DISSERTATION

THE DYNAMICS OF RINDERPEST IN NOMADIC PASTORAL SYSTEMS: THE SOMALI SURVEILLANCE EXAMPLE

Submitted by
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WE HEREBY RECOMMEND THAT THE DISSERTATION PREPARED UNDER OUR SUPERVISION BY STEFANO TEMPIA ENTITLED THE DYNAMICS OF RINDERPEST IN NOMADIC PASTORAL SYSTEMS: THE SOMALI SURVEILLANCE EXAMPLE BE ACCEPTED AS FULLFILING IN PART REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY.

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ABSTRACT OF DISSERTATION

THE DYNAMICS OF RINDERPEST IN NOMADIC PASTORAL SYSTEMS: THE SOMALI SURVEILLANCE EXAMPLE

The Somali economy is the only one in the world where over 60% of the population is dependent on nomadic pastoralism for its livelihood. Furthermore, contrary to most pastoral systems which are normally devoted to the household subsistence, the Somali system is radically oriented towards livestock trade and export.

However, the complete absence of veterinary control measures, due to the collapse of the Somali Government in 1991 and the lack of appropriate techniques for investigating disease events in nomadic conditions have rendered the Somali livestock industry increasingly vulnerable and susceptible to the imposition of bans from importing countries. The most recent bans have been related to the suspicion of rinderpest (RP) and Rift Valley fever (RVF) viruses circulating among the Somali livestock population.

It is currently believed that the only remaining foci of RP circulation in the world are situated in the so called Somali Eco-System which encompasses the Somali inhabited areas of Kenya and Ethiopia and the central and southern regions of Somalia. In order to delineate the extent of the remaining foci of RP in Somalia, a cross-sectional sero-survey based on a two stage cluster sampling was designed. To obtain a representative sample in the absence of a sampling frame, the random selection of the primary sampling units was attained by the use of random map coordinates. The survey was conducted in ten administrative regions of central and southern Somalia.

A total of 9,216 serum samples were collected from cattle aged 1 to 3 years in 562 sampling sites. The spatial dependency of the observed sero-prevalence was tested by means of Moran's I and Local Indicator of Spatial Autocorrelation (LISA) statistics. Both tests indicated a statistically significant

spatial autocorrelation of the observations, and two spatial sero-prevalence clusters were detected, suggesting the existence of two potential foci of RP maintenance in the country.

Furthermore, the integration of conventional statistical techniques (i.e. logistic regression models) with spatial analysis and Geographic Information Systems (GIS) allowed the determination of the risk factors for RP maintenance and spreading as well as the estimation of the spatial risk of RP occurrence in the study area. The latter was used to develop a risk based approach for the zonation of the country according to the guidelines of the International Office of Epizootics (OIE).

The study has demonstrated that the utilized methodology is able to produce reliable information from mobile livestock keeping systems. Furthermore, the study has generated important baseline data that will give a more focused direction towards the final eradication of RP in the country.

At present, the proposed methodology has been recommended by the African Union – Inter-African Bureau for Animal Resources (AU-IBAR) to simultaneously investigate the status of RP in the three countries of the Somali Eco-System in order to generate relevant information that will lead the final eradication of the disease from the Horn of Africa and the world.

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INTRODUCTION AND RESEARCH OBJECTIVES

1.1. Characteristics of pastoralism in Africa

The hottest climatic portion of the earth's surface lies between the Tropic of Cancer and the Tropic of Capricorn. Within this zone lies the East African Sahel. The evolutionary and anatomical development of humans made it possible, first, for African hunters and gatherers to live in the East African Sahel, followed thousands of years later by African pastoralists. Pastoralism was only possible due to one crucial anatomical and evolutionary development which is the ability to meet the challenge of exertion in a semi-arid zone with a hot, dry climate (Reader, 1997).

This conquest over heat stress was the forerunner to the symbiotic relationship which later developed in the savannah lands of the Sahel between pastoralist populations and domesticated animals, where one would not have been able to survive without the other (Drysdale, 2000).

The extremely harsh environmental conditions of these lands made the pastoral system a highly dynamic system, where size and herd/flock composition are constantly changing over time and space, and where animals are continuously moving in search of water and better pasture.

1.2. The "special" case of Somalia

The Somali economy is the only one in the world where over half the population is dependent on nomadic pastoralism for its livelihood. Furthermore, contrary to most pastoral systems which are normally devoted to the household subsistence, the Somali system is radically oriented toward livestock trade and export (Dietz *et al.*, 2001).

The Somali tradition of livestock export dates back to the 1940-1950s when the first commercial relationships were initiated with the Gulf Countries. In the seventies, due to the oil boom in Saudi Arabia and other Gulf countries, Somalia benefited from its strategic geographical position, its secular tradition in livestock keeping and the sharing of a common religion, to become the leading country in the supply of live animals to the Gulf countries. After the collapse of the military regime of Mohamed Siyad Bare in 1991, the economy of Somalia went underground, and many herders and traders have benefited from the growth in cross-border trade to neighboring countries and overseas export. To date Somalia remains the leading country in the supply of live animals to the Gulf (Steffen *et al.*, 1998).

A clear example is given by the livestock export figures provided by the Berbera Port Authorities of Somaliland. For instance in 1997, 2,700,000 small ruminants, 66,000 cattle and 52,000 camels were exported from the Berbera port to Saudi Arabia alone. The income generated by the total livestock export through Berbera to Saudi Arabia in the same year, at producer price, was around US $93 millions. These figures show an ongoing economy despite the lack of a recognized state. The 1997 figures in quantity terms represent a 150% increase on the 1977 exports of sheep and goats to Saudi Arabia, and a 319% increase on camel exports in the same year (Drysdale, 2000).

1.3. Factors negatively affecting the Somali livestock industry

Nowadays, the economy of Somalia is entirely unofficial. The Somali economy, however, has proven, in terms of organizational aspects, to be able to function despite the total absence of a recognized government.

Despite this fact, the complete absence of veterinary control measures has rendered the Somali livestock industry increasingly vulnerable and susceptible to the imposition of bans from importing countries, mostly related to the suspicion of rinderpest (RP) and Rift Valley fever (RVF) viruses circulating among the Somali livestock population. Bans and restrictions on Somali livestock that occurred over the past 20 years are listed below (Steffen *et al.*, 1998):

- 1983: RP ban imposed by Saudi Arabia
- 1998: First RVF ban imposed by the Gulf Countries
- 2000: Second RVF ban imposed by the Gulf Countries
- 2000: Kenya – Somalia border closed to the cattle trade due to the suspicion of RP virus introduced into Kenya from Somalia
- 2001: RP vaccination requirement imposed by Yemen for cattle imported from Somalia

Nevertheless, the lack of a recognized government and official veterinary services is not the only problem that has affected the Somali livestock industry. In fact, the creation of the World Trade Organization (WTO) and the subsequent signing of the agreement on the application of sanitary and phytosanitary measures (SPS Agreement) have laid the foundation for the reduction of tariff barriers to trade. As a result, sanitary barriers have become the only legitimate non-tariff barriers to trade in

livestock and livestock products. Countries wishing to export livestock and livestock products will be requested to substantiate claims of being free from specific livestock diseases.

