

**Foresight**

Infectious Diseases: preparing for the future

OFFICE OF SCIENCE AND INNOVATION

**T5.9: A case study of Rinderpest in Africa**

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*Rinderpest introduced into the Central Highlands of Ethiopia in 1988 by the cattle trade with southern Sudan*

## **REVIEW**

### **Foreword**

In developing our understanding of current risks, their drivers and their likely outcomes it is worthwhile to build on experience from more than a century of fighting rinderpest. Therefore this review starts with a history of rinderpest, primarily focused on Africa.

Not for nothing did Scott and Provost (1992) describe rinderpest as ‘... the most dreaded bovine plague known, belonging to a select group of notorious infectious diseases that have changed the course of history. From its homeland around the Caspian Basin, rinderpest, century after century, swept west over and around Europe and east over and around Asia with every marauding army, causing the disaster, death and devastation that preceded the fall of the Roman Empire, the conquest of Christian Europe by Charlemagne, the French Revolution, the impoverishment of Russia and the colonisation of Africa.’ That devastation continued through the 20<sup>th</sup> and even into the 21st Centuries.

## Introduction

Whilst the early years of the 20<sup>th</sup> Century were marked by widespread experience of cattle plague throughout Asia, parts of Europe and Africa, the written record is meagre and generally only particularly dramatic events are described. Few sought to record rinderpest events as diligently as Nawathe and co-workers in Nigeria (see, for example, Nawathe and Lamorde 1982; Nawathe and Lamorde 1985; and Nawathe et al 1983). Because of this it can be difficult to discern any epidemiological pattern within the overall picture of viral persistence. Such patterns are obscured by long-lived epizootic, even panzootic, waves of infection interspersed with interludes of apparent disease absence. The background enzootic rinderpest situation was frequently accepted as 'sporadic' occurrence without any recognition that this implies only that the epizootiological connections between outbreaks was not understood. Yet, as we have recognised in recent years, these epizootics must have had their origin in areas of enzootic persistence. Indeed the epidemiological situation can be glimpsed by examination of such 'grey literature' as does exist. Few formal publications on rinderpest occurrence in the field are available to consult after about 1970 because few chose to, or were permitted to, publish their observations. Recording rinderpest occurrence came to be seen as 'sensitive' because of its potential impact on the international trade in livestock and their products. This is probably the single most important factor that has bedevilled rinderpest investigation and control, leading to an under-recording of enzootic disease situations and even epizootics. Official disease records, such as those maintained by the OIE<sup>1</sup>, do not disclose the Second African Rinderpest Panzootic of the 1980s (see below) because many countries did not notify outbreaks, while others continued mechanically to report the presence of rinderpest even though it was not present.

During the 20<sup>th</sup> Century and into the early years of the 21<sup>st</sup> Century, rinderpest was confined essentially to Asia and Africa although its potential to invade other continents was clearly demonstrated. This was shown by the movement of the virus in trade cattle to Belgium, and then on to Brazil, from India in 1920, also via cattle infected by pigs in Singapore to Australia in 1923 (Throssel 1980), where it infected 28 herds within seven weeks and twice it found its way into Italy in the 1950s via wild animals from Somalia. Fortunately, even though the virus did start to become established in the first three of these long-distance rinderpest movements, it was eliminated rapidly by stamping-out combined with livestock movement controls.

Although progress was frequently disrupted by global and regional conflicts which favoured the resurgence of rinderpest, the 20th Century overall saw a progressive increase in its control and a reduction in its impact, particularly once safe and efficacious vaccines became widely available in mid-Century.

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<sup>1</sup> *Office international des Epizooties*, an international standard-setting organisation also known as the World Animal Health Organisation, and founded in 1924 to fight rinderpest and contagious bovine pleuropneumonia

In the post-colonial era, the collapse in the effectiveness of official veterinary services allowed the resurgence of rinderpest in Africa from reservoirs of infection not eliminated by institutionalised annual mass vaccination, feebly pursued.

## **Progressive control and eradication**

No-one reading an account of veterinary services during the early years of the 20<sup>th</sup> Century can fail to be struck by their preoccupation with rinderpest control. In many countries this was the prime reason for their foundation. However, as the 20<sup>th</sup> Century entered its last decade, we could be reasonably confident that rinderpest viruses were restricted primarily to a small number of persisting reservoirs of infection. Now, five years into the first decade of the 21<sup>st</sup> Century, only one of the three rinderpest virus lineages is possibly extant and in a relatively small area of the Greater Horn of Africa at that. There is growing confidence that the whole of Asia and most of Africa has been freed from the devastation of rinderpest.

Vaccines were initially used year-round to combat rinderpest outbreaks. But once they became routinely available, many countries implemented annual, pulsed vaccination campaigns throughout their territory or in border areas considered to be particularly vulnerable to invasion. However, little progress was made in Africa towards eradication until the advent of international coordination with the inception of Joint Project 15 (JP15; Lepissier, 1971). This was the first of the three internationally supported and coordinated rinderpest eradication campaigns which dominate the history of African rinderpest during the second half of the 20<sup>th</sup> century.

It had earlier been demonstrated in Asia by the Food and Agriculture Organization of the United Nations (FAO) that intensified vaccination and the international coordination of vaccination across international borders could be a successful technique to free large areas from rinderpest (Hambridge 1955; Hudson 1960). JP15 demonstrated this again in Africa but it neglected to develop the capacity to detect and eliminate residual reservoirs of infection. In the early 1980s a second rinderpest epizootic swept across sub-Saharan Africa, undoing most of what had been achieved. The resulting Pan-African Rinderpest Campaign (PARC) succeeded in reclaiming much of the situation (Taylor, 1990) yet it too ended in 1999 without eradicating the virus from Africa. At the present time the successor to PARC – the Pan-African Control of Epizootics (PACE) programme - is mandated to take the credit for the eradication of rinderpest from Africa.

Coordinated mass vaccination undoubtedly suppressed the disease considerably and even cleared large areas and their resident cattle populations completely. It did not, however, prevent periodic resurgences in panzootic form nor eliminate the reservoirs of infection. These were not clearly

defined until the advent of the Global Rinderpest Eradication Programme<sup>2</sup> (GREP) in the 1990s. A series of FAO Technical and Expert Consultation meetings held in Rome (FAO 1996, 1998 and 1999) concurred with the identification of six geographically-defined areas of prime concern because of the known or suspected existence of reservoirs of rinderpest virus. These comprised four areas in Asia (Pakistan with Afghanistan; Asiatic Russia with Mongolia and China; Yemen with Saudi Arabia; and Turkey with Iraq and Iran - 'The Kurdish Triangle'), and two in Africa (southern Sudan with contiguous areas of Kenya, Ethiopia and Uganda, and southern Somalia with northeastern Kenya and southern Ethiopia, comprising the southern 'Somali pastoral ecosystem', with periodic involvement of southern Kenya and northern Tanzania). This proved to be an accurate assessment and there has been no cause to suspect the presence of rinderpest endemicity outside the areas defined for special attention. These areas will be given emphasis in the following account together with an explanation of the development of control/eradication precepts. The 'Intensified GREP' was implemented from 1999 to 2004. It focused primarily on:

- refining our understanding of reservoirs and the containment of infection within them;
- raising community and professional awareness of rinderpest and the factors facilitating its persistence;
- strategy setting;
- intensive, focused immunisation to eliminate epidemiologically defined foci with seromonitoring of vaccination efficacy where possible;
- timely withdrawal of vaccination to allow verification of freedom.

Accompanying the eradication effort was a drive to enlist countries in the process of accreditation of rinderpest freedom by means of the 'OIE Pathway' (OIE 2004).

As a result, progress in assured rinderpest eradication has been rapid in the past 10 years. All but one of the areas of concern identified above, in five of which rinderpest was endemic into the second half of the last decade of the 20<sup>th</sup> Century, yielded to the eradication effort and have been cleared of infection. At the end of 2004, there can be little doubt that the whole of Asia has been free from rinderpest virus infection since early 2001 and the same can be assuredly stated for most of Africa except for a small part of eastern Africa. Remarkably, there has been no re-appearance of rinderpest in any of the former endemic foci, nor any resurgence of rinderpest elsewhere which could have indicated the presence of cryptic foci of infection. Its absence even from the warfare and its aftermath in Afghanistan and Iraq is quite remarkable and in sharp contrast to the situation which pertained in the previous decade. Then a resurgence of rinderpest in Turkey, Iraq and Iran resulted from the

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<sup>2</sup> Hosted and funded by FAO Regular Programme and donors through FAO Trust Fund and FAO Technical Cooperation Programme emergency projects

Gulf War of 1991, while rinderpest's spread in Afghanistan in 1995 was facilitated by civil disturbance.

From the 1980s, the Joint FAO/IAEA<sup>3</sup> Division, Animal Production and Health Section, provided technical guidance and assistance to GREP by coordinating seromonitoring campaigns for rinderpest. It subsequently evolved their focus to supporting rinderpest diagnosis and serosurveillance. This support continued as a PACE activity into 2004. In providing this support, the FAO/IAEA Division worked closely with the Institute for Animal Health, Pirbright Laboratory (formerly the Animal Virus research Institute), in UK, and in 1994 this laboratory was designated the FAO World Reference Laboratory for Rinderpest. Its provision of diagnostic and molecular technology for morbilliviruses made an invaluable contribution to epidemiological understanding and to control based on it. Molecular epidemiology demonstrated the existence of three clades of rinderpest virus responsible for outbreaks of rinderpest in the last 50 years, designated as the Asian Lineage, limited to Asia, and African Lineages 1 and 2, limited to the African continent.

