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PROVINCIALISING FOOT-AND-MOUTH DISEASE: THE CONSTRUCTION OF THE AFRICAN BUFFALO AS DISEASE RESERVOIR IN BOTSWANA, 1961–1976

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ABSTRACT

Global foot-and-mouth disease (FMD) control policy is largely based on nineteenth-century British ideas about the disease. This partiality risks creating the impression that these ideas of the disease are universal. In this article, therefore, we provincialise the notion of FMD by describing how veterinarians came to know the disease in colonial and postcolonial Botswana. Specifically, we focus on the 1960s and 1970s to write a postcolonial, more-than-human history that details how the African buffalo was identified as the reservoir host for the disease. We point to the local interactions between veterinarians and the buffalo, but also describe the 'external' pressure that veterinarians felt to find a culprit. As such, we present a geographically nuanced and historically informed understanding of FMD that challenges the assumed universality of British ideas about FMD, and exposes the ways in which these ideas shaped and continue to shape environmental and socio-economic realities in Southern Africa.

KEYWORDS: foot-and-mouth disease, disease reservoir, veterinary medicine, colonial science, Botswana

INTRODUCTION

> FMD [foot-and-mouth disease] in southern Africa is both virologically and epidemiologically a very different disease from that which exists elsewhere in the world, with the exception of East Africa.
>
> —Ian Scoones et al., 'Foot-and-mouth disease and market access'.[1]

Foot-and-mouth disease (FMD) is an infectious disease caused by a virus that affects cloven-hoofed livestock in most parts of the world. The World Organisation for Animal Health (WOAH, formerly the Office International des Epizooties, OIE) ranks it as one of the most economically important transboundary animal diseases and, together with the UN Food and Agriculture Organization (FAO), has targeted it as the next disease to be eradicated globally.[2] From a Western perspective, this target may make sense. Since the late nineteenth century, Britain and later the rest of Europe and North America have pursued and succeeded in eradicating endemic FMD. Only sporadically does the disease reappear through the import of infected livestock and meat, in response to which all infected livestock – and those to which the disease has potentially spread – are culled (such as in the devastating 2001 FMD outbreak in Britain). There thus seems to be a precedent and imperative to extend these eradication efforts.

However, the target of global eradication makes less sense when we adopt a Southern African perspective. Here, eradication is practically impossible, given that the FMD virus circulates not just in livestock but also in wildlife – specifically in the African buffalo (*Syncerus caffer*).[3] Scientists have found that buffalo maintain the FMD virus in their populations and that they are capable of transmitting the disease to cattle, with whom they may share pasture and watering sites.[4] Meanwhile, the buffalo themselves rarely, if ever, show clinical signs of the disease, and given their large number and free-roaming nature, they cannot all be vaccinated.[5]

Global FMD control policy, however, shows little recognition of this epidemiological role of the buffalo. It is based mostly on the understanding that emerged in nineteenth-century Britain that FMD is a serious livestock disease (rather than also a wildlife disease) that should be stamped out (rather than vaccinated for). While participants at the 2012 OIE–FAO global conference on FMD control noted the role of the buffalo in Southern Africa and accordingly adapted the international organisations' target to aim for eradication *in livestock only*, the successful implementation of this target remains unattainable in the presence of wildlife. This is because the target prescribes a situation of the full absence of FMD *without vaccination*, while vaccination is considered essential in Southern Africa, where livestock commonly co-occur with wildlife carrying FMD.[6] Only if wildlife and livestock are strictly separated, by veterinary fences or other barriers, would it (theoretically) be possible to eradicate FMD in livestock without vaccination. Yet this strategy remains unrealised in the wildlife-rich areas of Southern Africa primarily because elephants routinely break down fences.[7] Moreover, the policy of excluding local people and their livestock from wildlife areas is a colonial policy of 'fortress conservation'.[8] The fact that in the Southern African context buffalo are important actors in FMD epidemiology and cannot simply be put behind fences has largely escaped global FMD policy.

This lack of recognition signals unequal power and knowledge relations that reproduce historical patterns of colonial science. While British understandings of FMD have shaped global veterinary policy, contemporary Southern African understandings of the disease and its context are suppressed. In this article, we further problematise this lack of recognition by presenting a case study from Botswana in which we adopt a long-term historical and more-than-human perspective. By focusing on historical dynamics and non-human agency that led to the identification of the African buffalo as a disease reservoir for FMD, we reconstruct the colonial and postcolonial history of FMD knowledge in Botswana,

which was at the forefront of FMD research on the buffalo in Southern Africa in the 1960s and 1970s. In sum, our article makes two interrelated points: first, it shows that nineteenth-century British ideas about FMD and its control travelled to Botswana with serious consequences; second, it highlights how a local understanding of the disease emerged in Botswana that differed from British ideas, and – by extension – differs also from the WOAH's ideas about FMD today.[9]

Analytically, our article draws together postcolonial and more-than-human approaches in veterinary history. In Southern Africa, postcolonial scholars have examined knowledge exchanges between local veterinary practioners and scientists during the colonial era.[10] Beinart et al. have also argued that many colonial veterinarians were actually quite detached from the colonial agenda and developed a strong understanding of the local environment and animal diseases. Even if veterinary knowledge came from 'the metropole', it did not simply diffuse throughout the colonies but rather was translated to fit the local context.[11] Moreover, Cassidy et al. have reminded us that animals – next to human researchers and interlocutors, as well as scientific instruments and other co-constitutive parts of scientific inquiry – shape scientific, medical and veterinary knowledge production.[12] Similar to our approach, their study of the badger describes and analyses the role of animals in shaping understandings of a livestock disease (bovine tuberculosis) in Britain. Comparable to the African buffalo and FMD, the badger left various traces that were interpreted by people to mean that the wild animal maintained a livestock disease.

Our analysis takes us beyond European contexts and addresses the criticism of Saha and others who note that animal histories too often remain silent about historical dynamics in the Global South.[13] Specifically, our article shows how FMD science developed in its unique way in the African buffalo's natural geographic range (in our case looking specifically at Botswana). These findings suggest a need to provincialise FMD by taking seriously the

historical interaction between the African buffalo and colonial and postcolonial veterinarians that resulted in today's understanding of FMD in Botswana and Southern Africa.