To prove the absence of disease, a passive and historical register of disease occurrence is not enough. Official Veterinary Services **must have a credible surveillance system** where (a) any suspicious signs of disease activity are reported and (b) **statistically selected samples from the host population** are collected in order to detect clinical signs or other evidence of transmission of infection. In either case, suspicion of disease must be followed by quarantine, confirmatory diagnostic work and any other necessary disease control activities.

In addition to veterinary infrastructure, both of these aspects will require animal health data of a quality and quantity which are currently not available in a large number of countries, particularly developing ones. In fact, the quality of data, especially in certain African countries, is often affected by, among other factors, the lack of appropriate techniques to investigate events occurring under different conditions.

1.4. Rinderpest in Somalia

Until 1994, when a mild form of RP was detected and diagnosed in the Tsavo East National Park and subsequently in the Nairobi National Park (1994-96), the main endemically infected area in East Africa was believed to be Southern Sudan. From this source, infection regularly invaded adjacent areas of Uganda, Kenya and occasionally Ethiopia. All virus isolates recovered from Southern Sudan and these neighboring areas since 1983 were of the African type 1 lineage.

Initially the Tsavo RP outbreak was thought to have originated from southern Sudan but the molecular evidence clearly showed that the Tsavo virus and the isolates from Nairobi National Park were completely different genetically and fell into the African type 2 lineage. Isolates of this lineage had been recovered from West Africa as late as 1983 but not since 1962 in East Africa. Thus a second main focus of RP in East Africa was revealed after having remained undetected throughout the period of the Joint Project No. 15 (JP15) campaign and eight years of the Pan-African Rinderpest Campaign (PARC). The exact location of this focus was uncertain, but surveillance had concentrated on north eastern Kenya and southern Somalia (Barrett *et al.*; 1998).

1.5. The Global Rinderpest Eradication Effort and the Pan-African Rinderpest Eradication Programmes

In the 1970s the Food and Agriculture Organization of the United Nations (FAO), together with the International Atomic Energy Agency (IAEA), checked veterinary diagnostic laboratories in allegedly high risk countries in Africa and Asia. The objective was to identify the true prevalence of RP. In 1980 FAO recruited three consultants to prepare regional RP campaigns by assessing real disease prevalence, diagnostic facilities, vaccine production plans, vaccine quality control, available manpower, equipment, and estimates of budget for the eradication of RP (Spinage, 2004).

Their detailed report entitled "Global Eradication of Rinderpest" was presented, discussed and approved at a FAO Expert Consultation Meeting held in Rome in October 1992. The Expert Consultation issued a statement that global eradication of RP was achievable by the year 2010 (Anon., 1997). The 106th FAO Council authorized the Director-General to establish a priority programme known as the

Emergency System for Transboundary Animal and Plants Pests and Diseases (EMPRES). The initial focus was the Global Rinderpest Eradication Programme (GREP) (Spinage, 2004).

The Secretariat collaborates with other international organizations such as the International Office of Epizootics (OIE), the International Atomic Energy Agency (IAEA), the European Union (EU) and the African Unity / Inter-African Bureau for Animal Resources (AU/IBAR). The important link is the OIE, which oversees the zoosanitary approach necessary to achieve eradication using a practical moving pathway which every country has to follow culminating in a declaration of freedom from RP virus (RPV) (OIE, 2005).

According to the GREP's Blueprint for Africa, the continent should be free from infection by the end of the year 2008 and globally by the end of 2010. This goal will be accomplished through the implementation of a regionally modified "OIE pathway", which will take into consideration animal movements inside eco-zones based on epidemiological surveillance without respect to political borders. Targeted vaccination campaigns will be carried out in those areas where the presence of the virus has been demonstrated.

The first structured Pan-African RP eradication effort started in 1962 when an internationally funded and coordinated vaccination campaign, JP15, was initiated. At the termination of the vaccination campaign in 1976, over 70 millions heads of cattle in 22 countries were vaccinated at a cost of US$ 51 millions, but internecine difficulties in some countries, particularly Sudan and Ethiopia, meant that complete coverage could not be achieved and the disease remained enzootic in some areas (Spinage, 2004).

From Sudan the disease spread again and in 1980 RP was reported in several countries of East, West and Central Africa. This was followed up by an international conference in Kenya in November 1981 at which a Pan-African Rinderpest Campaign (PARC) was proposed under the aegis of the Organization for African Unity (OAU) and the Inter-African Bureau for Animal Resources (IBAR), which was to be conducted initially by FAO and OIE and after 1994 by GREP. The field operation started in 1987 (Anon., 1983).

While eradicating RP, PARC had strengthened animal health services to the benefit of livestock producers. In areas where government services were not functional due to civil conflict, economic depression and political instability, other partners have also being assisting AU/IBAR in the eradication of RP. This is the case in Somalia, where Non Governmental Organizations (NGOs) have been the implementers of PARC and the actual Pan-African Control of Epizootics (PACE) Programme.

In fact in the middle of the year 2000, PARC was replaced by the ongoing PACE Programme, which is currently running in 32 African countries again under the auspices of AU/IBAR. The aim of PACE is to enhance the epidemio-surveillance structure of the member countries. In doing that the final eradication of RP from Africa remains a priority.

1.6. Problematic issues

As mentioned before, the eradication of RP should be accomplished in Africa by the year 2008 and globally by the year 2010. So far Somalia or better the so called Somali eco-system, which includes central and southern Somalia, the North Eastern Province of Kenya and the Region 5 of Ethiopia, is generally considered to harbor the last foci

of RP in the world. The lack of structured veterinary services in Somalia and especially the inadequacy of available investigation techniques in nomadic systems represent a challenge for the final eradication of the disease.

In fact, so far, surveillance techniques have been developed assuming that certain conditions apply to a specific system being investigated. For instance, the random concept is at the base of a representative estimation of the level of a disease, meaning that a thorough knowledge of individuals composing the study population is available.

This condition is seldom applicable in most pastoral production systems in Africa. Furthermore, the requirement of estimation of specific parameters such as the variance between clusters, assumes that animal clusters maintain a rather static structure for a certain time span.

This condition is often found in sedentary or semi-sedentary husbandry systems, but it is hardly the case in nomadic herds. Sampling units, usually defined as herds/flocks/farms, are often suggested for epidemiological investigations in sedentary/semi-sedentary livestock production systems, while populations of animals concentrating at watering points or in villages are considered to be alternative sampling units in nomadic or transhumant livestock production systems. Both sampling units are believed to be unsatisfactory for highly dynamic livestock populations because herd/flock composition and size are constantly changing over space and time, while population sizes at watering points/villages are often very large, as well as very variable. In these conditions, interpretation of results is very problematic in longitudinal as well as in cross-sectional studies.

