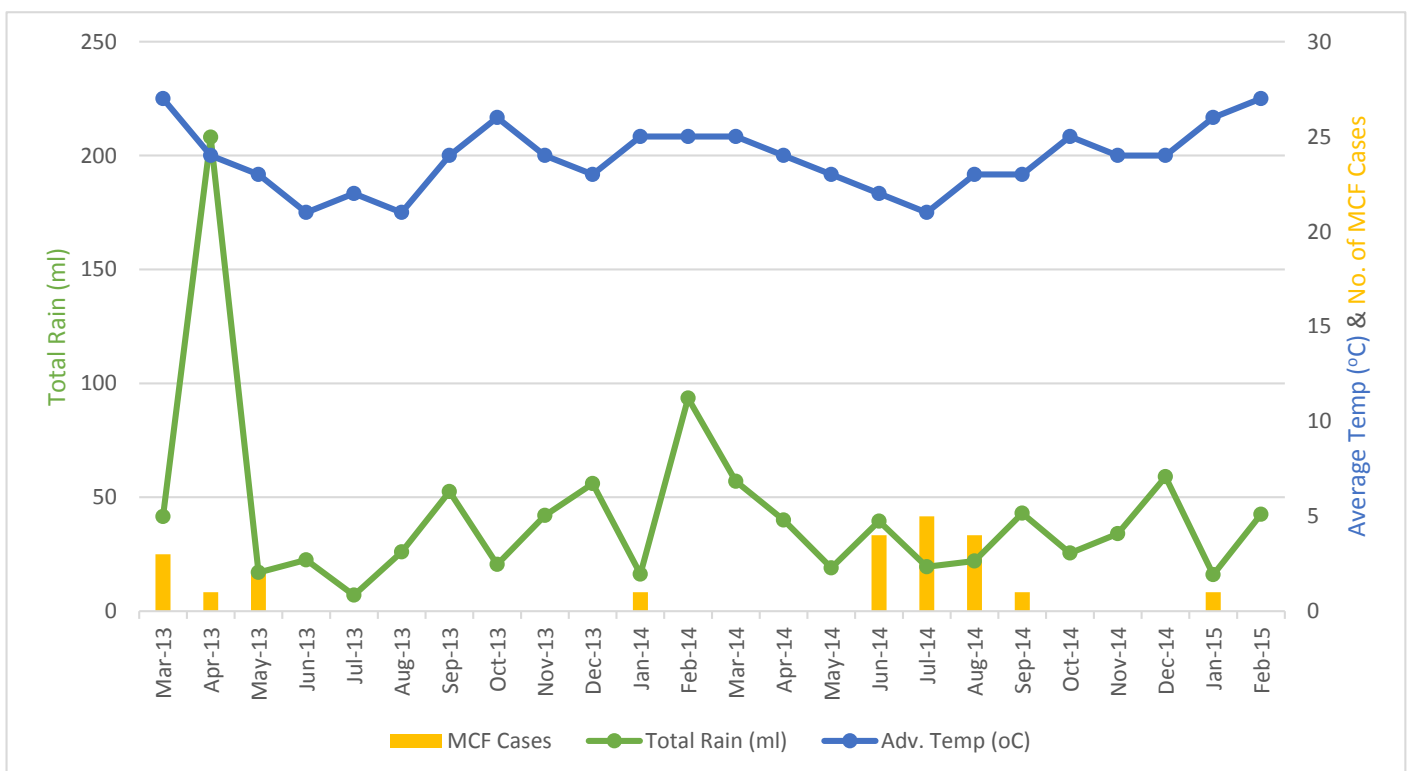


Figure 7: Graph depicting amount of rain and average daily temperature for each month of the study period. MCF cases per month have also been plotted to allow for visual analysis of correlation.