Beyond a historiographical contribution, such provincialisation problematises current disease control policies. By reconstructing how local historical dynamics resulted in the association between FMD and the African buffalo, our research promotes a geographically nuanced and historically informed understanding of FMD science in which clear boundaries between colonial and postcolonial veterinary knowledge, and human and non-human actors, cannot be drawn. Our research therefore challenges the assumed universality of nineteenth-century British ideas about FMD, and exposes the ways in which these ideas shaped and continue to shape social, natural and economic realities in Southern Africa and beyond. These insights are important for the development and uptake of just global disease control policies.

Our argument is structured as follows: In the next section we describe the veterinarisation of Botswana. We show how Britain's colonial effort to set up an export-oriented cattle industry in the country and regulate the import of beef, combined to create a situation in which Botswana (then called Bechuanaland) came to adopt a strict approach to FMD control. Subsequently, we focus on the emergence of veterinary understandings of wildlife in FMD ecology. Based on archival material from the Botswana National Archives (BNA), we show how the strict approach to FMD control pressured veterinarians to act in a precautionary manner and to identify the African buffalo as the reservoir host for FMD. We conclude our article by drawing together our findings and returning to the question of global FMD control policies.

THE VETERINARISATION OF BOTSWANA

With the adoption of germ and importation theory in the mid nineteenth century, European and North American countries started applying veterinary regulations domestically as well as internationally.[14] Any country exporting beef or cattle to Europe and North America had to comply with strict rules.[15] In Britain, globally the largest importer of beef for most of the twentieth century, FMD was initially seen as a mild disease. Only from the end of the nineteenth century did the disease become viewed as a threat to be controlled.[16] Inspired by Britain, the United States, another important importer, started regulating FMD in a similar way.[17] Furthermore, many veterinarians in British colonies in Asia and Africa were trained in Britain and thus brought with them a similar understanding of the disease.

Also in Bechuanaland, created as a British protectorate in 1885, veterinary concerns evolved together with its beef industry. Its pre-existing cattle economy became industrialised under the rule of the British, who saw cattle as the primary resource for exploiting the semi-arid country. Immediately after colonisation, however, rinderpest arrived and wiped out a significant part of the cattle population, as it did elsewhere on the continent.[18] This experience spurred the growth of the veterinary sector in Southern Africa.[19] When Bechuanaland's cattle population eventually recovered at the turn of the twentieth century, its export to neighbouring Transvaal (currently part of South Africa) was subject to the latter's veterinary regulations, which were linked to lungsickness.[20] To meet the new veterinary regulations, British colonisers placed a permanent veterinary officer in Bechuanaland in 1905.[21]

By the 1920s, live cattle from Bechuanaland were exported to the Union of South Africa (where they mostly supplied the Witwatersrand mines) and to Italy. But again, these exports were subject to veterinary restrictions aimed at keeping East Coast fever and lungsickness out of South Africa, from where the beef for Italy was shipped.[22] When the first recorded FMD outbreak in Bechuanaland occurred in 1933, South Africa placed an embargo

on Bechuanaland to protect its own FMD-free status.[23] This was important because South Africa exported to Britain, and thus could face an embargo itself if the disease spread.[24] Bechuanaland established a veterinary cordon and embarked on an inoculation campaign, but did so with very limited government resources. Owing to these limited resources, the measures depended heavily on local chiefs, who mobilised hundreds of Africans to establish veterinary cordons in order to control cattle movement.[25] Indeed, available research confirms that FMD control was largely accepted by African authorities who considered it a tool to protect their own cattle herds.[26]

The veterinarisation of Bechuanaland accelerated after the Second World War, when a lack of food and a change in colonial welfare policy led Britain to invest more heavily in the protectorate's cattle industry.[27] In the 1950s, the newly set up Colonial Development Corporation built a large export abattoir in Lobatse that slaughtered cattle according to European standards of meat hygiene. Moreover, the Department of Veterinary Services built two major veterinary cordon fences to control cattle movement. While this enabled export to Britain and provided good prices for livestock producers, most exports went through European and African elites, and few benefits were captured by the rural poor, women and ethnically marginalised groups.[28] Owners of large cattle herds, on the other hand, used these funds to commercialise and intensify their farming operations.

In the years after independence in 1966, the newly independent state of Botswana continued to invest in the cattle industry and to promote the cattle accumulation-oriented agendas of the country's elites. For the latter, it was important to retain access to the highly valued export market of Britain (and later Europe). This required a strong central state that managed the Lobatse abattoir and that maintained a strong veterinary regime in line with European ideas about disease risk. In return for taking up these tasks, the state benefitted from the elites' backing. Thus the development trajectory that was set out by the British prior

to independence continued to be the path taken by postcolonial Botswana.[29] With the backing of cattle-owning elites, who profited and continue to profit most from the cattle export industry, a strong state was formed with responsibility for controlling disease, most notably FMD.[30]

However, as we show in the next section, veterinarians, who were tasked with disease control by the state, continuously struggled with their task. With the lurking threat of halted exports, they were pressured to find the cause behind recurrent FMD outbreaks. They therefore launched investigations on the role of wildlife, and were quick to assign blame, treating any evidence of potential threats as evidence of real threats. This precautionary approach led to various attempts to control wild animal populations, most notably targeted at the buffalo through the building of so-called 'buffalo fences' and the concurrent curbing of buffalo movements.

THE EMERGENCE OF VETERINARY UNDERSTANDINGS OF THE ROLE OF WILDLIFE IN FMD ECOLOGY

How did veterinarians in colonial and postcolonial Botswana come to see the buffalo as a disease reservoir? The notion of animal disease reservoirs already existed when FMD first appeared in the country. In the late nineteenth century, Zulu cattle keepers identified 'big game' as a source of *unakane* (anglicised as *nagana*, also known as animal trypanosomiasis for animals, and sleeping sickness for humans), and colonial veterinarians soon launched investigations and came to the same belief.[31] In the early twentieth century, wildlife thus became increasingly seen as 'vermin' by the colonial administration in South Africa, which aimed to expand settler farmland and saw wildlife as a threat. Similar to other countries in Southern Africa, wildlife was culled as a disease control measure, but conservationists

opposed this measure and contested the link between wildlife and trypanosomiasis. The colonial administration therefore decided to support efforts to find alternative disease-control measures.[32]

In the case of rinderpest, Kenyan veterinarians also considered wildlife as playing an important role in maintaining and spreading the disease in the early twentieth century.[33] And in 1924, wildlife was shortly associated with FMD in the United States when it was thought that the disease had spilled over to mule deer during an outbreak, leading to 22,000 of them being culled.[34]

Regarding the role of the wildlife as an FMD reservoir in Southern Africa, however, there has been little published. The few existing studies that discuss FMD in Southern Africa are mostly limited to the social and economic impacts and the control of FMD.[35] As far as we are aware, only Whande's work discusses FMD knowledge in detail, but it does so only for a specific place, namely Wankie Game Reserve.[36] Wood's work on the history of FMD in Britain is very extensive but remains silent about developments in Southern Africa.[37] In this section we thus draw mostly on archival materials and documents written by involved veterinarians at the time.