1.7. Objectives of the study

The objectives of this study are:

1. To develop, evaluate, and assess a survey approach in highly dynamic nomadic pastoral systems using RP as a model. Analytical techniques for this proposed survey will be utilized.

2. To construct a model of pathways that explain RP dynamics in the pastoral system so as to lead to most focused and appropriate control/eradication interventions. This will take into consideration the dynamic of the Somali livestock population and its mechanisms of aggregation and dispersion over space and time.

The outcome of the study will assist to identify the most appropriate surveillance methodology and animal disease control measures for the Somali pastoral system so as to support the livestock industry of the country. The Somali example will be used as a model for other existing pastoral conditions.

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CHAPTER 2:
LITERATURE REVIEW

2.1. Pastoralism in Africa with special emphasis to the Horn of Africa

This chapter is not meant to provide an accurate socio-anthropological or economical analysis of the various pastoral production systems existing in the world. By contrast, the aim of this review is to provide an outline of some features of the pastoral milieu in Africa in order to offer to the reader a prospective of the "environment" where the research work has been carried out as well as some consideration on the "modern" pastoral production system particularly the Somalia situation.

Contemporary pastoralists can be roughly divided into two groups: those who live in cold and temperate areas and those who live in the hot, arid and semi-arid parts of the world.

It has been estimated that there are between 50 and 100 million nomadic or transhumant pastoral people in the world (Omar, 1992). In some countries, for example Kenya, nomadic or transhumant pastoralists occupy two-thirds of the country although they comprise less than 15% of Kenya's total population (Anon., 1981).

Pastoralists manage over 120 millions cattle or cattle equivalent units of livestock (McDowell, 1980), and they own most of the livestock in many countries including

Kazakhstan, Mongolia, Tibet, Jordan, Iran, Turkey, Iraq, Algeria, Morocco, Niger, Senegal, Libya, Tunisia, Nigeria, Sudan, Somalia, Peru and Chile.

Nomadic or transhumant pastoralists maintain a range of different livestock species in different parts of the world: sheep, goats, cattle, dromedaries and donkeys are kept in Africa; water buffalo are additionally maintained by transhumant people living in Rajasthan; Kazakh and Tibetan peoples maintain bactrian camels, horses, pigs and yaks; llamas and alpacas are herded by South American nomadic people (Macpherson, 1995).

2.1.1. The origin of African pastoralism

The Tropic of Cancer and the Tropic of Capricorn, approximately 23° latitude north and south of the Equator respectively, set the limits of the tropical belt, which represents the warmest portion of our planet. Between these parallels, at some part of the year, the sun shines directly overhead at midday. As a consequence this belt is also known as the Torrid Zone (Mackie, 1985). It is within this zone that lies the East African Sahel, especially the Rift Valley, the cradle of all humanity (Brauer, 1989; Cann *et al.*, 1987; Wilson, 1985), and of course, of the modern African Pastoralists. The evolutionary, anatomical development of humans made it possible, first, for African hunters and gatherers to live in the East African Sahel, followed thousands of years later by African pastoralists in ancient and modern times. Pastoralism in Africa was only possible because of one crucial anatomical development which could meet the challenge of exertion in a semi-arid zone with a hot, dry climate (Reader, 1997). The conquest over heat stress was the forerunner to the "symbiotic relationship" which later developed in the savannah lands of the Sahel between pastoralists and domesticated animals, where one of them would not have been able to survive without

the other – to this day. This unique "symbiotic relationship" allowed nomadic pastoralists to survive and make use of lands that were and still are almost off-limit for any form of agriculture (Drysdale, 2000).

The exact origin of African pastoralism is not clear. Clarck (1980) suggests that at first the Sahara had been occupied by nomadic hunters, gatherers and fishers, then depopulated following environmental changes, which must have been responsible for readjustment in game distribution, resulting in more concentration near to the water resources and so to closer proximity to the human settlements. Under such conditions of stress caused by increasingly sparse and patchy grazing, the movement toward controlling the wild herds in an effort to maintain the previous quality of life is not surprising. A direct consequence of this would have been restrictions on the animals' movements and feeding, along with restricting the gene pool in reproduction and manipulating the genetic material to produce "culturally" acceptable animals. In fact, it appears that the beginning of pastoralism in Africa stems from the close association that would have developed between hunters of large game and their prey.

With the amelioration of the Saharan environment around 7000 BP, a grassland ecological niche opened up that coincided with the early appearance of domesticated small stock probably coming from the Near East. This improved pasture environment permitted colonization of the Sahara by "pastoral" people who adapted a mixed herding strategy to include cattle domestication along with the exotic smaller caprines in order to cope with the still quite hostile environment.

Regardless of the source, nomadic pastoralists spread rapidly throughout the central and southern Sahara from the Nile Valley around the confluence of the Blue and White Niles (Krzyzaniak, 1976a & 1976b) to the Aïr Mountains and the northern

Tilemsi Valley. Further west pastoralists spread to the Araouane Lakes north of Timbuctoo (Joleaud, 1935 and Guérin & Faure, 1983). Along with this spread of pastoralism are certain traits held in common over this vast area from the Nile to the central Sahara in Mali and Niger, which characterize African pastoralists till today: the extreme mobility of these people and their herds / flocks in the continuous search of water and better pasture in order to survive in the hostile environment of the arid and semi-arid zones of Africa.

2.1.2. Grassland ecology and pastoralism with special emphasis to the Horn of Africa

Much of the anthropological interest in African pastoralism stems not only from the cultural responses of people that revolve around domestic animals, but also from the importance of herding economies in almost all countries. In fact the pastoral "responses" to an arid or semi-arid ecological context makes pastoralism the most effective traditional mean of resource exploitation and land use in the grassland of Africa (Topps, 1977). In actual fact the unique adaptation of pastoralists is to environments in which any other form of exploitation has lower productivity, namely semi-arid grasslands.

The special adaptation of a "livestock based" economy requires mobility to make use of the seasonal availability of pasture and water. In most parts of Africa where livestock is the economic mode, distinct dry and rainy seasons are to be found. Movement of pastoral camps will vary with the time of the year and access to the needed resources. It is a general pattern that camps are widely dispersed during the rain season when pasturage is abundant, and then tend to congregate around water-holes when the dry season is at its peak. The animals themselves play a role in

deciding when a camp should be moved. It is fairly obvious that certain animals, such as camels and goats, have lower water needs than cattle (Schmidt-Neilson, 1964); therefore, a pastoral economy based on camel husbandry will have a different mobility pattern from one based on cattle (Gulliver, 1955). In many parts of Africa, however, mixed animal herds are the most common; therefore the water needs of cattle, specifically young age animals, often determine when a camp should be moved (Smith, 1980). Mixed herds may also require that they break up at certain periods of the year with different animals going to different pasture zones (Gulliver, 1955; Spencer, 1973).