A brief history of rinderpest occurrence and its determinants since 1900 follows. In it, an attempt is made to provide an epidemiological overview rather than a precise catalogue of outbreaks in order to understand why and how rinderpest has been brought to the brink of extinction. It owes much to a retrospective analysis of events using information from project reports, many official meetings and informal discussions, combined with the evidence provided by molecular epidemiology.

## **The evolution of rinderpest in sub-Saharan Africa**

After its introduction into Eritrea in eastern Africa<sup>4</sup> in approximately 1890, from cattle imported to feed invading armies, rinderpest moved down the eastern seaboard of Africa in the form of a disease of grazing animals both domestic and wild, reaching the Transvaal of southern Africa before the end of the nineteenth century. It never penetrated the dense forests to the west. It is unclear whether rinderpest had already been present in west Africa before the fatal introduction into eastern Africa, but the contemporaneous panzootic was equally marked there.

The severity of the impact of rinderpest in southern Africa compelled the Cape government in South Africa to invite distinguished scientists to assist in the investigation of methods for controlling it. The bile method, which involved the injection of bile obtained from cattle that had died of rinderpest, led on to the simultaneous injection of serum from rinderpest convalescent animals and virus (bile or spleen tissue) from animals dead from rinderpest. The serum-virus injection method was further refined to become the first successful

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<sup>3</sup> International Atomic Energy Agency

<sup>4</sup> It is likely that rinderpest had been introduced previously on one or more occasions but failed to generate a panzootic such as was seen in the 1890s

procedure for mass vaccination against rinderpest (Anon 1905; Kolle and Turner 1897; Turner, 1906;).

This method formed the main tool of international disease control that was used systematically by southern African countries to control rinderpest progressively from the Cape of Good Hope in South Africa to the Central (east-west) Railway Line in Tanzania between 1900 and 1930's (Branagan and Hammond 1965). This region has remained free of rinderpest since 1940. Three factors stand out as major contributors to the success of this first African regional rinderpest control programme:

- Vigorous application of quarantine, movement control and specific disease control measures, i.e. serum-virus vaccination to all cattle in prescribed areas;
- Close inter-country collaboration which included cross-border involvement of veterinary officials from the region in the rinderpest control campaigns;
- A regional system of inspection and independent verification of rinderpest freedom, designed and implemented by the veterinary authorities of the region.

A regional early warning system was comprised initially of designated veterinary inspectors from the region posted at the Tanzania-Malawi-Zambian border interface. This system was dismantled once rinderpest was eliminated from southern Tanzania. Nevertheless, until now, some 60 years later, successive Directors of Veterinary Services in Tanzania have maintained a convention of informing their neighbours of rinderpest events.

At the turn of the century Abyssinia (now Ethiopia and Eritrea) still harboured rinderpest and with the return of public security to Sudan by the British reconquest of the country and the resumption of local trade across the border, rinderpest became a real threat to Sudanese livestock which had escaped infection. Rinderpest outbreaks continually disrupted trade to Egypt from Sudan, which was established in 1904 and involved 37,000 cattle being transported by land and sea in 1918. Although eliminated periodically from certain areas the disease was still endemic in 1961 (Jack 1961).

In Kenya during the First World War, rinderpest was diagnosed in giraffes (*Giraffa camelopardalis*), African buffaloes (*Synceros caffer*), elands (*Taurotragus oryx*), reed buck (*Redunca redunca*) and bush buck (*Tragelaphus scriptus*) at a time when the Masai herders were deliberately introducing rinderpest into their nomadic herds to immunise them. Widespread outbreaks were reported in wildlife in 1934 (Macauley 1973).

Kenya, Ethiopia, Eritrea and Sudan were clearly continuously affected, and were repeatedly seeding rinderpest virus infection into Uganda, Tanzania and what is now Rwanda and Burundi. A notable epizootic occurred in 1920 and persisted in overt form until 1926 on the Kenya/Uganda border. French East Africa (now Rwanda and Burundi) was affected in 1928 (Curasson 1936). This pattern of disease occurrence continued but by the early 1940s, rinderpest rarely moved south of the Tanzanian Central Railway Line – a line that came

to assume talismanic status in the fight against rinderpest in eastern Africa. After extensive vaccination programmes, the Tanzanian veterinary authorities eventually confined rinderpest to the north of the country in a small area around Loliondo, to the north of Ngorongoro, where the last small outbreaks occurred in 1965 and 1966 (Taylor and Watson, 1967, Macadam, 1968).

Thereafter Tanzania enjoyed some 14 years of freedom from the infection. Sadly this situation ended in 1982 when rinderpest was diagnosed in buffaloes in the north of the Serengeti National Park. At the time the origin of this outbreak was not satisfactorily back-traced. For a while the situation in Tanzania was extremely serious as it was found that the virus was extensively distributed across the north of the country, at first in wildlife and later in cattle. After apparent rinderpest freedom of more than a decade, rinderpest had re-emerged in the north-east of Uganda in 1979, rapidly spreading throughout the country and probably entering western Kenya in 1980 (Rossiter et al 1983). This epizootic left in its wake a reservoir of infection, related to that in southern Sudan, which persisted in Karamoja for more than a decade until at least 1994. From there rinderpest regularly entered western Kenya (West Pokot) throughout the 1980s to the mid-1990s by trade and raiding of cattle. At the same time, rinderpest in southern Sudan repeatedly spread until 1998 from the cattle of the Toposa tribe into Turkana in north-west Kenya.

Fortunately emergency vaccination campaigns undertaken in 1983 restored Tanzanian freedom from the disease. For another 14 years this status prevailed but in 1997 northern Tanzania was again infected with rinderpest. However, it is possible to link this to the reappearance in Kenya in 1994. The 1982-83 outbreak in cattle was finally halted by the application of three million doses of rinderpest vaccine in an emergency campaign facilitated by FAO. Thereafter, the European Union supported nationwide mass vaccination campaigns aimed at ensuring the eradication of the virus by the creation of a highly immune cattle population. These campaigns were undertaken in 1985, 1986 and 1987 respectively, in the course of which 23 million doses of vaccine were administered. In 1987, Tanzania became a member of the PARC project and reverted to the earlier strategy of maintaining a belt of immunised cattle in the districts along the border with Kenya. Throughout this period there were no further clinical reports of rinderpest in either domestic cattle or in ultra-susceptible wildlife species. Tanzania had apparently again become rinderpest free although this status was never subjected to the rigorous international scrutiny such a claim would nowadays require. The 1997 epizootic was again eliminated by intensive vaccination (Taylor et al 2002b) with resources mobilised by FAO.

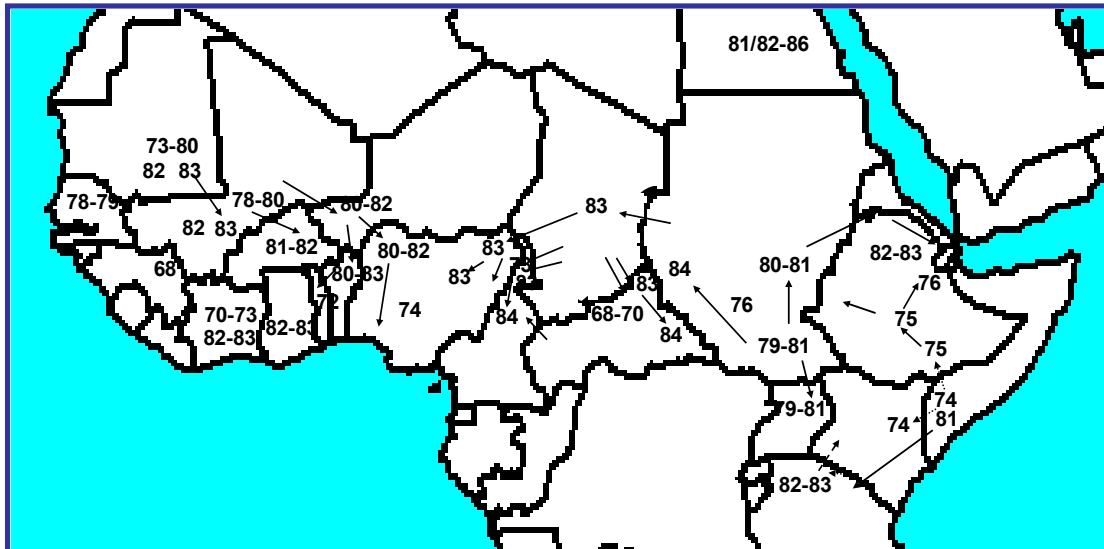
Somalia suffered a severe outbreak in 1928 but rinderpest was brought under a degree of control by 1930, although it was constantly re-introduced from Abyssinia (now Ethiopia) thereafter. From 1939 to 1953 there was no effective control and at that time it was considered to be widespread (Peck 1973). The record of rinderpest in Somalia is particularly meagre but Macfarlane (1970) records that there were 25 outbreaks in Benadir Region (around Mogadishu) in 1969/70. The last cases of classical rinderpest are often stated to have occurred in 1974 when a large outbreak occurred in the south, or else later in

1983. At that time it was also detected in cattle shipped to Oman as reported by the OIE (1983a and 1983b). There is also evidence for periodic episodes of rinderpest of exalted virulence west of the Juba River, peaking in 1981, 1987, 1991-3 and 1996 (Mariner and Roeder 2003).