When FMD broke out in Southern Rhodesia (now Zimbabwe) in 1931, veterinary authorities first believed that wildlife played a negligible role in the spread of infection, given that the disease was not found in wildlife areas.[38] Soon, however, opinion changed as veterinary reports came in of cattle getting infected after being in close proximity to wildlife and the latter showing clinical signs typical of FMD. When the disease spread to Bechuanaland in 1933, reports mentioned that foot lesions similar to those found in cattle were found in wildebeest, and that the role of wildlife needed investigation.[39]

However, investigation of the role of wildlife remained limited until the 1960s, and only very circumstantial evidence accrued. The most significant role that wildlife was

thought to play was in causing spill-backs of FMD under human-created conditions through aphthisation. This technique was employed widely in Southern Africa and involved the deliberate infection of cattle populations in cordoned-off areas with a live virus to pre-empt the possibility of the disease smouldering in those cattle populations. Aphthisation was seen to run the risk of spill-over to wildlife because of the large quantities of virus circulating, and some hypothesised that wildlife would later pass the virus back to cattle after the immunity of cattle waned.[40]

Even so, in 1948 Bechuanaland's veterinary director, E. Dawe, stated that 'while game transmission is accepted as a possible factor in the spread of the disease, I am not aware of any evidence that it is very common or readily possible'.[41] Correspondingly, measures targeting wildlife continued to be excluded from the plan to control FMD, with the exception of the shooting of wildlife along veterinary cattle fences to prevent fence damage.

The role of wildlife first prominently came to light in Bechuanaland in discussions concerning a proposed game control policy in 1961. These discussions, which we will analyse in more detail below, show how despite large uncertainties regarding FMD ecology, authorities in Bechuanaland framed wildlife as killable vermin based on the perceived concerns of European veterinary authorities and the accompanying threat to Bechuanaland's export-driven cattle industry.

The game control policy debate

The game control policy debate happened in 1961, the year after Chobe Game Reserve (now Chobe National Park) was established. This reserve, Bechuanaland's first major one, was established despite the protest of the sitting director of the Department of Veterinary Services, K. Unsworth.[42] He held that 'in fostering the preservation of game in unfenced reserves we

are simply deliberately endangering our only industry and perpetuating the necessity to spend large sums of money in the control of outbreaks of foot and mouth disease'.[43] In his view, the only sensible remaining course of action for Bechuanaland was to fence the reserve and shoot all wildlife outside it under a game control policy.[44]

Unsworth and other veterinary directors in Southern Africa were members of the Inter-Territorial Committee on Foot and Mouth Disease, and at its meeting in April 1961 they agreed that 'the control of game was the basis of all foot and mouth disease control in Southern Africa'.[45] They reasoned that since cattle were not proven to maintain the disease, wildlife must be the culprit – an understanding that was supported by observations of typical lesions in wildlife and other circumstantial evidence. Unsworth communicated this understanding directly to his interlocutors in the game control policy debate.

Unsworth was, however, challenged by some of his interlocutors. Many of them pointed out that much was still uncertain about the role of wildlife in the spread and maintenance of FMD. For example, the preparatory paper for the debate, written by a game official, stated that:

> Game officials as a general rule have in the past disputed the danger of infection through the migration of game which is emphasised by those associated with the livestock industry. It has in fact been claimed in some quarters that definite proof has yet to be forthcoming that foot and mouth has ever been transmitted from game to domestic cattle.[46]

This sentiment was repeated in the debate that followed. First in the Executive Council – which was to approve the preparatory paper before the Legislative Council – some members expressed concern for the lack of evidence. And second, in the Legislative Council – which

was to decide on principles for the control policy – a disagreement emerged over which species of wildlife were the ones responsible for outbreaks of FMD.[47]

Yet despite the recognition of uncertainties, the need to act was recognised too, and generally it was felt that the role of wildlife in FMD ecology could not be ignored. Unsworth, for one, was 'satisfied that we know enough at this stage to persuade us it is necessary to bring about rapid reduction in game in cattle raising areas of the country'.[48] After the Legislative Council set out principles for the policy, the Administrative Conference further deliberated on them and stated that it 'accepted the fact that the preservation of cattle markets in neighbouring and overseas territories necessitated a reduction in game–cattle contacts whether or not the potential danger of game as a disease carrier was as great as these authorities were inclined to believe'.[49]

With this position, the Administrative Conference circumvented questions of science and instead focused solely on economic and international affairs. Mainland European veterinary authorities purportedly refused to import beef from Bechuanaland (or any country in Southern Africa) for the reason that a disease reservoir existed in the country in the form of wildlife.[50] Neighbouring Southern Rhodesia and the Republic of South Africa were considered to be strict with the import of beef and cattle from Bechuanaland too, for they also wanted to get rid of the disease and to export to the European market. The fact that they had fenced their game reserves, and Bechuanaland had not, was considered particularly significant by Unsworth, given that the development could have provided Southern Rhodesia and South Africa access to Europe before Bechuanaland, and thus hurt the latter's competitive position.[51]

In the end the game control policy was never formulated and Unsworth's suggestion to fence Chobe Game Reserve and shoot the wildlife outside it were not followed.[52] But the debate shows how the veterinary gaze fixated on wildlife in response to the perceived

concerns of European veterinary authorities. These concerns became powerful through the strong position of Bechuanaland's export-driven cattle industry. In the following sections we further analyse these power dynamics, paying particular attention to the way veterinary science entered and mediated these relations.