In general terms, the direct correlation between soil nutrients, plant nutrients and grazing animal carrying capacity can be used to define the quality of the pastoral environment, as well as to show the constraints placed on the herds. Mobility is then a direct function of the respective environments. The ecological diversity of Africa, from tropical rain forest, through Mediterranean winter areas, to arid desert environments, is a response to precipitation, temperature and the wind parameters of the Intertropical Convergence Zone, as well as to the geological substrates and soils. Altitude differences have produced varied temperatures and pasture conditions, from C4 grasses at level below 2.000 m to C3 above 3.000 m (Tieszen *et al.*, 1979a & 1979b), and a concomitant response to sweet and sourveld grazing. Add the restrictive tsetse flies and one can see the wide range of pastoral environments that exist across the continent.

The Sahara and the Sahel: the largest nomadic pastoral zone in Africa is the vegetation belt on the southern fringes of the Sahara running from the Atlantic coast in Senegal and Mauritania, across Mali, Niger and Chad to the Nile Valley in the

Sudan. It is known as the Sahel, or *"shore"* in Arabic, i.e. it would have been the first vegetation encountered by the Arabic travelers crossing the Sahara in the ninth to the twelfth centuries. It is a summer rainfall area (the rains come between June and September), but precipitation varies from year to year. This restricts dramatically the amount of cultivation which can be practiced. The substrate varies between sandy soil of clay origin, forming dunes, and peneplains. These sandy areas are separated by numerous limonite clay depressions which become the catchments for run-off during the rains.

While much of the Sahara is devoid of adequate vegetation for pastoral use, the mountain areas, such as the Hoggar, Tibesti, and Aïr, have relatively shallow aquifers, so vegetation, particularly browse for camels, can be found. Furthermore, since water can be obtained from shallow wells, it is added encouragement for human occupation.

The vegetation is basically an *Acacia*-steppe grassland. The trees are short and good forage for camels, while the grasses – *Pennisetum, Panicum, Chenchrus,* etc. – are ideal for cattle and small stock grazing. In general the biomass of vegetation is low. In the northern part of the Sahel, on the edge of the Sahara, the biomass is only 500 kg. per ha dry matter. This increases to between 1,000 and 3,000 kg. per ha further south. In contrast, the *"bourgou"* or grass, *Echinochloa stagnina*, along the banks of the Niger River can attain a biomass between 6,000 and 17,000 kg. per ha (Penning de Vries & Djiteye, 1982). To the south of the Sahel is the savannah zone (*zone soudanienne*), a dense grassland with riparian tree cover where an upper stratum of the grasses can be over 80 cm in height and rainfall is between 600 and 1,000 mm per annum. This is also an area of tsetse infestation, particularly during the rain season; so it becomes the southern limit for pastoralism.

The greatest amount of movement by pastoral people in this area is during the rainy season, when standing water allows the southern fringes of the Sahara to be exploited. As the dry season continues, people are to be found around permanent water.

The Horn of Africa and Northeastern Kenya: Rainfall is low and somewhat unreliable in this area, which encompasses Somalia, southern Ethiopia and north eastern Kenya (the so called Somali Eco-System, this area being inhabited by Somali ethnic groups). For that reason camel pastoralism takes precedence, with the limited cultivation taking place along the rivers draining the western plateau southward to the sea. Because drainage is poor these rivers tend to be tsetse-infested during the rainy season from May to August, making life difficult for people at this time of the year (Grove, 1971).

In the higher ground away from the Indian Ocean the topography consists of open plains, broken in places by mountain ranges which rise abruptly. Below the mountain of northern Somalia, the slope is so slight that low velocity sheet wash during the rains carries clay and silt in suspension, along with organic matter (Greenwood, 1957). This area produces arcs of vegetation across the direction of the water flow. Animal excreta are highly visible, resulting in dark color soil. On some of these arcs *Andropogon cytrocladus* grows as high as one meter, and on others mixed *Acacia* spp. and grasses are present. The Somali nomads use these areas during the rainy season for pasturing their stock. During the dry period they are to be found on the periphery around permanent water-holes (MacFadey, 1950).

The localized and variable rainfall across the entire area can be seen in the annual precipitation of northern Somalia, where over 700 mm has been recorded at Hargeisa, while areas to the east often have less than 100 mm annual precipitations. This region

has two rainfall periods – April-June and September-November – which support a thorn-scrub vegetation. Grasses on sandy soils include *Amphiliphis radicans* and *Pennisetum orientale* among others (Collenette, 1931). Information on animal production in this semi-desert environment for north eastern Kenya and southern Somalia are described in detail by Pratt and Gwynne (1977). These areas cannot sustain a carrying capacity of livestock greater than one livestock unit to 40 ha, even when water supplies are adequate. In this area wooded grassland and shrub grassland occur locally, but open grassland are the main vegetation zones. Seasonally waterlogged grasslands produce mainly *Eriochloa* and *Lintonia*. Elsewhere, with continuous over-grazing, a *Sorghum purpureo-sericeum* annual grassland appears.

2.1.3. African pastoralism today

It is probable that the first pastoral people to be contacted by European exploration in the 15th century were the Khoikhoi of Southern Cape. In 1448 Bartolomeu Dias and his crew helped themselves to water at Mossel Bay on the southern trip of Africa. The result of this was that the local Khoi herdsmen threw stones at the sailors, causing Dias to shoot one of them with his crossbow. From the Portuguese explorers' basic misunderstanding of how Africans look on their natural resources, it is possible to start listing many of the worse excesses of the colonial exploitation of African people.

Europeans did not understand the relationship between African social organization and the economics of environmental exploitation in pastoral society. This is mainly due to the fact that Europeans consider private ownership of land to be first and foremost the only economically viable way of efficiently utilizing the land. Land ownership has meant an exploiting class and a class that has been exploited. Therefore, the communal ownership of land and water-points that was practiced by

African pastoralists was a threat to European values. Thus, the commonage of lands was initially strongly opposed by the European colonizers. It was only after a long time that the pastoral way of land exploitation proved to be the only solution to use viably arid and semi-arid areas, and it has remained almost unchanged over the centuries till today.

On the other hand the contact with the "Western World" has induced quite radical changes in the way in which pastoralists see their herds. Rigby (1985) makes a useful distinction between pastoralists who still see their herds as a means of production, and those who translate their animals into other commodities. The theoretical assumption underlying this distinction is that increasing contact with capitalist formations and outside influences generally requires a shift toward commoditization.

A particular case of capitalization of the pastoral "products" is offered by the Somali pastoral economy where over half of the population of the country is dependent on nomadic pastoralism. In fact, in the case of Somalia the pastoral system is devoted to both the household substance and the cross-border livestock trade and oversee export. It is this last that generates the added value to the Somali animals, which in return produces more cash flow for acquisition of other commodities and at the end also a better use of the natural resources. In fact, pastoralists, gaining from improved incomes as a result of higher prices obtained by the trade of their animals to importing countries, require less "livestock units" for purchase of other commodities essential to the household subsistence and therefore less pressure on the environment.