4. Discussion

During this study, a range of potential factors that could contribute to the problem of MCF have been highlighted. Bedelian *et al.* (2007) suggested that wildebeest are being forced to calve around a permanent water source instead of migrating to their traditional wet-season calving ground, due to human development. It is likely that in time more farms will be in the same position as the study farm, with permanent co-grazing with wildebeest, making this study relevant.

Currently, the main method used to prevent transmission is avoidance. One study found 90% of pastoralists move the majority of cattle away from home to avoid MCF (Lankester *et al.* 2015a). This is obviously not feasible for fixed farms. Another method is to chase off wildebeest from pasture. Not only is this ineffective, with the majority of animals returning within 24 hours, but may actually increase the likelihood of transmission to cattle as stress may increase the level of virus shed from wildebeest calves (Mushi *et al.* 1980b). On the study farm, cattle are grazed at close proximity to wildebeest calving but they do not co-graze. This means they do not come into direct contact with wildebeest foetal membranes, once thought to be a source of MCF virus. However, this theory has become less favourable since a study by Rossiter *et al.* (1983), not only because of the rapid inactivation of the virus through sunlight but also because observations showed membranes were scavenged rapidly (within an hour) reducing the risk significantly.

Transmission through fomites, such as commercial vehicles, horses, crops and by foot, could be possible on the study farm. Horses co-graze with both cattle and wildebeest on the farm and no studies have been produced to look at this as a possible risk. However, the theory that low-level persistent infection of the environment occurs, e.g. on pasture from foetal membranes that have now been scavenged, has been shown unlikely (Rossiter *et al.* 1983). The feed storage shed is not secured within the fence but 24 hour watchmen have never reported sighting wildebeest near it and no feed has been lost, suggesting this is not a route of contamination. AI is not screened for MCF virus but evidence from other studies suggests transmission between cattle doesn't occur.

In this study, there was no statistical evidence that a particular gender or breed were more susceptible to MCF. The rate ratio result (1.5) suggests that females are at more risk than males of contracting MCF. However, considering the disproportionate number of females to males in the study population, the strength of evidence is weak. This is consistent with other previous studies (Kalunda *et al.* 1981).

Results in this study gave statistical evidence that age is a significant risk factor. A previous study found 1-2 year old cows to be affected more than other age groups (Swai et al. 2013). Another study in South Africa found a higher incidence in adult cattle in particular periparturient females (Barnard et al. 1994). These are in contrast to the results of this study, with animals less than six months of age being associated with increased risk when compared to all other age groups. It is apparent from all these studies together that there is no consistent association between age of animal and risk of MCF. The risk may be associated with how animals are managed which may increase the risk of exposure to AIHV-1. On the study farm calves up to six months of age were not housed within the fence but had no apparent contact with the wildebeest. Calves, with their reduced immunity, may be more susceptible to lower levels of infection, perhaps via aerosol or mucosal secretions within the water or brought in on fomites.