The buffalo fence

In 1965, after the arrival of the first FMD vaccine, a research programme was started in Bechuanaland to study the epidemiology of FMD and the effectiveness of vaccination.[53] The programme was led by the Department of Veterinary Services together with the Animal Virus Research Institute (AVRI) in Pirbright, England, which had developed the vaccine.[54]

The programme studied among other things the role of so-called 'carrier animals': cattle that could harbour live virus for a long time after infection, even after having recovered from disease. It was believed that these animals might be causing the frequent recurrence of FMD outbreaks. However, the programme failed to demonstrate that carrier animals were able to transmit the virus to susceptible cattle. Still aiming to resolve the mystery, the focus shifted from cattle to wildlife, particularly buffalo. As P.J. Posthumus, at the time the deputy director of the Department of Veterinary Services, recounts:

> We therefore ended up with the problem that we must know where the Foot and Mouth disease causing an outbreak comes from. We looked at cattle as a source and we found that certain cattle had harboured virus at certain times. We tried to transmit and we were unsuccessful. Now, where does the virus come from? So we looked at the next best thing, and that was wild game.[55]

This shift in focus to wildlife as a factor in the spread and maintenance of FMD, however, was not a complete surprise. As described in previous sections, some had long suspected and even accepted wildlife to play a role in FMD ecology. The regions of Ngamiland, Chobe, and Boteti, all in the north of the country, were associated with most disease outbreaks, and J. Falconer, who had succeeded Unsworth as veterinary director, stated that 'one factor was common to all three [regions]. This was that buffalo herds existed in these areas and made frequent contact with the cattle population'.[56]

Indeed, of all wildlife species, it was particularly the buffalo that veterinarians suspected. This was further supported by results of one of the earliest studies of FMD in wildlife conducted that same decade. This study took serum samples from 1,323 wild animals, mainly in Rhodesia but also elsewhere in the continent, including Botswana, and it found that of all species sampled, the highest proportion of animals with significant antibody levels was found among buffalo (77 from a total of 116 animals, compared to 102 out of 1,202 for all other sampled species).[57] Learning of this, the AVRI researchers and Botswana veterinarians decided to launch further investigations into the role of buffalo.

The first of these investigation took place in 1968, during an FMD outbreak in northern Botswana in which buffalo were suspected as being the source of the outbreak.[58] Veterinarians sampled the throats of eight shot buffalo to check for live virus and found it present in two of the animals. Three of seventeen buffalo sampled in Rhodesia were found to harbour the virus too.[59] This spurred another two investigations in Botswana: one in the following year, where 62 buffalo were sampled and virus was found in 35 of them; and one in 1970, when another 40 buffalo were sampled, upon which 12 were found to harbour the virus.[60] The investigations showed that buffalo harboured the virus in significant numbers, and these findings suggested that the animal played an important role in FMD ecology.

However, the findings were not irrefutable evidence of buffalo being the reservoir host for FMD. They were only suggestive of this because, although evidence of the presence of the virus was found, there was no proof of transmission. Nevertheless, the investigations led to the erection in 1970 of an 'anti-buffalo fence' just north of the Boteti region, which would – in concert with the shooting of buffalo along the fence – halt the migration of the animal from the north down to the cattle-rearing areas around the Boteti River, and thus prevent contact.[61] In a few years' time, it was expected, buffalo would change their migratory habits so that shooting and patrolling would become expendable. The fence, which later came to be known as the Northern Nxai Pan Fence, was the first of what are now commonly called the 'buffalo fences' (see Figure 1).[62]

Yet the fact that Falconer and the Department of Veterinary Services continued research the role of buffalo in 1970 even after the decision was made to erect the fence shows that fencing was a precautionary measure, and taken in spite of continued uncertainties.

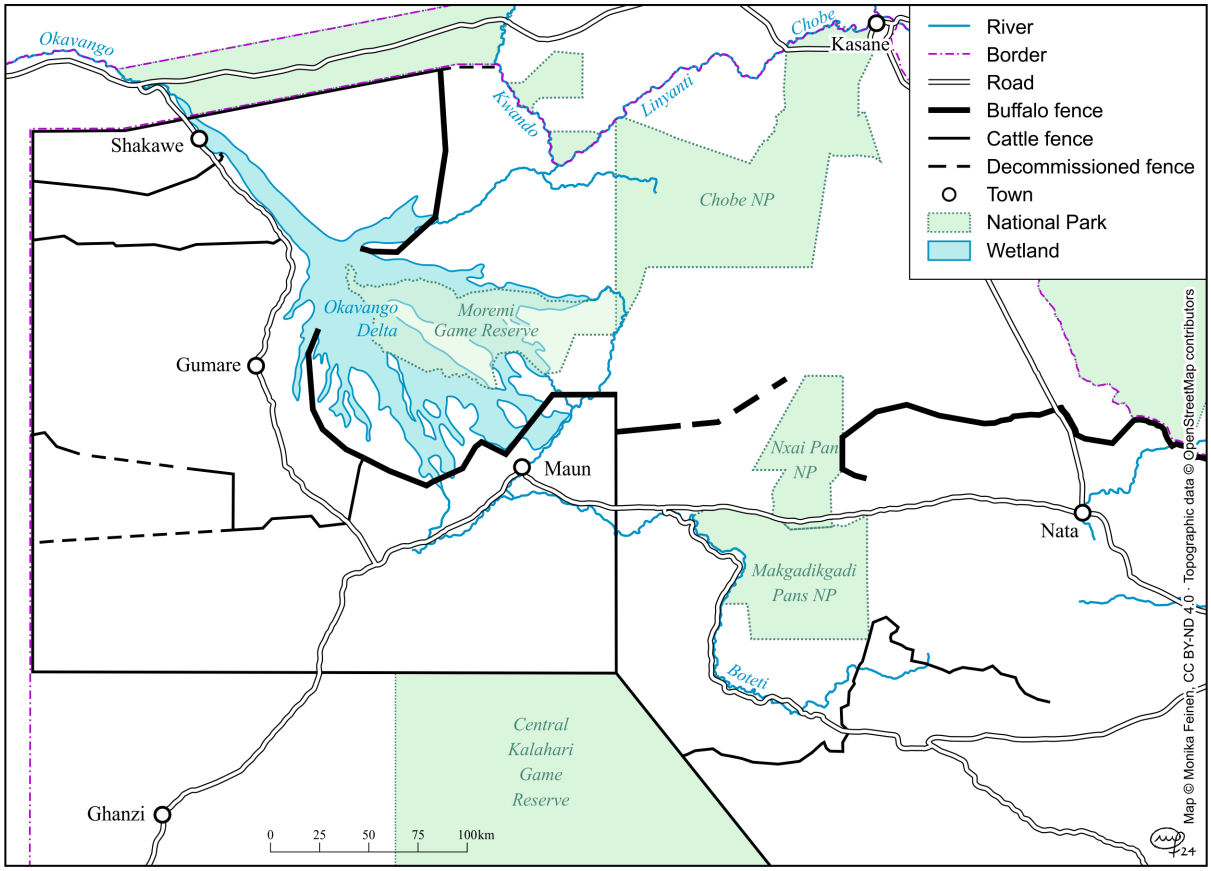

Figure 1: Buffalo fences in northern Botswana, 2025. Buffalo roam in the areas north and east of the fences, which include Chobe National Park and Moremi Game Reserve, while they are kept out of the areas to the south and west of the fences, which include the prominent cattle-rearing areas to the west of the Okavango Delta and around the Boteti River. Map by Monika Feinen.