### **The second African panzootic**

In hindsight, it is easy to see that residual reservoirs of infection left in the Senegal River basin of Mauritania and Mali and in the Greater Horn of Africa would be the source of rinderpest resurgence once disease control efforts waned with the phasing-out of donor support. Serendipity dictated that this should happen when Nigeria's economic strength was creating a high demand for beef. That demand was met by cattle traders supplying from as far away as Sudan, Ethiopia and Mauritania. The inevitable rinderpest re-invasion of countries freed over the previous decades constituted the second Great African Rinderpest Panzootic of the late 1970s and early 1980s, when rinderpest from east and west converged on Nigeria. Weak reporting systems led to a serious under-reporting of rinderpest outbreaks and the emerging problem was not recognised until the panzootic was well established. The livelihoods of livestock herders were devastated and, facing the loss of their herds and destitution, many Fulani herders committed suicide.

The scene had been set 10 years before after JP15. Its success caused complacency which led to the convergence in the early 1980s of two rinderpest epizootics in Nigeria. One came from the west. A study by Woodford et al (1984), together with country studies conducted in preparation for PARC, clearly documents the essentially unreported but widespread circulation of rinderpest derived from the persistent reservoir of infection in Mali and Mauritania. This was not eliminated until it was addressed by the Pan-African Rinderpest Campaign in the mid 1980s (Figure 1). Internal reports of the Malian veterinary service seem to indicate the continuous presence of rinderpest there from 1968 to 1986. The virus responsible for this persisting reservoir and extensions from it, as typified by a virus which entered western Nigeria in 1980 (Shantikumar and Atilola 1990), belonged to African Lineage 2. The other came from the east (Nawathe et al 1993) and belonged to African Lineage 1.



**Figure 1:** Rinderpest occurrence in cattle and wildlife in Africa between JP15 and 1984

Numbers represent the last two digits of the year of occurrence of a rinderpest event placed to indicate the approximate site of the event. Where known, linkages between outbreaks and direction of movement of infection are indicated by arrows. The Ethiopian events are recorded from personal experience and information.

In 1975, JP15 was ending and Ethiopia was generally considered to have been cleared of rinderpest. But reports of giraffe (*Giraffa camelopardalis*) and lesser kudu (*Tragelaphus imberbis*) mortality in southern Ethiopia presaged the later emergence of typical rinderpest in the cattle populations of the Rift Valley and neighbouring Arssi southern highlands of Ethiopia (Figure 1). Initiating control of the developing epizootic was hampered for some months by misdiagnosis as ‘pasteurellosis.’ There was unwillingness to acknowledge the presence of rinderpest (‘which had been eradicated’), exacerbated by poor laboratory diagnostic services. Subsequently, despite concerted efforts to control the disease, it spread slowly but progressively northwards along the Rift Valley and around the central massif of the Ethiopian Highlands. It entered the Afar rangelands in 1976 and spread westwards to cross into Sudan in about 1978. It is salutary to note that virtually all of these events went unrecorded officially and did not feature, in the OIE reporting system. This build-up to the Second African Panzootic was not registered by OIE.

Somewhat surprisingly, rinderpest viruses obtained from eastern Ethiopia in 1994 and 1995 were identified as being of African Lineage 1 (Gopilo and Barrett, personal communication) rather than African Lineage 2, which might have been expected if the virus was derived from the Somali pastoral ecosystem as the observations suggested at the time. Sadly no viruses have survived from the Ethiopian outbreaks in the 1970s. However, the finding is compatible with unpublished reports of rinderpest virus from Sudan sweeping around the northern end of the highland massif of northern Ethiopia to enter

Afar region in the 1980s. Once in Sudan, from 1978 the virus of Ethiopian origin seems to have joined forces with a resurgence of rinderpest from the south of Sudan and it is impossible to tell whether one or other virus dominated in subsequent events. But we know that the virus from Sudan moved steadily across central Africa and on into west Africa, entering the east of Nigeria in 1992, and that it belonged to African Lineage 1.

The Second African Rinderpest Panzootic was a cause of great concern for the national veterinary authorities of affected countries (once faced with escalating cattle mortality), for the international community and for the livestock owners of affected countries. It extended from the Atlantic to the Indian Oceans. FAO provided assistance for many affected countries and worked with the Organisation of African Unity and donors to organise a fresh campaign. By 1987 when the PARC began operations with EU funding, rinderpest distribution had again been reduced greatly by FAO-supported vaccination campaigns. In west Africa, rinderpest was last seen on the border between Ghana and Burkina Faso in 1988, the Mauritania/Mali reservoir having been eliminated several years earlier. PARC was replaced in 1999 by a third internationally coordinated programme, PACE, which retained as an objective, but did not prioritise, the final eradication of rinderpest from the continent. In 2005 the programme was extended by two years.

### **The Demise of African Lineage 1 Rinderpest Virus**

From the limited historical material still available, it appears that African Lineage 1 rinderpest virus has generally been confined to eastern Africa with a distribution stretching from Egypt through Sudan to Ethiopia, Kenya and Uganda. The only evidence of a change in this distribution occurred in the early 1980s when it crossed from Sudan to eastern Nigeria as part of the rinderpest panzootic that engulfed much of sub-Saharan Africa at that time. However, by 1995, African Lineage 1 rinderpest virus maintenance was limited to Ethiopia and Sudan.

In Ethiopia, investigations sustained from 1989 to 1992 established that reservoirs of infection were to be found in the Afar pastoral area of lowland eastern Ethiopia and in an area west of Lake Tana in western Ethiopia. The latter focus had earlier extended to the Dinder National Park in Sudan which abuts the Ethiopian border where rinderpest was present until at least 1972 (Ali 1974). Another focus might have persisted until as late as 1993 in the south of Ethiopia in the lowland area between the highland massif and the Somali border and this could have been the source of the epizootic experienced from 1975.

Later, it became clear that repeated outbreaks in and around the Rift Valley of central Ethiopia and the southern highland massif arose from two sources. The first was via a linkage with the Afar reservoir where virus was transferred by the exchange of draft oxen and heifers – characterised as African Lineage 1 for viruses of 1994 and 1995. The second source for central Ethiopia arose from occasional introduction from the pastoral areas of southern Sudan by trade routes moving cattle towards central Ethiopia. A virus isolated in 1993 was later shown to belong to a different clade within Lineage 1 from that found

in the Afar region. Rinderpest from west of Lake Tana was moved eastwards repeatedly when seasonal cattle migrations brought cattle into the contiguous western highland areas to graze on the aftermath of crop production. On the other side of Ethiopia, rinderpest regularly crossed into the highland massif to the west as draft oxen were sold through markets to highland farmers. These outbreaks tended to be initiated towards the end of the year after harvest, when farmers had cash available. These rinderpest introductions into the highlands resulted in epizootics which advanced slowly but progressively through the highland areas. In 1992 rinderpest spread from market to market for some months after introduction from the Afar reservoir and in 1994 to 1995 it crossed to the west of the highlands and spread northwards, eventually reaching Asmara in Eritrea.

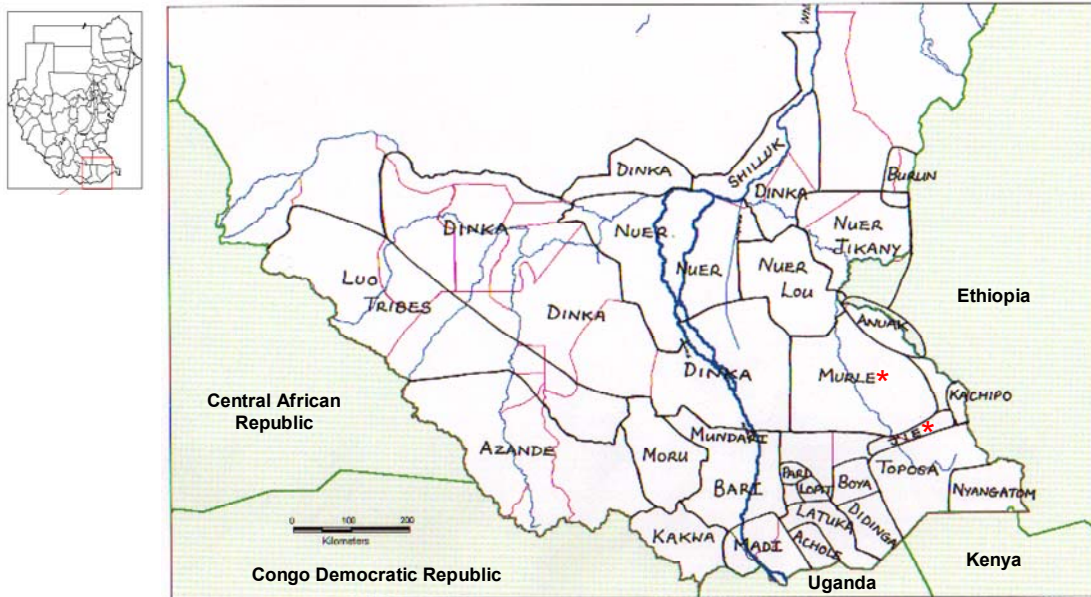
Once this pattern of rinderpest's enzootic persistence and the extension of epizootics from reservoirs was established, this understanding could be used as a basis for developing a science-based eradication strategy. A strong team of veterinarians working with PARC pioneered this work. Setting aside country-wide mass vaccination as unnecessary and unachievable, the new approach sought to contain rinderpest virus within the reservoirs and focus vaccination on eliminating infection from them, establishing minimal vaccination buffer zones to protect especially vulnerable areas and strengthening surveillance and emergency preparedness in event of infection escaping (Roeder, van't Klooster, Asfaw and Gopilo, unpublished papers). Attempting to provide nationwide vaccination coverage would have required vaccinating more than 30 million cattle a year. This had been attempted for many years but even one third of this figure had never been achieved. The new strategy required vaccination of fewer than three million cattle per year. Key to it was the fielding of community-based animal health workers to work in insecure areas of difficult access and the use of a thermostable rinderpest vaccine to achieve high herd immunity rates (Mariner 1996).