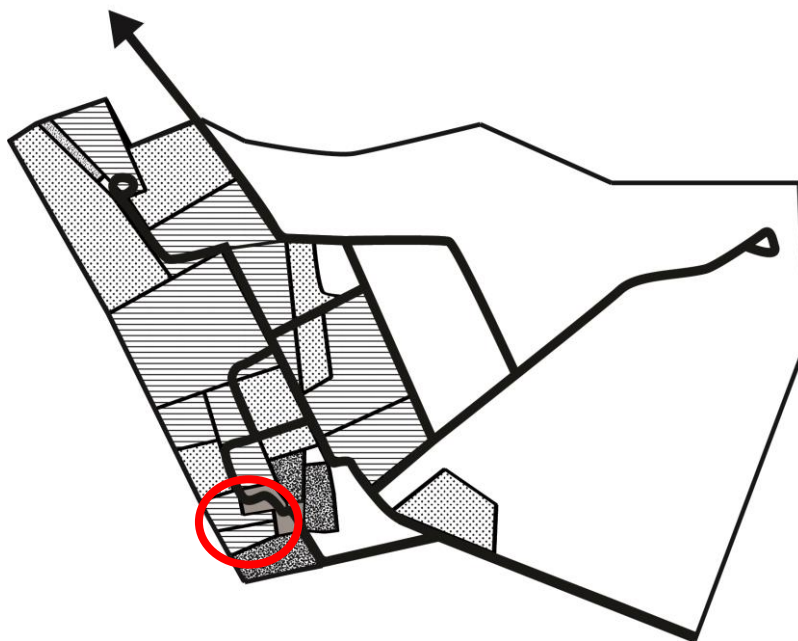


Figure 8: Overall map of farm with the location of calves marked

As seen on Figure 6, there is a trend of increased incidence from late spring to early summer. These results fit with previous reports of times of year with increased incidence (Barnard and Van de Pypekamp 1988). Season has long been considered a risk factor for MCF, with previous studies reporting that outbreaks coincide with the migratory period of wildebeest (Swai *et al.* 2013). Conflicting this view are reports of sporadic outbreaks (Penny 1998, Swai *et al.* 2013). As wildebeest were of permanent residence here, it is unlikely that migration had an impact on the study farm. Rainfall may be an important factor, separate to seasonality. Another study suggests that incidence of MCF was highly dependent on number of wildebeest calving in an area, which in turn depended on availability of grass and therefore rainfall (Bedelian *et al.* 2007). Temperature may also be a factor for consideration, as MCF is readily inactivated by strong sunlight. When exposed directly, 99.6% of the virus is lost within 25 minutes at midday (Rossiter *et al.* 1983). In this

study, the results showed no apparent relationship between increased rainfall or decreased temperature and increased incidence of MCF. Further statistical analysis would be required to fully evaluate this association but relatively low numbers of clinical cases may limit the statistical power to detect an association.

Fencing was constructed in response to several years of cattle losses due to MCF (Figure 8). Fencing has been recognised as a control measure but has not always been possible due to finance or location (Bedelian et al. 2007). The results of this study showed no statistical evidence to support that the fence impacted the rate of MCF on the farm although again low numbers of clinical cases may limit the statistical power. Its apparent ineffectiveness could be explained by the timing of its construction. As discussed previously, there appears to be an annual pattern for MCF incidence. Some sections of fencing were not completed until May. Which areas were fenced last, and therefore cattle protected last, were unknown. The incubation period of MCF is thought to be ≤ 68 days, which means that cattle could have become infected prior to the fence erection. A longer follow-up may be needed to establish the fence's true effectiveness and the economic benefits.



Figure 8: A section of fence constructed to separate wildebeest from cattle. The fence consisted of 3 layers. The main structure consisted of 5ft posts, approx. 10ft apart, joined by four lengths of barbed wire. Bamboo poles were then strung vertically to give the fence height, which could be easily seen by wildlife. An electric fence ran around the outside of the main structure, approximately one metre from it. The electric fence consisted of four wires strung between 3ft high wooden posts. Finally, there was a fence running internal to the main structure, this time five metres out to prevent cattle-wildebeest contact. This internal fence was 3ft high, with a single electrified wire and wooden posts – this wire was moved depending on the grazing fields in use.

Potential errors in the study may have arisen due to missing records, some estimation of animal parameters and low statistical power due to the number of cases seen during the study period. A change of farm managers during the study period led to confusion over some data with some records being lost and, potentially, some cases not being recorded. Unknown breed classifications meant that breed as a risk factor could not be fully evaluated. Clinical signs used to categorise each individual MCF case were not recorded, and no laboratory testing was performed on suspected diseased animals, meaning some animals may have been misclassified. However, as collectively the clinical signs are pathognomonic for MCF and animals are vaccinated against the other common differential diagnoses, there is evidence that the reliability of the clinical diagnosis is strong.