2.1.4. Pastoralism and "pastoral economy" in Somalia

After the government collapse in 1991, the former Republic of Somalia has been progressively subdivided by clanic conflicts in unrecognized entities namely: Somaliland (North-West), Puntland (North-East), and the Somalia (central and southern Regions).

Livestock production still forms Somalia's predominant food supply by providing about 55% of the overall calorie intake of Somali people, but also contributes by about 60% to the incomes or subsistence of the Somali population. Livestock, especially cattle, camel and small ruminants also plays an important role in the socio-economic welfare of all Somali. They depend directly or indirectly on livestock for cash, food, household power, soil fertility and social status.

Data on the livestock population in Somalia are outdated and subject to a wide margin of error, and the current livestock population can only be estimated. Projections are about 5.2 million cattle, 13.5 million sheep, 12.5 million goats and 6.2 million camels with the cattle population concentrated in the south and the majority of camel population located in the centre and the north. However, comparing livestock on the basis of Tropical Livestock Unit (1 TLU=250kg live weight) clearly shows that in terms of body-mass camels are most important (41%), followed by sheep and goats (35% combined) and then cattle (24%). Average livestock density in Somalia therefore is about 0.25 TLU/ha or about 0.43 TLU/ha of area classed as rangelands (some 55% of Somalia) (Terra Nuova, 2001a).

2.1.4.1. The production system

With about 60% of the Somali population being nomadic pastoralist, nowhere in Africa is nomadism of greater significance than in Somalia. Somali nomadic pastoralism using traditional techniques is characterized by its large herd sizes and high mobility. Combined with herd diversification (in terms of livestock species) and herd dispersion, this allows better utilization of scarce pastures and serves as a risk-reduction strategy in case of local forage shortage, raising, and disease outbreaks.

Minimum input at the production level, resulting in a low economic value of the individual animal, reduces the economic risk to the livestock owner but also reduces the productivity of the system. The Somali nomadic livestock husbandry has proved to be highly extensive but nevertheless highly efficient in terms of input conversion.

The advantage of the nomadic production system is its adaptation to the harsh and unpredictable environmental conditions of the drier areas covering most of the country. The maintenance of mobility as a risk management strategy to cope with dry season and drought is a key reason why Somalia's livestock sector has not suffered as much as other areas of the economy after the government collapse. In fact, the ability of nomadic people to cope with "environmental stresses" and to avoid areas where threat was caused by deadly livestock diseases (e.g. tsetse infested areas) had proven to be also extremely conducive to avoid areas of open conflict after the civil war. Therefore, mobility has basically provided the opportunity for the "Somali livestock industry" to remain almost unaltered even during the 14 years of civil conflict, which has made Somalia the only stateless country in the world.

Furthermore, even during the "government time" very little or no veterinary services were available to the nomadic communities, since they were very difficult to reach.

Thus, the collapse of the government had very little impact on the day-by-day life of the Somali pastoralists.

In comparison, agro-pastoralist and settled mixed farming systems, representing 25% of the Somali population, as well as urban staff feeding (15%), can be considered a less effective production system, if regarded in terms of their contribution to national economy. They can be characterized by low mobility and herd size and increased demand of external fodder supply. This less mobile system was much more vulnerable to the political instability that has characterized Somalia during the 14 years of civil war. Agro-pastoralist and mixed farming are therefore associated with fodder cultivation and cropping areas located in the sub-humid and inter-riverine areas in part of southern Somalia. Similar systems based on cultivation of rain-fed crops combined with open-range livestock herding are common in the Northwest. Nomadic systems are significant suppliers of livestock for trade and export, while agro-pastoral and mixed farming systems for livestock and milk especially to internal markets and urban stall-feeding plays a substantial role in supplying local peri-urban milk and meat markets (Terra Nuova, 2001a).

2.1.4.2. The livestock trade and oversea export

Livestock trade in Somalia is a long lasting tradition and sizeable commercial sales from nomadic herds have been routine for decades. Therefore, in comparison to other countries in the sub-region of eastern Africa, the extensive pastoral livestock production in Somalia is also a market-integrated commercial system, as well as being subsistence-oriented in many areas. Although livestock trade is known to have started in this region in the 14th century, livestock export escalated in the 1960's when the oil

boom increased the demand for live animals in Saudi Arabia and other countries of the Arabian Peninsula (Terra Nuova, 2001a).

Until 1975, Somalia was the world's largest exporter of live animals and in 1985 ranked third after Australia and Turkey in the number of exported sheep and goats. As a result the Somali economy became significantly dependant on pastoral livestock production with livestock export contributing to some 80% of the foreign currency earning, more than 60% of employment opportunities and about 40% of GDP. Over 95% of the livestock export still goes to Gulf countries with Saudi Arabia being the major importer.

As an example, production and annual exports of small ruminants from the ports of Berbera and Bossaso actually exceeded pre-war levels and showed no signs of slowing down until an import ban was imposed by Saudi Arabia because of the fear of importing RVF virus from the country (Little, 2003).

In East Africa, the last confirmed epidemics of RVF occurred after the heavy rains ("El Nino rains") between October 1997 and January 1998. The outbreaks were confined to the North Eastern, Central, Eastern and Rift Valley Provinces of Kenya and the Gedo, Hiran, and Lower Shabele Regions of Somalia. During these outbreaks no reports were received from northern and north eastern Somalia. Nevertheless, in 1998 a ban was imposed by the Gulf countries on the Somali livestock export. Although no sound scientific evidence of the real status of the disease in the country was provided by a joint FAO / WHO mission carried out to assess the risk of RVF in the Republic of Somaliland, the ban was lifted soon after the disclosure of the encouraging mission results. When the ban was lifted in April 1999 exports recovered

quickly, reaching a record level of 2.9 millions animals during that year (FEWS, 2000).

Two years later, a second ban on imports of all livestock from Somalia, among other Horn of Africa countries, was imposed by the Gulf countries on 19th September 2000 after reports of hemorrhagic fever in humans, some resulting in mortality, and a series of abortions and deaths in small ruminants in Yemen, which were later confirmed as RVF cases. This ban is enforced to date.

Before the RVF bans, which affected mainly the export of small stocks, a RP ban was imposed by Saudi Arabia in 1983. Also this ban is still in force today.

In conclusion, it appears that the nomadic pastoral system because of its characteristic of mobility has proven to be the most efficient way of exploitation of natural resources in arid and semi-arid areas. The system is so robust that it can guarantee subsistence even during periods of open conflict and civil strife. Furthermore, even if extensive by nature, the pastoral system is not necessarily only devoted to the household subsistence, but, as the Somali example has shown, it can also be tailored toward trade and export. Moreover, as previously discussed, the commercialization of "pastoral products" may generate higher income at producer level due to the increased value that the animals acquire when sold in international markets, and may indirectly reduce the exploitation of the natural resources.