After a major struggle for acceptance, the strategy was implemented from 1993 and rapidly proved successful even if it was not without its challenges. An epizootic started by cattle from the Afar focus began in eastern Ethiopia in 1994 and provided a severe test of the strategy. Although the disease seemed at first to be controlled by movement control, including market closure enforced by the army, combined with focal vaccination, it crossed to the western side of the highlands by illicit trade movements and even moved north from there to Asmara in Eritrea, where it was rapidly extinguished. In all there were approximately 50 outbreaks before the status quo was restored. Resumption of mass vaccination was narrowly avoided. By the end of 1995, rinderpest had been eliminated and has not returned. The last cases were reported in October 1995 in the west of Ethiopia (originating in southern Sudan) and in November 1995 in northeastern Ethiopia, being the last remnant of the Afar reservoir. Both belonged to African Lineage 1.

The final stage in the demise of African Lineage 1 rinderpest virus, which required elimination from its last stronghold in southern Sudan, can be seen to date from 2001. For more than a decade, rinderpest in Sudan had essentially been contained within the extensive pastoral communities of the south.

Classical severe rinderpest of African Lineage 1 had last been confirmed in 1998 by the FAO World Reference Laboratory for Rinderpest from samples collected in the extreme south of Sudan. These originated from an outbreak in sedentary cattle belonging to a community of agro-pastoralists living in Torit County of Eastern Equatoria Region, to which it was probably introduced by cattle traded from the neighbouring Toposa tribe. Formerly a constant and serious threat to the livelihoods of the southern Sudanese pastoralists and the neighbouring Turkana tribes of Kenya, and feared by cattle owners throughout the country to the north, rinderpest was progressively brought under control. This was achieved by an animal health programme which combined conventional veterinary services with innovative community-based approaches to animal health service delivery in remote areas which had been marginalised from conventional animal health service delivery (Leyland 1996; Mariner 1996). The coordinated approach through the United Nations Operation Lifeline Sudan (OLS) Livestock Program, with the involvement of many Non-Governmental Organisations (NGOs), was so successful that many of the formerly severely affected pastoral communities were freed from the disease progressively from the early 1990s. By 2000, surveillance had confirmed that suspicions of the persistence of rinderpest infection were limited to very few areas in the extreme southeast of the country.

Towards the end of 2000, persisting into 2001, reports started to be received of mortality in the cattle herded by the Murle nomadic pastoralists in the vicinity of Gumuruk in Pibor County of Jongeli State, Upper Nile Region in southern Sudan. They identified the disease as rinderpest and clamoured for vaccination for their cattle. Timely investigations were difficult to mount but despite the remoteness and insecurity of the affected area, Sudan Government veterinarians supported by the FAO OLS Livestock Programme eventually reached the affected area. It was estimated that approximately 200 cattle had died. However, it was not possible to access actively infected herds and laboratory confirmation of rinderpest was not achieved. Serological investigations were compromised by the start of emergency rinderpest vaccination. In total the Murle and closely associated Jie communities herded some 800,000 cattle with 700,000 of these belonging to the Murle. These were essentially unvaccinated populations. At that time, unlike in earlier years, there was no evidence or suspicion that rinderpest was occurring in the cattle herds of any of the other major livestock-dependent ethnic groups (Dinka, Nuer, Anuak, Toposa etc.) in southern Sudan. Nor was there real cause for suspicion elsewhere in the country further north, although there were several areas in the south where access was denied by severe civil insecurity. Earlier studies in 2000 and 2001 assisted by FAO's Technical Cooperation Programme had confirmed the absence of rinderpest in recent years in cattle in the central sector of the country, or undertaking annual, seasonal migrations into and from the southern zone. Combined with disease intelligence gathering led by the NGO *Vétérinaires sans Frontières* Belgium, responsible for implementing the PACE Sudan (south) project entitled 'The fight against African Lineage 1 Rinderpest', an educated guess was that the cattle herds of the Murle and, possibly, Jie communities were the last harbouring the virus in Sudan (Figure 2).



**Figure 2** Sketch map illustrating the approximate distribution of ethnic groups in southern Sudan used for planning at the time of the outbreak. The Murle and Jie communities referred to in the text are indicated by red asterisks.

It was considered pragmatic to act on this assumption and an intensive focused vaccination programme was initiated by FAO for the Sudanese government under the leadership of the District Veterinary Officer based in Pibor, who had detected the problem originally. The campaign was supported by FAO, which had recently assumed operational responsibility for the OLS Livestock Programme, with funding from the European Commission Humanitarian Organisation and the United States Office for Foreign Disease Assistance. It was highly successful, receiving enthusiastic compliance by the livestock herders who had been seeking rinderpest vaccination. Approximately 650,000 cattle were vaccinated with quality-assured thermostable rinderpest vaccine, mainly between March and December 2001. The programme was extended until June 2002 for the livestock populations belonging to the Murle and Jie peoples as they were considered to be at highest risk of rinderpest maintenance. Another 120,000 cattle of the adjacent Toposa people were vaccinated because of their close interaction with the Jie. This was achieved by conventional and community-based animal health workers despite an ongoing civil war and the rainy season (June to October), which floods extensive areas of land and makes transport difficult. Vaccination ceased outside the extreme southeast of Sudan, west of the Nile, by December 2001 and everywhere in June 2002. At this time, all rinderpest vaccine was withdrawn from the field and all unissued stocks were sequestered in vaccine banks in Khartoum and Lökkichoggio (in Kenya) for the northern and southern sectors of OLS respectively.

The hypothesis that rinderpest was not present elsewhere in the ecosystem proved to be correct. Investigations confirmed that herds in Pibor

County had instead experienced outbreaks of a cattle disease variously known as *batiboy* and *achoke*. Since that time rinderpest surveillance has been strengthened considerably, using all available techniques and sometimes seizing fleeting ceasefire opportunities to reach areas such as the Nuba mountains and the Sobat Basin, which are usually inaccessible. All evidence confirms that rinderpest was eliminated from this reservoir in 2001.

The final thrust for the elimination of rinderpest from this ecosystem had taken off from a sound platform of earlier progressive rinderpest control together with demographic and disease information built up over the previous decade by NGOs working under the OLS umbrella. Coordination was supported administratively and financially by UNICEF<sup>5</sup> with OFDA, EU and other donors and with technical support from Tufts University (Leyland 1996). This programme had seen the geographical extent and incidence of rinderpest fall progressively to the point where a final eradication effort was feasible. Key to the latter stages of elimination of rinderpest from this ecosystem was the '*The fight against African Lineage 1 Rinderpest*' project implemented by Vétérinaires Sans Frontières, Belgium, within the EU-funded PACE programme, coordinated closely with the work of the northern Sudan element of PACE-Sudan and FAO OLS staff in both northern and southern sectors. In addition to providing clinical services to marginalised and war-torn pastoral communities, the coordinated activities sustain a disease information system capable of detecting rinderpest, and reporting and investigating possible rinderpest.

In 2002, 2003 and the first five months of 2004, approximately 99, 101 and 22<sup>6</sup> epidemiologically significant disease events were reported respectively. Of these 24, 21 and 5 respectively specifically raised a suspicion of rinderpest. Follow-up participatory disease searching<sup>7</sup> (PDS) (Mariner and Roeder 1997) by northern and southern sectors of PACE Sudan, combined with investigation of epidemiologically-significant events raising suspicions of rinderpest, have all failed to detect evidence of rinderpest. In many cases outbreaks could be dismissed as not being rinderpest on clinical and epidemiological appearance. Others were confirmed as being caused by a range of enzootic diseases including foot-and-mouth disease, trypanosomiasis, contagious bovine pleuropneumonia and theileriosis. In 2004 two particularly alarming reports of mortality of more than 50 cattle were confirmed by the WRL-RP to be malignant catarrhal fever by PCR<sup>8</sup>. None of the investigations detected rinderpest nor has there been any reason to suspect the persistence of the virus in this ecosystem, which extends into the south-western lowland areas of Ethiopia, northeastern Uganda (Karamoja) and northwestern Kenya (Turkana and West Pokot). Increasingly supported by PDS programmes which record a past history of rinderpest up to 2001, but not subsequently, and complementing the disease reporting and investigation

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<sup>5</sup> United Nations Children's Fund

<sup>6</sup> Reports peak during the rainy season from June onwards, accounting for the low rate early in the year.