The 'buffalo letter'

As we noted above, apart from the question of whether or not buffalo carried the virus, veterinarians also had to ask whether buffalo could transmit it. In this regard, the investigations came across a surprising finding. They found that buffalo harboured a virus of a serotype that had never been recorded in the country's cattle population.[63] Up to that date and since the Southern African Territories (SAT) serotypes were identified in 1948, cattle in Bechuanaland/Botswana had been found to be infected with the SAT1 and SAT3 serotypes, but researchers had not found the SAT2 virus in the cattle population.[64] If buffalo were

harbouring the SAT2 virus in their throats, as the investigations found, and cattle were not, could that mean that the virus circulated independently in buffalo and cattle populations?

Falconer, who had remained director of Botswana's veterinary service after independence, and AVRI researchers differed in their interpretations of the findings. For AVRI's director, J. Brooksby, it was clear: transmission from buffalo to cattle may not be as frequent or likely as had previously been thought. Buffalo might harbour the virus, but the findings suggested that spill-over to cattle was infrequent and occurred only under special circumstances. For Falconer, however, this was one step too far. He was convinced that 'buffalo … play a dominant role in the epizootiology of the disease in this country' and that the explanation for the SAT2 virus not having been transmitted to cattle may lie in 'simply a chance factor'.[65] It was essential that the parties agreed on a common understanding as they were co-authors of a scientific paper on the investigations, known as the 'buffalo letter'.

Ultimately, however, their professional opinions and interpretations of the findings mattered little. Rather what mattered was the way that British veterinary authorities received the findings. As Falconer suggested in his correspondence with his AVRI partners, the publication of the 'buffalo letter' could contribute to Britain getting stricter about accepting imports of beef from Botswana.[66] While the AVRI researchers tried to convince Falconer that the letter would be welcomed by the international community and that it should be published, Falconer was concerned that it might put Botswana in a bad light. Up until that point, vaccination in Botswana was done with inactivated virus of the SAT1 and SAT3 serotypes. The SAT2 serotype was never recorded in cattle and thus also not included in the vaccine formulation. The fear was that reporting the existence of SAT2 in the resident buffalo population would raise concern internationally.[67]

So even if the discovery of the SAT2 serotype in buffalo might suggest that transmission to cattle was absent or rare, Falconer decided to pre-empt the risk of

transmission by extending disease control. To protect cattle from the SAT2 virus, in 1970 the serotype was incorporated in the vaccine formulation and cattle were vaccinated with a trivalent vaccine from then onwards.[68] Despite Falconer's strong convictions regarding buffalo, he called this a 'precautionary measure', thus seemingly admitting that transmission from buffalo to cattle was uncertain.[69] The final text of the 'buffalo letter' (published in the British journal *Veterinary Record*), however, was less ambiguous, and stated that while the likelihood or frequency of transmission was uncertain and possibly low, the risk was there.[70]

This view, as expressed in the 'buffalo letter' and reflected in the decision to incorporate the SAT2 serotype in the vaccine, thus represented a shift in understanding of the role of buffalo and wildlife in FMD ecology. Initially, vaccination replaced aphthisation and aimed at stopping cattle from transmitting the virus to other cattle and wildlife (and if wildlife could not get infected, it could also not spill the virus back to cattle). Later, the incorporation of the SAT2 serotype was aimed at stopping wildlife potentially transmitting the virus to cattle. In this latter view, transmission of the virus from wildlife to cattle was to be characterised not as a spill-back but a spill-over: it was not a *re*introduction but an introduction. Indeed, it was at this moment that buffalo were constructed as a disease reservoir – a species that maintained the virus without the involvement of cattle, and capable of spreading it to cattle populations.

The buffalo exercises

While the issue of transmission remained uncertain, so too was the question of how commonly buffalo harboured the FMD virus. Following the first investigations in the late 1960s and 1970, veterinarians considered large-scale investigations necessary for more accurate knowledge. These started in 1972, when buffalo were sampled in large numbers in

the first of what some of the involved veterinary officers called the ‘buffalo exercises’. The buffalo exercises were a series of sampling operations between 1972 and 1976, in which, during each year with the exception of 1975, around 200 buffalo were darted (immobilised by being shot with a dart containing tranquiliser) and sampled (see Figure 2). The research project was led by the Department of Animal Health together with AVRI, but the Department of Wildlife was involved as well, and it was funded by the British government.[71]

Serious costs were involved, mainly for the hire of the helicopter used for finding and darting buffalo. In the first two years alone, the project spent 32,452 rand, equal to 17,623 British pounds at the time and more than a third of the government’s total planned spending for agricultural research for the period from 1970 to 1975.[72] These costs were seen to be warranted by the social and economic impact that the project was purported to have. As one researcher noted, ‘although the project is a basic research one, its practical importance lies in the fact that it is necessary to have a complete knowledge of the virus state in both domestic and wild animals to exercise sophisticated control’. And this – the project proposal added – was crucial to maintaining the export of livestock products that ‘a large proportion of the population will continue to be dependent on’.[73]

The operating team for the exercises was led by a German veterinarian and consisted of a further five vets, three or four technicians, game wardens and support staff, most of them foreigners.[74] One vet from Kruger Park in South Africa also spent some weeks in Botswana to help, and the team was regularly joined by AVRI officer R.S. Hedger from Britain. He had been involved in most of the research on FMD in Botswana up to then, as well as in neighbouring countries, and was responsible for most of the reporting.[75] The Department of Wildlife saw the mass immobilisation of buffalo as an opportunity to do some research of its own on the biology of buffalo and was interested in gaining experience with immobilisation

techniques (which were considered important for wildlife management but were still in their infancy).[76]

In the first two exercises in 1972 and 1973, 201 and 202 buffalo respectively were sampled, and virus was recovered from around 60 per cent of the sampled population in both years. A third exercise was conducted in 1974, sampling 168 buffalo, but this time researchers found that only 20 per cent of the animals were infected. While reasons related to methodology could explain this surprising result (for example, more calves rather than adults were sampled, and samples were handled and stored incorrectly), the possibility was raised that the disease might be dying out, and funding was sought for another exercise to test this hypothesis.[77] The hypothesis was, however, rejected, after another 196 buffalo were sampled in 1976, and the percentage of infected animals was found to be as high as 70 per cent.[78]

Figure 2: Shooting buffalo with tranquiliser darts during the buffalo exercises. Source: 'Department of Animal Health and Tsetse-Fly Control activity report

1975–1980', BNA BNB 4798.