5. Conclusion

The incidence of MCF in Kenya is difficult to estimate because few cases are reported to the authorities and it is not a notifiable disease. However, the literature suggests that it is a disease of significant economic and environmental importance in some farming systems. This study is the first known to describe a MCF problem on a large scale dairy farm with known exposure to wildebeest and provided evidence that breed and gender did not increase the risk of contracting MCF. Age was found to be a factor of significance, with calves less than 6 months being more at risk from MCF than any other age. With treatment still unavailable, the focus should be on implementing stringent control of cattle-wildebeest interaction. A study into the cost-benefit analysis of constructing a fence would be beneficial. In the interim, a potential option suggested by previous studies is starting and finishing daily grazing 1-2 hours later than is currently practised (Rossiter et al. 1983, Bedelian et al. 2007). This is to allow time for destruction of any virus on the pasture through sunlight exposure. Other areas that require investigation are the importance of biosecurity, the role (if any) of equine species in MCF transmission and the development of an effective vaccine for farms that have a significant problem with WA-MCF.

Acknowledgments

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References

- Barnard, B. J. and Van de Pypekamp, H. E. (1988) 'Wildebceest-derived malignant catarrhal fever: unusual epidemiology in South Africa', *Onderstepoort J Vet Res*, 55(1), 69-71.
- Barnard, B. J., van de Pypekamp, H. E. and Griessel, M. D. (1989) 'Epizootology of wildebeest-derived malignant catarrhal fever in an outbreak in the north-western Transvaal: indications of an intermediate host', *Onderstepoort J Vet Res*, 56(2), 135-9.
- Barnard, B. J., Van der Lugt, J. J. and Mushi, E. Z. (1994) 'Malignant Catarrhal Fever. J. A. Coetzer, G. R. Thomson, and R. C. Tustin (eds.)', *Oxford University Press, Cape Town, Infectious diseases of livestock with special reference to Southern Africa*, 946-957.
- Bedelian, C., Nkedianye, D. and Herrero, M. (2007) 'Maasai perception of the impact and incidence of malignant catarrhal fever (MCF) in southern Kenya', *Prev Vet Med*, 78(3-4), 296-316.
- Ensser, A., Pflanz, R. and Fleckenstein, B. (1997) 'Primary structure of the alcelaphine herpesvirus 1 genome', *J Virol*, 71(9), 6517-25.
- Haig, D. M., Grant, D., Deane, D., Campbell, I., Thomson, J., Jepson, C., Buxton, D. and Russell, G. C. (2008) 'An immunisation strategy for the protection of cattle against alcelaphine herpesvirus-1-induced malignant catarrhal fever', *Vaccine*, 26(35), 4461-8.
- Kalunda, M., Dardiri, A. H. and Lee, K. M. (1981) 'Malignant catarrhal fever. I. Response of American cattle to malignant catarrhal virus isolated in Kenya', *Can J Comp Med*, 45(1), 70-6.
- Lankester, F., Lugelo, A., Kazwala, R., Keyyu, J., Cleaveland, S. and Yoder, J. (2015a) 'The economic impact of malignant catarrhal fever on pastoralist livelihoods', *PLoS One*, 10(1), e0116059.
- Lankester, F., Lugelo, A., Mnyambwa, N., Ndabigaye, A., Keyyu, J., Kazwala, R., Grant, D. M., Relf, V., Haig, D. M., Cleaveland, S. and Russell, G. C. (2015b) 'Alcelaphine Herpesvirus-1 (Malignant Catarrhal Fever Virus) in Wildebeest Placenta: Genetic Variation of ORF50 and A9.5 Alleles', *PLoS One*, 10(5), e0124121.
- Li, H., Cunha, C. W. and Taus, N. S. (2011) 'Malignant catarrhal fever: understanding molecular diagnostics in context of epidemiology', *Int J Mol Sci*, 12(10), 6881-93.
- Matzat, T., Eulenberger, K. and Muller, H. (2015) '[Investigation of the presence of the etiological agents of malignant catarrhal fever in clinically healthy ruminants in zoological gardens]', *Berl Munch Tierarztl Wochenschr*, 128(5-6), 218-24.
- Mirangi, P. K. and Kang'ee, F. M. (1999) 'Diagnosis of malignant catarrhal fever using the polymerase chain reaction', *Vet Rec*, 145(19), 558-9.
- Mushi, E. Z., Karstad, L. and Jessett, D. M. (1980a) 'Isolation of bovine malignant catarrhal fever virus from ocular and nasal secretions of wildebeest calves', *Res Vet Sci*, 29(2), 168-71.