<sup>7</sup> An epidemiological technique based on participatory rural appraisal techniques

<sup>8</sup> Polymerase chain reaction

system operated by the governments of Sudan, Ethiopia, Uganda and Kenya, these findings increase our confidence that the reservoir of infection no longer exists and that African Lineage 1 rinderpest virus is extinct.

### **African Lineage 2 Rinderpest virus**

The evidence suggests that African rinderpest virus Lineage 2 was much more widely distributed than Lineage 1, taking in both western and eastern Africa. It was isolated in Nigeria in 1958 and in Kenya in 1962. When rinderpest re-emerged in west Africa in the early 1980s, the virus that spread eastwards from Mauritania/Mali (where it had been persisting for many years) to western Nigeria belonged to African Lineage 2. When this was demonstrated in the 1990s, it appeared as if west Africa had been the only source of this lineage. However, we now know that its distribution had not altered. Instead the virulence of the eastern African representative of the lineage had become so low as to make it clinically difficult to detect, thereby allowing it to escape detection for more than 30 years.

A severe outbreak of rinderpest occurred in 1994 and 1995 in the lesser kudu, eland and African buffalo populations of Tsavo National Park in southern Kenya (Barrett 1998; Kock et al 1999). Some 60 per cent of the buffaloes were killed. It is also now recognised by wildlife veterinarians and zoologists that the northern Kenya population of lesser kudus fell by up to 90 per cent during the early 1990s, most likely as a result of rinderpest. After more than a year in which the virus seemed to have disappeared, another event occurred in the neighbouring Nairobi National Park and in the Amboseli National Park to the south, close to the border with Tanzania. In addition serological and epidemiological evidence indicates that the virus also penetrated Meru National Park and in some other wildlife populations in the east of Kenya around that time. In the Tsavo outbreak, the virus exhibited a high level of virulence in buffaloes and kudus. In the Nairobi outbreak, buffaloes and eland were similarly affected with mild disease in young domestic cattle. When isolates from Tsavo National Park were examined by nucleotide sequencing they were shown to belong to African Lineage 2, as did other viruses from Nairobi National Park. This was surprising because no representative of Lineage 2 had been unearthed in eastern Africa since 1962. Furthermore, while it was now recognised that at an earlier date Lineage 2 had been distributed across the continent, the last time its presence had been detected was in Sokoto, Nigeria, in the early 1980s. Thus, the unexpected reappearance of this lineage of virus in Kenya in 1994 could not be explained in terms of spread from another well-known focus.

Although representatives of Lineage 2 were isolated in Kenya and Tanzania in 1961-1962, the records of the Kenyan and Tanzanian Veterinary Services appear to show the demise of rinderpest in the Masai ecosystem of these two countries around 1966 even though rinderpest persisted further to the north (Atang and Plowright 1969). The picture of rinderpest in northern Tanzania is one of shrinking distribution and fluctuating virulence. It suggests that the northern Tanzanian/southern Kenya focus from which a number of mild strains were isolated in 1962 was a relict population of African Lineage 2 rinderpest virus derived from the enzootic situation that had existed in east

Africa for over 60 years. From the 1930s, outbreaks associated with a low level of field virulence were consistently observed within this enzootic area. In 1959, Robson et al (1959) described the isolation of a naturally cattle-attenuated strain of rinderpest virus from an eland in northern Tanzania which retained virulence for certain wildlife species. Unfortunately this strain seems to have been lost and its phylogenetic lineage cannot be determined.

In Tanzania it seems that rinderpest was capable of making a silent entry into cattle herds, and increasing in virulence gradually, to the point of producing classic disease signs after the herd had been infected for 3 - 6 weeks. The virus was capable of transmitting to new herds in its virulent form. On at least one occasion no such increase of virulence for cattle was observed. There is also evidence that the avirulent variant was in fact virulent for game animals. This accords with the belief that these animals were responsible for the introduction of mild rinderpest into uninfected herds of cattle (Branagan and Hammond 1965).

It has been demonstrated that the two best-characterised Lineage 2 viruses isolated in east Africa in 1962 have completely differing clinical effects in cattle. The Kenyan virus, RGK/1, isolated from a sick giraffe near Garissa in northern Kenya, has a high level of virulence for cattle, causing severe clinical signs and the death of 60-70 per cent of experimental cases. It has retained this characteristic on passage in cattle and cell culture for many years. By contrast, its contemporaries from northern Tanzania and southern Kenya (of which the RBT/1 and 2 strains are the only surviving representatives) were very mild in their effects on village cattle. Under controlled laboratory conditions a number of RBT1 infected animals failed to develop mouth lesions and could not have been clinically diagnosed as having rinderpest, even by experienced observers.

When strain RBT/1 was inoculated into cattle at low dilutions, the virus was virulent, but when high dilutions were used it was avirulent. This has been taken to suggest the possible presence of at least two variants in the stock preparation. It was not possible to increase the virulence of another RBT/1-like virus (obtained from a sick buffalo) by back-passaging it in cattle, and it remained mild in cattle. Perhaps then, the low virulence RBT strains from Tanzanian cattle in 1962 represented viruses that had been selected for their long-term survivability under east African conditions. Northern Tanzania supplies us with evidence that such persistence did in fact happen and that an unsuspected rinderpest focus persisted in the Sonjo valley of Loliondo District, northern Tanzania from 1961 to 1965. Even after its discovery in 1965, the virus managed to remain at large until 1966. Further, evidence is recorded compatible with a residual rinderpest virus infection disclosed by disease in the buffalo (1964 - 1967) and warthog (1964 - 1968) populations of the Serengeti National Park. This pocket of infection was not recorded clinically in cattle in the area. Disease and infection subsequently died out in the buffaloes.

After 1968, rinderpest was not recognised again in east Africa until 1980 when it seems to have entered northern Tanzania from eastern Kenya. Unfortunately, the virus involved was never isolated or typed and it is not

possible to draw any firm conclusions about its lineage. The virus appeared to possess a high level of virulence for both cattle and game. It ultimately spread throughout the cattle of the Masai ecosystem of northern Tanzania and also involved the buffalo populations of Tarangire and Serengeti National Parks and the Ngorongoro Conservation Area. At the end of the outbreak in 1982, the virulence of the virus appeared to decline and it finally died out. However, retrospective analysis discloses that the severe clinical events of 1982 were preceded in northern Tanzania by 'mild' rinderpest which was not recognised as such (Nyange *et al.* 1985). Thereafter, there were no recorded outbreaks of rinderpest in either Kenya or Tanzania until the Tsavo outbreak of 1994, although outbreaks were certainly occurring. They were particularly associated with the cattle trade from north-eastern Kenya to settled cattle ranches. These viruses associated with the 1980s rinderpest incidents in Tanzania and Kenya seem to have belonged to African Lineage 2. This implies the existence of a cryptic rinderpest focus over a period of nearly 30 years somewhere in eastern Africa. The mild form of rinderpest that had persisted in Loliondo District up to 1965/66 is unlikely to have been unique.

The virulence for cattle of the re-isolated representative of Lineage 2 (Nairobi eland 1996) is not dissimilar to that of one of the RBT/1-like suite of viruses which was virulent for buffaloes but mild for cattle. This lack of cattle pathogenicity would account for the apparent ability of the virus to move between Tsavo and Nairobi National Parks and southwards to Amboseli National Park, presumably by transmission among cattle. Clinical disease in cattle in Kajiado (in the southern Rift Valley of Kenya) was confirmed as rinderpest before this identification was officially denied and later admitted. In experimental studies carried out by the World Reference Laboratory for Rinderpest, of ten cattle experimentally infected with the Kenya/Eland/96 strain, two showed no clinical signs of disease yet seroconverted, three had pyrexia, three developed mild transient oral erosions and two died with classical acute rinderpest (Anderson and Anderson personal communication). Clinical observations during the related outbreak in the field in northern Tanzania and Kenya in early 1997 mirrored the experimental evidence. Against a background of mild to subclinical disease, typical severe rinderpest was observed in a small number of herds. Similarly, clinical rinderpest was also seen in cattle in southern Kenya at the time, as it had been at the time of the outbreak in Nairobi National Park in 1996 (Rossiter 2001). Masai cattle raisers reported high losses of cattle in the previous year from what they recognised to be rinderpest. Clinical rinderpest was in fact confirmed as rinderpest in the southern Rift Valley of Kenya in Kenya in 1997.

Serology provides support for the understanding that infection was much more widespread than was clinical disease recognisable as rinderpest, at least in northern Tanzania where, again unfortunately, the lineage of the infecting virus was not determined. Within this evidence then, we can see both the widespread sub-clinical spread of this virus in cattle and elements of a selection pressure tending towards the recovery of a lethal potential.

Somali veterinarians describe experiencing a problem for many years of 'bovine viral diarrhoea' in young cattle but their reports do not conform to the

known epidemiology of that disease. These and other anecdotal reports of infectious bovine rhinotracheitis together with other signs suggestive of mild rinderpest in southern Somalia could well indicate the persistence of enzootic mild rinderpest. They cover the periods from the 1970s to the 1980s and come from expatriate veterinarians. The detection of atypical mild rinderpest in northern Kenya in 1996, accompanied by clinical disease in calves about four to 12 months of age during a drought, and linked epidemiologically to reports of similar disease in southern Somalia, gives some clue as to the most recent location of African Lineage 2 virus (Mariner and Roeder 2003). The redistribution of infection from this focus was perhaps anticipated by Plowright (1982) who suggested that: 'rinderpest virus periodically invades Kenya (and Uganda) from the north and it is not difficult to imagine how infection could spread across the Tana and Galana Rivers down the coast and into the Tsavo, Amboseli or Kajiado Districts' - and, one could add, extending into northern Tanzania as occurred in the 1980 and 1996/7 epizootics.