What the buffalo exercises showed was that buffalo harbored the virus, either alive in their throats at the moment of measurement, previously present as shown by their antibodies or clinically apparent as lesions on the tongue or between hooves. Moreover, they harboured all three of the region's known virus serotypes. Yet transmission from buffalo to cattle was never demonstrated. The only research that aimed – but failed – to show the occurrence of this phenomenon took place in Rhodesia a few years prior.[79] The buffalo exercises therefore concluded that still further research was needed, especially concerning the transmission of the virus from buffalo to cattle. However, instead of noting a lack of understanding regarding the *occurrence* of transmission, the reporting noted uncertainty regarding the *mechanism* of transmission, assuming its occurrence.[80] Similar to the reporting in the buffalo letter, the buffalo was thus again cast as a disease reservoir.

Since the late 1970s, a further three buffalo fences have been built in Botswana, largely without questioning the role of buffalo in FMD ecology. By the time transmission of FMD from buffalo to cattle was convincingly demonstrated in experimental and field conditions in 1994, the country-wide network of buffalo fences was already nearly complete.[81] Today, there still remains uncertainty regarding the mechanism and frequency of transmission, but this has not resulted in a full-blown controversy. While one buffalo fence has been partly removed after lobbying from wildlife conservationists, the others remain.[82] Namibia, South Africa and Zimbabwe also still have buffalo fences (although their extent is much less).

CONCLUSION

This article has shown that the historical interaction between veterinarians and the African buffalo played a constitutive role in FMD science as it was practised in Botswana. Yet, we have also shown how – besides this local interaction – external pressure played a role in constructing the buffalo as a disease reservoir. This pressure originated from the export-oriented cattle industry set up by the British and their ideas about FMD as a contagious disease to be controlled. In writing this history, we have built on postcolonial and more-than-human work in veterinary history that has carved out a space for Southern African and more-than-human understandings of animal diseases.

In relation to present-day global FMD control policies, our historical analysis deepens our understanding of current knowledge/power relations by showing how since the colonial period British ideas about the disease have penetrated knowledge and lives in Southern Africa, while this has not yet really happened in the reverse direction. Such reverse movement, however, is essential because we have also shown that the Southern African understanding of how the disease is maintained and spread is unique, while it is not adequately recognised in global FMD control policy. Thus our hope is that our contribution to a historically informed and geographically nuanced understanding of FMD is a step towards the development and uptake of FMD control policies that challenge colonial power asymmetries.

Indeed, global FMD control policy has given little way in acknowledging and accommodating the difference between FMD ideas in Europe and Southern Africa with its target of the global eradication of FMD in livestock. It directs countries towards the spatial segregration of wildlife and cattle through the building of buffalo fences, which is unsustainable and especially harmful for migrating wildlife and farmers living close to buffalo in buffer zones where disease control efforts are concentrated.[83] Immunisation through vaccination would instead allow farmers to live with FMD and buffalo, and recently

the WOAH has recognised vaccination (in combination with a thirty-day quarantine and specified slaughter procedures) as reducing the risk of disease transmission sufficiently for international trade to take place safely. Such a 'commodity-based trade' approach can replace the fence-based approach to FMD control.[84] It would be befitting to support the countries of Southern Africa in their pursuit of this approach, and to stop prescribing the *global* target of eradication of FMD in livestock.

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fence damage, lack of protection zones, livestock theft and owner apathy: Complex socio-ecological dynamics in foot and mouth disease control in Southern Africa', *Pastoralism* **6** (2016): 1-12.

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[9] In this article we thus use the case of Botswana to analyse the relation between Britain/Europe and Southern Africa rather than regional dynamics in Southern Africa.

[10] W. Beinart and K. Brown, *African Local Knowledge and Livestock Health: Diseases and Treatments in South Africa* (Martlesham: Boydell and Brewer, 2013).

[11] W. Beinart, K. Brown and D. Gilfoyle, 'Experts and expertise in colonial Africa reconsidered: Science and the interpenetration of knowledge', *African Affairs* **108** (2009): 413-433. For a constrasting perspective, see W. Mwatwara, 'Veterinary history in Southern Africa', *Oxford Research Encyclopedia of African History* (2024): 1-24. In our case, we also focus on these colonial and foreign scientists, who played an important role in constructing the buffalo as a disease reservoir. We are, however, critical of their interests and acknowledge the limitation of our study by not including African understandings of FMD.

[12] A. Cassidy et al., 'Animal roles and traces in the history of medicine, c.1880–1980', *British Journal of the History of Science Themes* **2** (2017): 11–33.

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[14] M. Worboys, 'Germ theories of disease and British veterinary medicine, 1860–1890', *Medical History* **35** (1991): 308-327.

[15] R. Perren, *Taste, Trade and Technology: The Development of the International Meat Industry Since 1840* (London: Routledge, 2017).

[16] A. Woods, *A Manufactured Plague: The History of Foot-and-Mouth Disease in Britain* (London: Routledge, 2004).

[17] A.L. Olmstead and P.W. Rhode, *Arresting Contagion: Science, Policy, and Conflicts over Animal Disease Control* (Cambridge, MA: Harvard University Press, 2015).

[18] J. Falconer, 'History of the Botswana Veterinary Services – 1905–66', *Botswana Notes and Records* **3** (1971): 74-78.

[19] K. Brown and D. Gilfoyle, 'Introduction: Livestock diseases and veterinary science in Southern Africa', *South African Historical Journal* **58** (2007): 2-16.

[20] S. Milton, '"To make the crooked straight": Settler colonialism, imperial decline, and the South African beef industry c. 1902–42' (PhD thesis, University of London, 1996).

[21] Falconer, 'History of the Botswana Veterinary Services'.

[22] Milton, 'To make the crooked straight'.

[23] While the 1933 outbreak is the first *recorded* outbreak of FMD, the disease appears to have been around far longer. According to an evolutionary analysis of the genes of FMD, the virus arrived in Africa from Europe at the start of the eighteenth century and went through a bottleneck during the rinderpest epizootic at the end of the nineteenth century. See D.C. Tully and M.A. Fares, 'The tale of a modern animal plague: Tracing the evolutionary history and determining the time-scale for foot and mouth disease virus', *Virology* **382** (2008): 250-256.