- Mushi, E. Z., Rossiter, P. B., Karstad, L. and Jessett, D. M. (1980b) 'The demonstration of cell-free malignant catarrhal fever herpesvirus in wildebeest nasal secretions', *J Hyg (Lond)*, 85(2), 175-9.
- Mushi, E. Z. and Rurangirwa, F. R. (1981) 'Epidemiology of bovine malignant catarrhal fevers, a review', *Vet Res Commun*, 5(2), 127-42.
- OIE (2013) 'World Organisation for Animal Health. MALIGNANT CATARRHAL FEVER.', *OIE Terrestrial Manual*, 2.4.15.
- Palmeira, L., Sorel, O., Van Campe, W., Boudry, C., Roels, S., Myster, F., Reschner, A., Coulie, P. G., Kerkhofs, P., Vanderplasschen, A. and Dewals, B. G. (2013) 'An essential role for gamma-herpesvirus latency-associated nuclear antigen homolog in an acute lymphoproliferative disease of cattle', *Proc Natl Acad Sci U S A*, 110(21), E1933-42.
- Parameswaran, N., Russell, G. C., Bartley, K., Grant, D. M., Deane, D., Todd, H., Dagleish, M. P. and Haig, D. M. (2014) 'The effect of the TLR9 ligand CpG-oligodeoxynucleotide on the protective immune response to alcelaphine herpesvirus-1-mediated malignant catarrhal fever in cattle', *Vet Res*, 45, 59.
- Penny, C. (1998) 'Recovery of cattle from malignant catarrhal fever', *Veterinary Record*, 142(9), 227-227.
- Pfizer, S., Last, R., Espie, I. and van Vuuren, M. (2013) 'Malignant Catarrhal Fever: An Emerging Disease in the African Buffalo (*Syncerus caffer*)', *Transbound Emerg Dis*.
- Plowright, W., Kalunda, M., Jessett, D. M. and Herniman, K. A. (1972) 'Congenital infection of cattle with the herpesvirus causing malignant catarrhal fever', *Res Vet Sci*, 13(1), 37-45.
- Reid, H. W. and Van Vuuren, M. (2004) 'Bovine malignant catarrhal fever. In: Coetzer J A W and Tustin R C. (eds)', *Oxford University Press, Cape Town, Second edition(Infectious Diseases of Livestock)*.
- Russell, G. C., Benavides, J., Grant, D., Todd, H., Deane, D., Percival, A., Thomson, J., Connelly, M. and Haig, D. M. (2012) 'Duration of protective immunity and antibody responses in cattle immunised against alcelaphine herpesvirus-1-induced malignant catarrhal fever', *Vet Res*, 43, 51.
- Swai, E. S., Kapaga, A. M., Sudi, F., Loomu, P. M. and Joshua, G. (2013) 'Malignant catarrhal fever in pastoral Maasai herds caused by wildebeest associated alcelaphine herpesvirus-1: An outbreak report', *Vet Res Forum*, 4(2), 133-6.
- Unknown (2013) *Lake Naivasha*, 300x264, www.wild-eye.co.za.
- Wambua, L., Wambua, P. N., Ramogo, A. M., Mijele, D. and Otiende, M. Y. (2015) 'Wildebeest-associated malignant catarrhal fever: perspectives for integrated control of a lymphoproliferative disease of cattle in sub-Saharan Africa', *Arch Virol*.