### **The Enigma of Mild Rinderpest**

When rinderpest virus of African Lineage 2 was recognised as the cause of an epizootic in buffaloes in Tsavo National Park of Kenya in 1994, it was at first suspected that the source of the infection had been Sudan. This was logical because southern Sudan was known to constitute a reservoir of African lineage 1 rinderpest and the virus had repeatedly entered the Turkana region of Kenya from southern Sudan in the 1980s and 1990s and spread to central Kenya around Nairobi in 1985/86 (Wamwayi *et al.* 1992). However, once virus characterisation results were made available by the FAO World Reference Laboratory for Rinderpest, identifying the virus as lineage 2, it became clear that the source had to be sought elsewhere. There were many reasons to suggest that the Somali ecosystem (essentially an area west of the Juba River and spanning the Kenya/Somalia border) could at that time have constituted a reservoir of rinderpest virus infection. Studies conducted in 1996 added weight to this belief (Mariner and Roeder, 2003). Local stock owners maintained a verbal record of the presence of rinderpest within the ecosystem over several decades, and recognised fluctuations in its innate virulence. This focus may represent the last bastion of rinderpest on Earth. Defining and extinguishing it are the last two questions standing in the way of global rinderpest eradication.

The results of extensive serosurveillance studies conducted under the PACE Somalia programme in central and southern Somalia in 2001 to 2003 purport to demonstrate the continuing presence of rinderpest there. Unfortunately the results have not been made available for scrutiny by others. Working in Somalia, and especially southern Somalia, has been particularly problematic for nearly two decades. However, the team involved also conducted surveys for clinical disease and were able to detect a syndrome claimed to be compatible with mild rinderpest, although laboratory examinations were not confirmative. Outbreaks of rinderpest in wildlife (primarily buffaloes) had been repeatedly observed in Kenya before and after the Tsavo epizootic of 1993-5. In 2003 a Kenyan epizootiologist detected foci of disease in cattle which were compatible with a presumptive diagnosis of mild rinderpest (Chibeu personal

communication). Despite the clinical findings, and some suggestive serological results - where these have not been compromised by sporadic vaccination - it has not proved possible to link the 'mild rinderpest' syndrome detected in the field with rinderpest virus infection by laboratory examination. Most recently, in 2004, an incident caused alarm in southern Ethiopia, reliably free from rinderpest for more than 25 years. Acting on information provided by the Kenyan Veterinary Department that a 'mild rinderpest' compatible syndrome had been detected in northern Kenya close to the junction of the borders of Kenya, Ethiopia and Somalia, the Ethiopian Veterinary Team undertook a PDS investigation in contiguous areas of Ethiopia. A similar syndrome was detected in a small area in Ethiopia. Intensive laboratory investigations for virus and antibody backed up by the World Reference Laboratory for Rinderpest showed that the symptoms had not been caused by rinderpest.

Another complication is the fact that PCR products and some tissue extracts submitted to the World Reference Laboratory for Rinderpest from the East African Regional Rinderpest Reference Laboratory contained nucleic acid which when sequenced indicated that the virus was identical to the Kabeete O vaccine/standard challenge strain. Normally one would dismiss this as evidence of laboratory contamination, but for the fact that on several other occasions investigations in east Africa have given the same result. These include cases identified clinically as mild rinderpest in southern Kenya and northern Tanzania in 1997. It is difficult to conceive how - other than by escape from laboratories or reversion to virulence of vaccine - could rinderpest virus belonging to this ancient clade really be circulating today in Africa? Faulty laboratory testing is more likely as the use of PCR is beset by such problems in developing countries.

Rinderpest virus (in this case African Lineage 2) was last detected in tissues sent to the FAO World Reference Laboratory for Rinderpest from buffaloes in Meru National Park in Kenya in 2001. No subsequent investigations of suspicious events in Kenya have confirmed the presence of rinderpest virus. What then causes the 'mild rinderpest-compatible syndrome' that has been detected repeatedly in Kenya and Somalia and recently in Ethiopia? On historical grounds it has every right to be rinderpest, but why do laboratory examinations, which have proved so effective elsewhere, fail to give the desired result? Hence the enigma, against which background future events and proposed actions in eastern Africa need to be assessed. We cannot say whether the cryptic Somali Lineage 2 rinderpest focus is still extant. This question needs to be answered by field and laboratory investigations before implementing more mass vaccination programmes. Or are there grounds for believing that rinderpest could actually have been eradicated already?

Assuming the Somali livestock ecosystem maintains the only cryptic form of African Lineage 2 rinderpest virus, the determinants of the disease have not yet been defined nor has its relationship to what is ostensibly a syndrome of mild rinderpest in cattle been confirmed. Despite the excellent and highly informative wildlife surveillance work conducted in eastern Africa under the auspices of PARC and PACE, debate continues, sometimes inadequately

informed by an understanding of what has gone before. There is a tendency to equate mild rinderpest with African Lineage 2 rinderpest virus, and to associate this in turn with rinderpest maintenance in wildlife in east Africa. However, mild rinderpest has not been restricted to eastern Africa, nor even to Africa, nor does it relate only to the African lineage 2 rinderpest virus - which itself has not been restricted to eastern Africa. Despite this, the assumptions are used to make pessimistic predictions about the feasibility of rinderpest eradication.

One widely held belief concerning African Lineage 2 rinderpest virus in eastern Africa is that it is uniformly virulent for wildlife whilst being mild for cattle. However, there are numerous indications from earlier years that such viruses are not always uniformly virulent for wildlife (see for example Plowright 1963; Branagan and Hammond 1965). A strain belonging to this clade that was highly virulent for cattle was isolated in 1962 from a giraffe in Kenya. There is a suggestion that the rinderpest in some of the last wildlife incidents in Kenya has been less than fully virulent (Kock and Thomson, personal communications). Robson et al (1959) stated that: 'The natural selection in wild game of mutants attenuated for cattle has been postulated but the hypothesis is unsupported by the presented evidence.'

When it comes to the dilemma of rinderpest maintenance by wildlife, little progress is evident since Plowright (1963) indicated that, 'The arguments crystallised into the expression of two apparently irreconcilable points of view, neither based on sound evidence, much less quantitative data.' One view, the consensus of informed veterinary opinion at the time, was that rinderpest would not be eliminated from cattle because they co-existed with large concentrations of highly susceptible wild animals. The other camp supported the view that if rinderpest could be eliminated from cattle it would die out completely in wildlife because it rapidly burnt itself out in the wild. Plowright (1963) himself was rather pessimistic at that time about the prospects of eradication. He referred to the importance of large accumulations of highly susceptible wild animal species in parts of east Africa in which Reid (1949) described 'a reservoir of smouldering rinderpest', although at the same time the latter author indicated that the disease died out in less dense wildlife communities in central and southern Tanganyika (now Tanzania) and also in large areas of Kenya and Uganda. In the current circumstances, based on studies of rinderpest epizootics in wildlife in eastern Africa in the last decade, Kock (personal communication) suggests an upper limit of four years for virus circulation in wildlife, even for lesser kudu over which there hangs a suspicion of virus maintenance.

Rinderpest was enzootically established at least by the early 1930s in northern Tanzania and was continuously enzootic subsequently. Yet seroconversion in the wild animals of the Serengeti ecosystem reduced progressively from 1959 and was not detected after 1967 when immunisation campaigns for rinderpest in cattle were intensified. This caused Plowright (1982) to conclude that, 'By themselves, the large numbers of susceptible game animals in the Serengeti Region in 1962 to 1963 were not capable of maintaining the strains of virus current at that time.' Apart from incursions from

Kenya in the early 1980s and late 1990s Tanzania has stayed free from that time.

Nowhere today are there comparable dense wildlife populations, with the exception of the Serengeti/Mara herds, which are undoubtedly free from rinderpest. The situation today has changed and the likelihood that rinderpest could 'smoulder' continuously for many years must be considered very low. However, this is not to say that rinderpest cannot 'burn' through a relatively large wildlife population for an extended period of time as it clearly did in the buffalo and eland herds of the Tsavo National Park in Kenya in 1993-1995. Then it acted as a conduit for infection between two widely separated cattle populations to the east and west of the National Park.

Of course one has to take into account the genetic factors of the host in determining the outcome of virus/host interactions. But mild rinderpest has been regularly encountered within and outside Africa and with all three lineages of rinderpest virus. For African Lineage 2 the evidence is briefly described above. For African Lineage 1 it was recorded in Kenya in the mid 1980s (Wamwayi et al 1992) and in Egypt. In Egypt mild rinderpest was recognised in the 1980s but could have dated back to the early years of the 20<sup>th</sup> Century. It could be related to cattle imported from Iraq, notorious as 'Baghdadlis' (Littlewood 1905). Incidents of mild rinderpest were also a feature of rinderpest in the then 'Indo-China' (now referred to generally as Southeast Asia) in the 1950s involving indigenous cattle, buffaloes, swine and wild bovid species.