[24] In contrast, when domestic demand for beef rose in South Africa in the 1940s, and export became less important, FMD regulations towards Bechuanaland were eased significantly. See

M. Hubbard, *Agricultural Exports and Economic Growth: A Study of the Botswana Beef Industry* (London: KPI, 1986).

[25] S. Milton, 'Western veterinary medicine in Colonial Africa: A survey, 1902–1963', *Argos* **18** (1998): 312-322.

[26] D.J. McGahey, 'Maintaining opportunism and mobility in drylands: The impact of veterinary cordon fences in Botswana' (PhD thesis, University of Oxford, 2008). The African authorities and their circles, as well as other wealthy Africans and European settlers, largely controlled the boreholes that were drilled by the colonial government in the 1930s and thereby came to dominate much of the beef industry. See P.E. Peters, *Dividing the Commons: Politics, Policy, and Culture in Botswana* (Charlottesville: University of Virginia Press, 1994). For a detailed exploration of African reactions to colonial veterinary policy, see W. Mwatwara, '"Even the calves must dip": East Coast fever, Africans and the imposition of dipping tanks in Southern Rhodesia, c. 1902–1930', *South African Historical Journal* **66** (2014): 320-348.

[27] The lack of beef was caused by the rise of domestic demand in the United States and Australia, and New Zealand exports shifting from Britain to the United States. Meanwhile, British colonial policy aimed to make its colonies more economically independent to prepare for decolonisation. Hubbard, *Agricultural Exports and Economic Growth*.

[28] A. Petitt, 'Women's cattle ownership in Botswana: Rebranding gender relations?' (PhD thesis, University of Uppsala, 2016).

[29] This applied to Southern Africa more widely. Mwatwara, 'Veterinary history in Southern Africa'.

[30] Ø. Gulbrandsen, *The State and the Social: State Formation in Botswana and Its Precolonial and Colonial Genealogies* (Oxford: Berghahn Books, 2022); B.G. Gumbo, 'The

dynamics of cattle farming, bovine disease and state intervention in North West District of Botswana, 1966–2000', *Botswana Notes and Records* **50** (2018): 72-84.

[31] M.A.D. da Silva, O. French, F. Keck and J. Skotnes-Brown, 'Introduction: Disease reservoirs: From colonial medicine to one health', *Medical Anthropology* **42** (2023): 311-324.

[32] K. Brown, 'From Ubombo to Mkhuzi: Disease, colonial science, and the control of nagana (livestock trypanosomosis) in Zululand, South Africa, c. 1894–1953', *Journal of the History of Medicine and Allied Sciences* **63** (2008): 285-322. In Bechuanaland, veterinarians also defined 'big game' as a reservoir for *nagana*, and in the period from 1940 till the 1960s, over sixty thousand animals were killed. 'The history of tsetse fly control in Botswana', Botswana National Archives (hereafter BNA) BNB 6076.

[33] T. Sunseri, 'The politics of rinderpest control on the Kenya–Tanganyika border, c. 1920–1940', *Journal of Eastern African Studies* **18** (2024): 627–648.

[34] G.V. Weaver, J. Domenech, A.R. Thiermann and W.B. Karesh, 'Foot and mouth disease: A look from the wild side', *Journal of Wildlife Diseases* **49** (2013): 759–785.

[35] See, e.g., I. Scoones and W. Wolmer, 'Land, landscapes and disease: The case of foot and mouth in Southern Zimbabwe', *South African Historical Journal* **58** (2007): 42-64; G. Miescher, *Namibia's Red Line: The History of a Veterinary and Settlement Border* (New York: Palgrave Macmillan, 2012).

[36] T.E. Whande, 'Human, livestock and wildlife interactions at the boundary of Hwange National Park and Tsholotsho Communal Areas in Zimbabwe' (PhD thesis, University of Cologne, 2023).

[37] Woods, *Manufactured Plague*.

[38] J.B. Condy, 'A study of foot-and-mouth disease in Rhodesian wildlife' (FRCVS thesis, Royal College of Veterinary Surgeons, 1971).

[39] J. Walker, 'Some observations on foot-and-mouth disease in the Bechuanaland Protectorate', *Journal of the South African Veterinary Medical Association* **5** (1934): 73-91.

[40] Falconer, 'History of the Botswana Veterinary Services'; 'Department of Animal Health and Tsetse-Fly Control activity report 1975–1980', BNA BNB 4798.

[41] 'Control of cattle movement', BNA S.52/2/2.

[42] There was a prior attempt at establishing a game reserve in Chobe in 1932, but this was not approved for financial reasons. A no-hunting zone was established instead, which lasted until 1943. Even then, the proposal was vehemently opposed by the veterinary department. See A. Campbell, 'Establishment of Botswana's national park and game reserve system', *Botswana Notes and Records* **36** (2004): 55-66.

[43] 'Foot-and-mouth disease, 1960', BNA S.578/9.

[44] The veterinarian view that wildlife and its conservation pose a threat has also been described by others. See, e.g., J. Carruthers, 'Influences on wildlife management and conservation biology in South Africa c.1900 to c.1940', *South African Historical Journal* **58** (2007): 65-90; J.M. MacKenzie, 'Experts and amateurs: Tsetse, nagana and sleeping sickness in East and Central Africa', in J.M. MacKenzie (ed.), *Imperialism and the Natural World* (Manchester: Manchester University Press, 2017), pp. 187-212; T. Sunseri, 'The game factor: Tanganyikan rinderpest campaigns, fence ecology and the wildlife threat to Southern Africa, 1938–1956', *Journal of Southern African Studies* **50** (2024): 263-284; Brown, 'From Ubombo to Mkhuzi'.

[45] 'Foot-and-mouth disease, 1960', BNA S.578/9. The Inter-Territorial Committee on Foot and Mouth Disease was an informal body for directors of veterinary departments in Southern Africa to discuss FMD (and later also other diseases). It was established in the late 1940s and continued to convene every few years until the 1970s. Falconer, 'History of the Botswana Veterinary Services'.

[46] 'Game control policy', BNA S.584/1/1.

[47] Ibid.

[48] Ibid.

[49] Ibid.

[50] It is unlikely that the presence of a wildlife reservoir would have been the real motivation behind the refusal of beef imports. The European Economic Community (established in 1957) hampered beef imports not just from Bechuanaland but also from Argentina, where no wildlife reservoir was suspected. A. Woods, 'Foot and mouth disease in twentieth-century Britain: Science, policy and the veterinary profession' (PhD thesis, University of Manchester, 2002).