However, it appears more and more clearly that an important limitation on the exportation of animals originating from nomadic pastoral systems is due to the lack of information on the health status of the animals being exported. In fact with the creation of the Word Trade Organization (WTO) and the subsequent signing of the agreement on the application of sanitary and phytosanitary measures (SPS

Agreement), the sanitary barriers have become the only legitimate non-tariff barriers to trade in livestock and livestock products. Countries wishing to export livestock and livestock products will be requested to substantiate claims of being free from specific livestock diseases.

To do so Veterinary Services **must have a credible surveillance system** where (a) any suspicious signs of disease activity are reported and (b) **statistically selected samples from the host population** are collected in order to detect clinical signs or other evidence of transmission of infection.

While the pastoral systems can guarantee production with no or limited veterinary interventions, the application of investigation techniques that could satisfy international standards for trade and export remain a challenge for the Veterinary Services. This is due to the difficulties that apply to the study of "events" in very dynamic and mobile systems. Therefore, the exploitation of the potential of pastoral systems for trade and export relies on the set-up of "internationally recognized" epidemio-surveillance systems adequate to investigate disease events in very mobile animal production "environments".

2.2. The Rinderpest virus

2.2.1. A brief history

RP is an acute or subacute contagious viral disease of cattle and other *Artiodactyla* characterized by necrosis and erosions in the gastrointestinal tract that result in severe diarrhea and dehydration. Morbidity and mortality rates may exceed 90%, but unapparent infections may also occur. The decimation of cattle populations by RP has influenced events in human history (Gamgee, 1866; Ofcansky, 1981) and through the

decimation of cattle, wildlife and (indirectly) tsetse flies, may have altered the ecology of large areas of southern and central Africa (Stevenson-Hamilton, 1911; Taylor & Watson, 1967; see also Section 2.3.). The control and eradication of RP was a major factor in the establishment of the first veterinary school in Europe during the 18th century: a tribute to the ravages of the disease.

It has been assumed that the disease originated in Asia since it regularly invaded Europe with armies from the Orient. A monoclonal antibody study (Norrby *et al.*, 1985) has suggested that RP is the archetypal Morbillivirus from which the other Morbilliviruses of measles and distemper evolved some 5000 years ago, possibly in the river civilization of southwest Asia where the domestication of cattle is believed to have occurred (Epstein, 1971). However, one can only speculate on the possible original home of RP. Sergejew and Semmer, quoted by Friedberger and Fröhner (1886 – 87), considered that it might have been the black earth region of southern Russia near the Black Sea and the lower Don, where there is a deep humus and luxuriant vegetation. Others, they concede, consider an origin outside of European Russia in the Asiatic steppes as more probable. We know that there was an epizootic cattle disease in the region of inner Mongolia in 88 BC (Di Cosmo, 1994). It seems likely to have evolved with domestication of cattle on the Great Hungarian Plain and then taken eastward through Asia by the wars and movements of nomads which characterized the steppes; only when nomad herds had reached a certain critical density might we expect a virus strain to have become endemic among them.

The Russian name for RP is *tchouma* or *tchouma reina*, which indicates an Asiatic origin as the word *tchouma* was used by the Mongols and nomad Tartars of central Asia to denote a malevolent deity, something of the nature of a vampire. The Osmanli

Turks, the Persians and the Afghans called it *taoun*, a word believed to be derived from *tchouma*. Considered endemic in the Asiatic steppes and parts of India, early epidemics could have exercised a marked influence on the migration of pastoralists away from centers of permanent infection, but thus spreading the disease. It is possible that the Indo-Aryans, in establishing themselves in central Asia, were fleeing RP and other diseases.

Since then, RP has been for centuries the most dreaded ox plague known. The first effective control measures were edicts drawn up in 1715 by Giovanni Maria Lancisi, the pope's personal physician. Quarantine and isolation were enforced. Suspected sick cattle were killed and the effort of the Papal State succeeded, but elsewhere the belief that RP could be cured prevailed. At that time the principal professor of medicine in Padua (Italy) was Bernardino Ramazzi who attempted to keep cattle alive by scarification in the belief that pustules would protect as if it was smallpox.

In 1875 Fleming wrote that RP was known, apart from Russia and India, to be present in Mongolia, China, Cochin-China, Burma, Hindistan, Persia, Tibet and Ceylon. From these areas it was periodically introduced in Eastern Europe. When European countries closed their borders to cattle, the Tsar was stimulated to build several experiment stations in 1860 to elucidate the cause of RP and its nature. Professor E. Semmer led the Dorpat Group, which discovered in 1893 that, if serum collected from recovered cattle was inoculated into a susceptible one, it was protected against RP. Shortly, thereafter the Great African RP Pandemic hit southern Africa and the prophylactic role of RP-antiserum was confirmed. It is consider that RP was first introduced in Africa in 1841 by importation of infected Russian cattle through Egypt.

A second theory attributes its introduction in Africa to the Italian army which imported live animals from the Arab peninsula in order to feed the Italian troops fighting against the Ethiopian militia of King Menelik II. A detailed history of RP in Africa is provided in Section 2.3. (Spinage, 2004).

Antiserum alone was in vogue until 1920 but its protection was short lived. Aid personnel from Europe developed the antiserum-virus simultaneous method of immunization which conferred a life long resistance.

At the turn of the 19th century, the virus was still endemic in several countries in Asia including China, which was the source of epidemics in Indonesia and Japan. Japan's response was to develop an inactivated tissue vaccine which was a novel glycerinated organ pulp, developed by Dr. C. Kakizaki (1918). By 1925 every RP laboratory was producing belatedly an inactivated vaccine.

During the First World War, RP spread again from Asiatic Russia into Poland and then into south-east Europe. The remnants of the epidemic were eliminated in Greece in 1921. In 1930 Europe was free of the virus except in eastern Turkey.

A spectacular illegal outbreak occurred in 1920 when a cargo of infected zebu was sent from India to Brazil via Antwerp. The disease broke out in Belgium and Brazil. This is the only known episode of RP in the Americas. The cattle from India had originally been refused transit at Marseille and were taken to Antwerp, where they were put into quarantine sheds. This proving to be an unsuspected route of infection, France called an international conference to discuss RP and other contagious diseases, as a result of which the *Office International des Epizooties (OIE)* was created with its headquarter in Paris. A second spectacular outbreak occurred in Australia in 1923. The origin was never established, but suspicion fell upon pigs that were live provision

bought in Singapore. The surviving pigs were sold to a butcher in Freemantele. The outbreak was stamped out within days by slaughtering 3,000 cattle, pigs, sheep and goats. Since then Australia has remained free of RP (Spinage, 2004).