So what can one conclude? If it is unlikely that the virus within the eastern African pastoral ecosystem will die out spontaneously in cattle, its hidden presence poses a grave risk for the rest of the continent and eventually the world. As clinical reporting seems unable to define its distribution, planning and implementing vaccination and zoosanitary procedures to remove rinderpest from the Somali pastoralists' herds poses a major problem. The issue is one of finding the management skills and the means to define its geographic distribution, and to undertake the concerted action which will be necessary.

Is mildness the result of a chance mutation of a rinderpest virus? Or is it possible that mildness is an attribute of certain viruses which is induced by selecting strains for dominance from within a quasi-species swarm (Smith et al 1997) of rinderpest virus after an outbreak? The latter would be compatible with field observations that virulence is not a fixed attribute of rinderpest viruses and can evolve in either direction. Reversion to full virulence is perhaps to be expected should movement into suitable susceptible populations occur. Pragmatically, the latter assumption has been adopted by GREP. Fortunately, the history of southern Africa, eastern Africa and Southeast Asia in the 20<sup>th</sup> Century suggests that the circulation of rinderpest between domesticated livestock and a susceptible wildlife population does not preclude area-wide elimination of the virus.

## **Prospects for the ultimate success of GREP in Africa**

There is growing confidence that west and central Africa has been free from rinderpest since the last cases occurred on the Burkina Faso/Ghana border in 1988. There is no suspicion of a persisting reservoir of rinderpest virus there. The OIE accreditation process is providing confidence in the fact that west and central African countries have been free from rinderpest since then. Rinderpest virus of African Lineage 1 persisted in Ethiopia until 1995 and in Sudan until 2001. In both countries there is convincing evidence for the absence of virus circulation since then. These were the last strongholds of African Lineage 1 rinderpest virus, which has almost certainly joined the Asian Lineage in being consigned to history in the wild.

Having resolved the last focus of rinderpest in east Africa to the level of the Somali pastoral ecosystem where it may - or may not - still persist, it is only the resolution of this critical issue that prevents us from entertaining the notion that rinderpest has been eradicated from Africa, and the world. There is, however, every cause for concern. There exists a significant body of experience recording the recent presence in Kenya and Somalia of a disease syndrome in cattle compatible with the concept of 'mild rinderpest' and close to the area in which 'mild' rinderpest virus strains were described in the 1950s and 1960s and rediscovered in the 1990s. The last definitive detection of rinderpest virus was in African buffaloes (*Syncerus caffer*) of Meru National Park adjacent to the Somali pastoral ecosystem in eastern Kenya in 2001. This was unequivocally identified as virus of African Lineage 2 by the FAO World Reference Laboratory for Rinderpest. Yet, all subsequent investigations of what could be a mild form of rinderpest in cattle in Kenya and Somalia (and most recently in a contiguous area of southern Ethiopia in 2004) have failed to provide clear evidence of rinderpest virus presence whether by virus detection or serology. Even wildlife serosurveillance, a valuable indicator of rinderpest virus circulation, has suggested that seroconversion of wildlife sentinels is no longer occurring in the high-risk area of eastern Kenya (Kock personal communication). The current situation is an ongoing international problem of considerable magnitude. Failure to resolve it threatens to compromise the ultimate success of GREP.

If the situation in the Somali pastoral ecosystem is not resolved, and quickly at that, rinderpest may no longer remain a disease of the past. A dramatic and devastating resurgence is all too possible.

## **Risks resulting from rinderpest persisting in Africa**

Having established how rinderpest evolved and survived in Africa since its introduction to the continent, let us examine the risks which rinderpest presents today, the drivers of those risks and the possible outcomes.

## **(A) Risk: A panzootic resurgence of rinderpest**

Two separate drivers of a panzootic rinderpest resurgence in Africa are identified below. Their 'Sources', 'Pathways', 'Hazards' and 'Outcomes' are largely similar.

**DRIVER 1:** The driving force for rinderpest resurgence would be that the continuing presence of rinderpest within the Somali pastoral ecosystem is not resolved by IBAR/PACE and directly run EU programmes, neither containing rinderpest virus within the ecosystem nor eliminating it.

Subcomponents of this driver which could contribute to driving the risk of panzootic resurgence are:

- Lack of surveillance capacity in countries renders them incapable of detecting rinderpest in a timely manner.
- Complacency on the part of national authorities in countries believed to be free, and IBAR and the international community, leads to failure to take seriously the first signs of an impending panzootic.
- Emergency preparedness planning is inadequate at national and continental level and money is not available fast enough to take appropriate action at an early stage.
- Concealment by national authorities of the initial stages of the evolving epizootic to protect trade, and attempts at clandestine control, lead to a failure to contain infection.

Herd immunity levels to rinderpest are low to zero in all countries of sub-Saharan Africa because vaccination has ceased. This is an essential stage of vulnerability thought to be necessary to disclose occult foci of infection and is therefore a pre-requisite for entry onto the OIE Pathway for accreditation of rinderpest freedom.

**Sources:** A persisting reservoir of endemic rinderpest within the Somali pastoral ecosystem

**Pathways:** A mild strain of rinderpest could spread undetected for some time through pastoral migrations and trade before being detected, and then evolve to re-assume full virulence

- Rapidly growing demand for cattle in Ethiopia to meet the demands of the lucrative meat and live animal export market to Egypt which opened up in 2005 will draw cattle into southern Ethiopia from southern Somalia where rinderpest could still be present.
- The nascent regrowth of Nigeria's economy could, if it continues, once again create a demand for cattle over virtually the whole of sub-Saharan Africa and an inflow of cattle from distant countries, providing a potent stimulus for rinderpest spread. Cross-African trade to supply Nigeria in the 1980s drew in cattle from as far away as Mauritania and Ethiopia.

- A severe drought in eastern Africa could recapitulate the events of 1992-3 that caused unusual, major migrations of pastoralists' cattle herds to invade national parks. Rinderpest found a fertile substrate for its rapid transmission in the high density of cattle resulting around water points and areas of available grazing. Floods can create similar conditions. Rinderpest can greatly increase the effects of these natural disasters that are relatively common in the Somali pastoral ecosystem.
- Conflict such as the invasion of Somalia by a foreign force could displace people and livestock to favour rinderpest resurgence.
- Initial spread through local marketing systems – farmers who realise they have rinderpest panic-sell incubating cattle into local markets.
- Panic on the part of livestock rearers – in the face of a severe outbreak, pastoralists may flee with their herds over long distances, thus disseminating infection widely and rapidly.
- The most likely route would be from Somalia (Transjuba)/Kenya into the Ethiopian Somali Regional State to Oromiya, spreading along into the Afar Regional state to the east of the central highland massif.

**Hazard:** That rinderpest will re-emerge from the Somali pastoral ecosystem and give rise to an epizootic that evades control to spread widely, reverting to virulence and evolving into the third sub-Saharan panzootic.

**Likelihood:** Medium to high, increasing with time

**Outcomes:** Rinderpest resurgence would have devastating effects on the livelihoods of pastoral communities in sub-Saharan Africa and those whose livelihoods depend on livestock. There would be immediate direct effects on livelihoods exacerbated by indirect and longer-term effects. National economies of rinderpest-free countries would be affected by the impact on livestock trade.

Elements contributing to the outcomes are:

- Devastation of livestock-based agriculture, leading to shortages of both crops and foods of cattle origin – pastoralists, agropastoralists and crop producers would all be affected.
- Food insecurity and even famine, especially in pastoral communities at first.
- Destruction of trade. Trade bans imposed by trading partners within Africa and outside would further damage rural livelihoods and the developing meat and animal trade, for example in Ethiopia, Sudan and Somalia.
- Death of wildlife and even extinction of some relict populations, leading to loss of biodiversity and damage to viability of national parks with long-term effects on tourism.
- Destruction of developing improved farming systems such as per-urban dairying, loss of indigenous and improved genetic material.

**DRIVER 2:** Should the GREP Secretariat be mistaken in its understanding that there is no risk of persisting rinderpest reservoirs outside the Somali pastoral ecosystem, such reservoirs could lead to a rinderpest resurgence.

Subcomponents of this driver that could contribute to driving the risk of pandemic resurgence are:

- Surveillance programmes promoted by IBAR PACE could have failed to detect endemic foci.
- Lack of surveillance capacity leaves countries incapable of detecting rinderpest occurrence in a timely manner.
- Complacency on the part of national authorities in countries believed to be free, and in IBAR and the international community, leads to failure to take seriously the first signs of an impending panzootic.
- Emergency preparedness planning is inadequate at national and African level, and money does not come through fast enough for appropriate action at an early stage.
- Concealment by national authorities of the initial stages of the epizootic to protect trade, and attempts at clandestine control, lead to a failure to contain infection.
- Herd immunity levels to rinderpest are low to zero in all countries of sub-Saharan Africa because vaccination has ended. This is an essential stage of vulnerability thought to be necessary to disclose occult foci of infection and is therefore a pre-requisite for entry onto the OIE Pathway for accreditation of rinderpest freedom.

**Sources:** Occult reservoir(s) of rinderpest endemicity other than that suspected to be present in the Somali pastoral ecosystem.