[51] 'Game preservation committees', MOA2 6/35.

[52] C. A. Spinage, 'Further gleanings of game affairs in the Bechuanaland Protectorate', *Botswana Notes and Records* **24** (1992): 33-38.

[53] J. Falconer, 'The Epizootiology and Control of Foot-and-Mouth Disease in Botswana', *Veterinary Record* **91** (1972): 354-359; Falconer, 'History of the Botswana Veterinary Services'.

[54] For the history of the AVRI see 'Our history', Pirbright Institute, https://www.pirbright.ac.uk/about-us/our-history (accessed 17 September 2025).

[55] 'Foot and mouth disease research', BNA MOA2 11/16.

[56] Falconer, 'Epizootiology'; 'International territorial foot and mouth disease advisory committee', BNA MOA2 6/4.

[57] J.B. Condy, K.A.J. Herniman, and R.S. Hedger, 'Foot-and-mouth disease in wildlife in Rhodesia and other African territories: A serological survey', *Journal of Comparative Pathology* **79** (1969): 27-31; R.S. Hedger, J.B. Condy and J. Falconer, 'The isolation of foot-and-mouth disease virus from African buffalo (*Syncerus caffer*)', *Veterinary Record* **84**

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[58] R.S. Hedger, 'The maintenance of foot and mouth disease in Africa' (PhD thesis, London School of Hygiene and Tropical Medicine, 1976).

[59] Hedger et al., 'Isolation of foot-and-mouth disease virus'; R.S. Hedger, 'Foot-and-mouth disease in wildlife with particular reference to the African buffalo (*Syncerus caffer*)', in L.A. Page (ed.), *Wildlife Diseases* (New York: Plenum Press, 1976), pp. 235–244.

[60] R.S. Hedger, 'Foot-and-mouth disease and the African buffalo (*Syncerus caffer*)', *Journal of Comparative Pathology* **82** (1972): 19–28; Falconer, 'Epizootiology'.

[61] 'Foot and mouth disease research', BNA MOA2 11/16; 'International territorial foot and mouth disease advisory committee', BNA MOA2 6/4.

[62] It was, however, not the first veterinary wildlife fence. In Botswana the first one was built in 1945 between Maun and the Okavango Delta to facilitate the tsetse fly control department with game control (to curb the spread of the tsetse fly). Elsewhere on the continent fences were erected even earlier. For example, in 1941/42 a wildlife fence was built in Tanganyika to stop the spread of rinderpest into Northern Rhodesia. 'History of tsetse fly control', BNA BNB 6076; Sunseri, 'Game factor'.

[63] Hedger et al., 'Isolation of foot-and-mouth disease virus'.

[64] Hedger, 'Maintenance of foot and mouth disease'.

[65] 'Foot and mouth disease research', BNA MOA2 11/16; Falconer, 'Epizootiology', 359.

[66] This concern arose in the context of a British import ban that due to miscommunication was understood by Falconer to target African territories specifically, but was later clarified by Britain to target all exporting countries where it considered FMD to be endemic. This import ban was announced in response to the devasting 1967/68 FMD outbreak in Britain, and at

first only targeted Argentina, but in a diplomatic move to improve relations with Argentina came to include all exporting countries. Woods, *Manufactured Plague*.

[67] 'Foot and mouth disease research', BNA MOA2 11/16.

[68] 'International territorial foot and mouth disease advisory committee', BNA MOA2 6/4; 'Department of Animal Health annual report, 1972', BNA BNB 2068; Falconer, 'Epizootiology'.

[69] 'International territorial foot and mouth disease advisory committee', BNA MOA2 6/4.

[70] Hedger et al., 'Isolation of foot-and-mouth disease virus', 516.

[71] 'Department of Animal Health and Tsetse-Fly Control activity report 1975–1980', BNA BNB 4798.

[72] M.B.K. Darkoh and J.E Mbaiwa, 'Globalisation and the livestock industry in Botswana', *Singapore Journal of Tropical Geography* **23** (2002): 149-166.

[73] 'Foot and mouth disease research', BNA MOA2 11/16.

[74] L. Patterson, *Aiming to Save: A Vet's Life in Conservation* (Market Harborough: Book Guild, 2022).

[75] 'Foot and mouth disease research', BNA MOA2 11/16; 'Foot and mouth disease research project proposals', BNB MOA2 7/15.

[76] 'Studies on the population biology of the African buffalo (*Syncerus caffer*) in Botswana', BNA BNB 7400; 'Report on the Department of Wildlife and National Parks 1969–70', BNA BNB 1764; 'Report on the Department of Wildlife and National Parks 1971–1972', BNA BNB 2069; N. Dräger, L. Patterson and D. Breton, 'Immobilization of African buffalo (*Syncerus caffer coffer*) in large numbers for veterinary research', *African Journal of Ecology* **14** (1976): 113-120.

[77] 'Foot and mouth disease research', BNA MOA2 11/16; 'Department of Animal Health and Tsetse-Fly Control activity report 1975–1980', BNA BNB 4798.

[78] 'Foot and mouth disease research', BNA MOA2 11/16.

[79] JB Condy and RS Hedger, 'The survival of foot-and-mouth disease virus in African buffalo with non-transference of infection to domestic cattle', *Research in Veterinary Science* **16** (1974): 182-185.

[80] 'Department of Animal Health and Tsetse-Fly Control activity report 1975–1980', BNA BNB 4798; 'Foot and mouth disease research', BNA MOA2 11/16.

[81] P.S. Dawe et al., 'Natural transmission of foot-and-mouth disease virus from African buffalo (*Syncerus caffer*) to cattle in a wildlife area of Zimbabwe', *Veterinary Record* **134** (1994): 230-232; P. S. Dawe et al., 'Experimental Transmission of Foot-and-Mouth Disease Virus from Carrier African Buffalo (Syncerus Caffer) to Cattle in Zimbabwe', *Veterinary Record* **134** (1994): 211-215.

[82] Kalahari Conservation Society, 'Removal of decommissioned veterinary fences in Ngamiland District, Botswana' (Gaborone: Kalahari Conservation Society, 2005).

[83] Darkoh and Mbaiwa, 'Perceived Effects of veterinary fences'; L.V. Basupi, C.H. Quinn and A.J. Dougill, 'Historical perspectives on pastoralism and land tenure transformation in Ngamiland, Botswana: What are the policy and institutional lessons?' *Pastoralism* **7** (2017): 1-14.

[84] SADC and AHEAD, 'Guidelines'.