Meantime, in India Dr. J. T. Edwards (1928) obviated the major risk in the anti-serum – virus simultaneous method of immunization of transmitting other pathogens in the virulent virus inoculum. He passaged a bovine strain of RP virus serially through goats to "fix" it and, fortuitously, produced a stable goat-adapted virus virulent for goats and yet attenuated for cattle. Anti-serum simultaneously was not necessary anymore: one dose of goat-adapted vaccine inoculated into a yearling calf protects it for life, but the goat-adapted vaccine was far from being the ideal. Vaccinated animals developed fever that lasted for 4-5 days. Dr. Junji Nakamura visited the Indian Veterinary Research Institute in the 1920s and was impressed by Edwards's idea on passaging a virus serially in a foreign host. When he returned to Korea, he passaged his local strain of RP serially in rabbits, goats being few in Korea. His Nakamura III strain was considered "fixed" after 600 passages and it proved to be more attenuated than Edwards's goat vaccine. This lapinised vaccine was used first in Mongolia and later in China. Nakamura forwarded a sample of his strain to the Kenya Veterinary Department in 1948 which was passed onto the new East African Veterinary Research Organization. Two years later Spinage was producing lapinised vaccine which was used in Kenya to immunize only pure-bred and grade cattle (Spinage, 2004).

Tentative steps at mass vaccination to control RP were taken in China in the late 1940s and in Equatorial Africa in the 1950s. India initiated a national eradication scheme in 1954 using goat-adapted vaccine. Over the first few years the success was remarkable; outbreaks fell from thousands per year to a few hundred per year. In the

first 10 years, 117 million cattle and buffaloes were vaccinated out of a population of 132 millions; as a result two-thirds of the country was free from RP.

In 1952 an Inter-African Bureau for Epizootic Disease was created by the Commission for Technical Disease in Africa south of the Sahara specifically to study RP. Dr. W. G. Beaton was the first Director and he initiated Joint Project 15 (JP 15) to rid Africa of RP. JP 15 operated in the field between 1962 and 1976 in 22 African countries. In 1962, 17 had active RP whereas in 1976 only 2 countries reported the disease. The goat-attenuated vaccine was replaced by Plowright's new cell-cultured virus vaccine in the 1960s.

Vaccination campaigns brought RP under control in most of the affected areas worldwide, and by 1970, the disease was restricted to parts of tropical Africa and parts of Asia, particularly India and Nepal, but in Africa soon after the termination of the JP 15 in 1976 the gains of the intervention were tragically undone. A virulent virus focus was missed in southern Sudan. It spread south through Uganda in cattle looted by the victorious Tanzanian troops. Meantime, a hidden focus in the Niger suddenly erupted and spread across the Sahel. One third of the cattle of the Fulani nation died. In Nigeria alone 2 million fell sick and half a million perished. In 1979 the situation deteriorated not only in Africa but also in the Near East when the disease spread to Lebanon, Syria and the Gulf countries as a result of increased movements of slaughter stock from the enzootic zones of Africa. In 1982 RP broke out again in Iran and Turkey and, later that year in Syria. An intensive vaccination campaign in Turkey prevented its further spread. At the same time RP was also present in India, Nepal and Kampuchea. In 1987, Sri Lanka became infected after 40 years of freedom where it was again eradicated in 1998. In 1987 a fresh Pan-African Rinderpest Campaign

(PARC) began field operations simultaneously in 34 countries and a similar initiative was undertaken in the Asian countries.

In 1992, the 106th FAO Council authorized the Director-General to establish a priority programme known as the Emergency System for Transboundary Animal and Plants Pests and Diseases (EMPRES) (see also Section 1.5.). The initial focus was the Global Rinderpest Eradication Programme (GREP), which considered the final eradication of RP from the world achievable by the year 2010 (Spinage, 2004).

GREP was severely tested when launched at the end of 1994 by two novel epidemics; the first was caused by a rare lineage II strain of the virus that appeared to be virulent in wildlife but undetectable in domesticated cattle (Barrett, 1996) (see also Sections 1.4. & 2.4.). A second novel epidemic was in yaks and cattle in Pakistan in 1994. Illegal cross-border movements of calves placed cattle at risk in Iran and Turkey. In 1995 rustled cattle were infected in Turkana, Kenya. Turkey had a minor outbreak in 1996. Yemen experienced sporadic outbreaks until 1996, and a persisting endemic infection was suspected, while the status of Saudi Arabia was uncertain. A serious outbreak occurred in 1998 in the Amur Region of Russia. This outbreak was totally unexpected because the Russian Federation was considered to be free of the disease. The source is still unknown. At the end of 1998, it was believed that RP outside Africa remained present only in Pakistan and potentially in Afghanistan. By the end of the year 2003 RP was believed to remain in only one site in the world, among the Somali nomadic cattle herds of southern Somalia, north eastern Kenya and southern Ethiopia: the so-called Somali eco-system (Spinage, 2004).

2.2.2. Etiology and replication

For want of a better term, in 1865, RP was classed as a "zymotic" or fermentative disease, a category introduced by Farr in 1842. Many veterinary authorities in Germany asserted for more that 50 years to the mid-19th century that RP was the precise counterpart of abdominal typhus or enteric fever of man (Ravitsch, 1864), an opinion so widely supported at the beginning of the 1865 outbreak in Britain due to reports of veterinarians and others who were not familiar with medical cases that it was almost a matter of popular belief. Others believed it to be identical with typhus, based on a supposed analogy between the etiologies: the fact that both were very contagious. Dr. James Tucker, in a report to the Lord Lieutenant of Ireland, stated that *"The purple gum, the black, saltless blood, and some other symptoms of the African typhus, may be recognized in the Rinderpest"* (Tucher, 1866). In France, it was called the *"typhus contagieux"*.

Its current classification is in the Order Mononegavirales of negative-sense, single-stranded, RNA viruses in the Family Paramyxoviridae, Subfamily Paramyxovirinae, and Genus *Morbillivirus*. The genus is a clade of six viral species descended from RP virus. The other related species are peste des petits ruminants (PPR), a disease of sheep and goats, cetacean morbillivirus, canine distemper (in Africa canine distemper has recently undergone a mutation and has infected and killed 3,000 lions in the Serengeti's area), and phocid distemper, measles of humans and New Castle disease of poultry. They differ in their natural host range but otherwise all look alike, have similar physicochemical properties, share antigens, and express identical epidemic patterns characterized by devastating dieback epidemics and pandemics when they infect susceptible unchallenged host populations.

Believed to represent the original or archaevirus of the group, some consider that RP may have evolved with ancestral Bovidae in the Pleistocene. Measles virus was the first descendant, emerging about 5000 – 7000 years ago when humans began to associate together in riverine communities and domesticated cattle. canine distemper virus followed measles, and lastly, the PPR virus split off within the past 3000 – 7000 years or so (McCullough *et al.*, 1986).

Polymerase chain reaction (PCR) based molecular sequence analysis, has enabled grouping of recent strains of the RP virus into three distinct lineages, one Asian and two African. Historically, five lineages could be distinguished, but only two were represented in outbreaks after 1983 (Chamberlain *et al.*, 1993; Wamwayi *et al.*, 1995).