**Pathways:** Infection could escape from an unknown reservoir by the following pathways:

- The nascent regrowth of Nigeria's economy could create new demand for cattle over virtually the whole of sub-Saharan Africa and lead to an inflow of cattle from distant countries, providing a potent stimulus for rinderpest spread. African trade to supply Nigeria in the 1980s drew in cattle from as far away as Mauritania and Ethiopia.
- Conflict and civil strife could displace human populations and their livestock, to favour rinderpest resurgence.
- Initial spread through local trade – farmers who realise they have rinderpest panic-sell incubating cattle into local markets.
- Panic on the part of livestock rearers – in the face of a severe outbreak, pastoralists may flee with their herds over long distances, disseminating infection.

**Hazards:**

- Rinderpest starts to spread from the occult focus and evolves into a national epizootic, which evolves rapidly to panzootic proportions.
- National authorities and IBAR PACE fail to rapidly implement science-based control programmes and fail to contain the epizootic.
- After the end of the PACE programme in 2007, there will be no organisation able to coordinate and fund the necessary actions.

**Likelihood:** Low

**Outcomes:**

- Devastation of livestock-based agriculture, leading to shortages of both crops and foods of cattle origin – pastoralists, agropastoralists and crop producers would all be affected
- Food insecurity and famine.
- Destruction of trade. Trade bans imposed by trading partners within Africa and outside would further damage rural livelihoods and the developing meat and animal trade, for example in Ethiopia, Sudan and Somalia.
- Death of wildlife and even extinction of some relict populations, leading to loss of biodiversity and damage to viability of national parks with effects on tourism.
- Destruction of developing improved farming systems such as per-urban dairying; loss of indigenous and improved genetic material.

**(B) Risk: rinderpest continues to pose a constraint to trade prospects**

**DRIVER 3:** Should the Somali pastoral ecosystem rinderpest problem not be resolved but the virus be contained with the Somali ecosystem, a combination of localised rinderpest resurgences and occasional escape from Africa in consignments of trade cattle will alert trading partners to the continuing presence of rinderpest.

**Sources:** An occult reservoir of rinderpest in the Somali pastoral ecosystem.

**Pathways:** Rinderpest infection within the Somali ecosystem reservoir follows the usual cycle of enhanced virulence at five-year intervals and occasionally induces clinical disease in trade cattle and recipient communities.

- The demand for livestock in Middle East markets such as those in the United Arab Emirates continues despite the known risk, because of the reduced cost of animals.
- Traders into the Middle East use the threat of rinderpest to negotiate prices downward and enhance their profits.

- Long distance clandestine trade into the Far East could provide a dramatic demonstration of the risks.

**Hazards:**

- Rinderpest continues to cause livestock losses within the Somali pastoral ecosystem and occasionally causes devastating losses in wildlife.
- Rinderpest is seeded into Asia producing localised or even extensive outbreaks.

**Likelihood:** High

**Outcomes:** These would relate to immediate direct effects on livelihoods exacerbated by indirect and longer-term effects:

- Destruction of trade. Trade bans imposed by trading partners within Africa and outside would further damage rural livelihoods and the developing meat and animal trade, for example in Ethiopia, Sudan and Somalia.
- Death of wildlife and even extinction of some relict populations leading to loss of biodiversity and damage to viability of national parks, with reduced tourism.

**(C) Risk: failure of global rinderpest eradication programme**

**DRIVER 3:** Should the Somali pastoral ecosystem rinderpest problem not be resolved, neighbouring involved countries – Somalia, Kenya, Ethiopia, Djibouti, Sudan, Uganda and Tanzania - will continue to be prevented from completing the process of accrediting their rinderpest freedom.

**Sources:** An occult reservoir of rinderpest endemicity persisting in the Somali pastoral ecosystem.

**Pathways:** Rinderpest infection within the Somali ecosystem reservoir follows the usual cycle of enhanced virulence at five-year intervals and occasionally induces clinical disease in trade cattle and recipient communities.

- The demand for livestock in the Middle East markets such as those in the United Arab Emirates currently continues despite the known risk because of the relatively low cost of animals.
- Traders into the Middle East use the threat of rinderpest to negotiate prices downward and enhance their profits.
- Long-distance clandestine trade into the Far East could provide a dramatic demonstration of the risks.

**Hazards:**

- Rinderpest continues to cause livestock losses within the Somali pastoral ecosystem and occasionally causes devastating losses in wildlife.

- Rinderpest is seeded into Asia, producing localised or even extensive outbreaks and possibly the re-establishment of endemic rinderpest in its old strongholds.

**Likelihood:** High

**Outcomes:** Whether or not rinderpest is actually present within the Somali pastoral ecosystem, the concerned countries will be prevented from following the OIE Pathway to full accreditation of rinderpest freedom.

As a result:

- The GREP, a time-bound programme, will fail to drive all countries to OIE accreditation of freedom from rinderpest infection by 2010. It is unlikely that another internationally coordinated programme will be mounted again.
- Attrition of wildlife in eastern Africa will continue.
- Development of trade opportunities will continue to be constrained.

**Comment:** A similar but lesser effect would occur if the virus is no longer present but uncertainty persists to cause grumbling suspicions about the risks of livestock trade.

### **The role that detection and identification did play, could have played or should play in managing the risks**

The African rinderpest diagnostic network was established by FAO through a separately-funded epidemiology project within the PARC programme with technology support from IAEA through the Joint FAO/IAEA Division. It made a very significant contribution to rinderpest surveillance. Providing certainty in diagnosis led countries, sometimes reluctantly, to be more open about their rinderpest status. Countries were, and still are, reluctant to notify disease outbreaks without laboratory confirmation given the trade implications. Valuable time was often lost in establishing control. A seromonitoring programme played a significant role in the intensive eradication programme mounted in northern Tanzania to eliminate the 1997 epizootic. Elsewhere in Africa, during PARC, seromonitoring failed to make the impact it should have on the management of vaccination programmes because the results were not provided in time to be used for influencing actions.

More recently, as fighting rinderpest gives way to proving rinderpest freedom, serosurveillance is increasingly important and is a prerequisite for OIE accreditation of rinderpest freedom. The portfolio of laboratory tests used in surveillance programmes comprised immunocapture ELISAs<sup>9</sup> for the differentiation of rinderpest and the closely related peste des petits ruminants, and competitive monoclonal-antibody-based ELISAs. These are capable of differentiating between the antibodies induced by the two viruses. The tests were developed by the Institute for Animal Health Pirbright Laboratory with

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<sup>9</sup> Enzyme-linked immunosorbent assays

some contributions on PPR being made by the CIRAD laboratory at Montpellier, France. A major breakthrough came with the establishment of RT-PCR<sup>10</sup> and sequencing for diagnosis and virus characterisation in the mid 1990s at the IAH. Designated as the FAO World Reference Laboratory for Rinderpest (WRL) and given token financial support, from 1995 the laboratory provided a free referral diagnostic and rinderpest molecular epidemiology service. The latter was crucial to understanding the global rinderpest situation and thereby in the development of GREP strategies. Amongst other issues, this helped to clarify the relationships between the South Asian rinderpest reservoirs and outbreaks in the Arabian Peninsula; the relationship between



enigmatic outbreaks in Georgia and border areas of Russia with Mongolia and China; and the relationships between outbreaks and reservoirs of infection in Africa. The most dramatic example of the value of this WRL service was when rinderpest was rapidly determined to be the cause of wildlife mortality in Tsavo National Park in Kenya, after tests conducted at the Kenya Agriculture Research Institute had proved negative. What is more, viral characterisation of the F gene refuted the suggestion that the virus could have come from southern Sudan, as was initially suggested, because the virus was identified as belonging to African Lineage 2. Viruses

of this lineage had not been seen in eastern Africa for some 30 years and this meant that a previously undisclosed focus of virus persistence had to be sought.

A rapid chromatograph strip test developed as a 'pen-side test' for rinderpest by the Pirbright Laboratory and commercialised through Svanova Pty Ltd was widely employed in the final stages of the rinderpest eradication programme in Pakistan. It made a critical contribution in detecting the final, single cases. It proved to be a tremendously enabling technology for field veterinarians, encouraging them to report their suspicions to incredulous senior officers. It also helped to confirm that rinderpest was no longer occurring in the Landhi Dairy Colony of Karachi where the virus had been constantly present for

<sup>10</sup> Reverse transcriptase-polymerase chain reaction

decades. It has also proved useful in the southern Sudan campaigns but less so in Kenya and Somalia where animal health workers seem to have had problems using it.

In the final stages of the rinderpest eradication effort, which must focus on the Somali pastoral ecosystem of eastern Africa where a virus of low virulence (often virtually subclinical) has been involved in rinderpest persistence, effective epidemiological studies and control require efficient and timely laboratory services. Sustaining this laboratory service within eastern Africa has proved difficult in recent years contributing to a lack of resolution of the rinderpest enigma.

Should rinderpest outbreaks occur anywhere in the world outside the suspected reservoir in eastern Africa, characterising the causal virus as a means of tracing its origin will be essential to regaining control. New tools such as marker vaccines and DIVA tests would also be a valuable asset in future should the virus re-emerge for whatever reason, natural or malign. These tools could also prove crucial to timely eradication of the focus of 'mild' rinderpest in Somalia and Kenya, should it be confirmed as present, by allowing vaccination and serosurveillance to continue simultaneously.

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