The common shape of the RP virus is a spheroid 100 – 300 nm in diameter, containing tightly coiled serrated nucleocapsid. Less common are filaments up to 1 μm in length with regularly coiled serrated nucleocapsid. Both forms are encased in protein envelopes bristling with minute projections (Scott *et al.*, 1986). In 1995, the entire genome sequence of the virus was described, 15,881 bases in length, similar to that of the measles virus and slightly longer than that of canine distemper (Baron & Barrett, 1995). There are 6 genes separated by conserved noncoding sequences that contain termination, polyadenylation and initiation signals. Most of the gene products are structural proteins found in virions. The peplomers are composed of two glycoproteins: a hemagglutinin-neuraminidase protein (HN) and a fusion protein (F). Both proteins play a key role in the pathogenesis of the RP virus. Cell attachment is mediated via the HN protein. This glycoprotein elicits neutralizing antibodies that inhibit the adsorption of virus to cellular receptor. The F protein is present on newly formed virions in an inactive form that is activated by proteolytic cleavage by a

cellular protease. After cleavage, the newly generated amino-terminal sequence of the protein has a hydrophobic domain, and it is postulated that this is involved directly in fusion.

Syncytium formation in cell cultures and *in vivo* is a characteristic feature of infection. The RP virus replicates within the cytoplasm. Virions attach via their HN protein to cellular sialoglycoproteins or glycolipid receptors. The F protein then mediates fusion of the viral envelope with the plasma membrane, at physiological pH. The liberated nucleocapsid remains intact, with all three of its associated proteins (N, P and L) being required for transcription by the virion-associated, RNA-dependent RNA polymerase transcriptase. The genome is transcribed progressively into 6 discrete unprocessed mRNAs by sequential interrupted synthesis from a single promoter. Full genome-length positive-sense RNA is also synthesized and serves as a template for the replication of negative-sense genomic RNA. Control of these processes is mainly at the level of transcription.

Virion maturation involves (i) the incorporation of viral glycoproteins into patches on the host cell plasma membrane, (ii) the association of matrix protein (M) and other non glycosylated proteins with this altered host cell membrane, (iii) the alignment of nucleocapsid beneath the M protein, and (iv) the formation and release via budding of mature virions (Murphy *et al.*, 1999).

The RP virus is extremely labile to the effects of heat or desiccation and is destroyed easily by common disinfectants and lipid solvents. As long as putrefaction has not begun, meat and other products can remain infective for a relatively long time (Spinage, 2004).

2.2.3. Pathogenesis

Experimental infections can be established by all routes of parenteral inoculation and, more variably, by intranasal or conjunctival installation. Natural infection usually occurs through the mucosa of the upper respiratory tract after aerosol exposure, multiplying after several hours in the pharyngeal and submaxillary lymph nodes and tonsils, targeting epithelial cells of the alimentary, respiratory and urogenital tracts; provoking fever, stomatitis, rhinitis, and extensive gastroenteritis, producing a loathsome stench (Taylor *et al.*, 1965; Plowright, 1982). Another way of natural infection is via the oropharynx after ingestion of infected material (Curasson, 1932; Hall, 1933; Hornby, 1934; Plowright, 1964 & 1968; Scott, 1964; Todd & White, 1914). Seldom occurring free in the plasma, the virus has a selective affinity to lymphocytes and the epithelium of the mouth and alimentary tract (Daubney, 1928; Plowright, 1968; Schein, 1917; Todd & White, 1914). Destroying the superficial epithelium, the entire alimentary tract is affected by erosion of the mucous membrane. Attaching firmly to mononuclear lymphocytes, it destroys the lymphoid tissue, particularly lymphocytes, which suppresses the normal defense mechanism of the host, allowing other latent infections (such as coccidiosis, theileriosis, bebesiosis, piroplasmosis and trypanosomiasis in African animals) to express themselves. Initially, lymphocytes numbers rise to combat the infection but, after 4 days, decline to some 18% of normal at termination of the disease; leukocytes decline to some 28% of normal. An almost total destruction of lymphocytes in spleen and lymph nodes in some animals, especially calves, causes rapid death without the characteristic gross lesions occurring (Maurer *et al.*, 1955). There is an inverse relationship between increasing attenuation and the degree of viral multiplication in lymph nodes of cattle (Scott, 1964). Virulent strains of RP Virus have a greater ability to infect lymphoid

cells and mononuclear phagocytes and may grow to higher titers in these cells than do strains which include mild disease (Scott, 1982).

The cell attenuated variant of the Kabete 'O' strain of RP virus, which is the most commonly used vaccine, only produces low levels of infectivity in lymphoid tissues and is barely detectable in the blood (Taylor & Plowright, 1965). These low levels of viraemia are probably one reason why attenuated and very mild strains cause so little epithelial damage.

The virus has a predilection for T lymphocytes and attains higher titers in the T4 and T8 subsets of the T cells than in lymphoblasts of B or null cell origin (Rossiter *et al.*, 1988; Rossiter & Wardley, 1985). During disease the virus is also found in non mucosal organs, such as the lung, liver and kidneys (Boynton, 1917; Daubney, 1928; Leiss & Plowright, 1964; Taylor & Plowright, 1965; Taylor *et al.*, 1965; Todd & White, 1914). Immunofluorescence has shown that antigen-bearing cells in these organs are usually associated with reticuloenothelial and perivascular connective tissue (Rossiter & Jessett, 1982a).

The severity of cytopathology caused by the virus before the onset of antibody development influences the course of the disease. Virulent strains cause severe lesions before being restrained by the immune response, and such animals, if sufficiently damaged, will still die despite high titers of antibody and low or undetectable amounts of virus (Scott, 1959; Scott & Brown, 1961).

2.2.4. Clinical manifestations

The clinical signs of RP have been extensively described (Bansal, 1986; Curasson, 1932 & 1942; Gamgee, 1866; Henning, 1956; Plowright 1968; Provost & Borredon,

1963; Sanderson, 1866; Scott, 1967 & 1981). The following description gives the basic signs in the sequence in which they usually occur in severely affected cattle.

Acute infection is characterized by an unusually rapid course, and signs of illness may be noted within 3 – 5 days of contact with the virus, although it may take 9 days. Inoculation can cause symptoms in 2 – 3 days. Pyrexia heralds the onset of clinical disease. The body temperature rises rapidly to 40 - 41.5 °C and this can be maintained for up to two weeks, though usually less, before gradually declining to normal. During the first one or two days of fever there may be depression or restlessness, a degree of inappetence and a reduction in milk yield. The visible mucosae are congested and serous oculonasal discharge, one of the characteristic signs of RP, becomes apparent. Between the second and fifth days of fever the mucosal lesions become obvious, initially as pin-point white foci on the gums and lips which spread rapidly to involve the buccal mucosa and ventral and lateral aspects of the tongue. The foci enlarge and collapse into plaques of caseous necrotic debris which desquamate easily, leaving circumscribed erosions. The halitosis is memorable. Similar lesions may be seen on the palate, posterior dorsum of the tongue, pharynx, and in the nares and vagina. Severe cases often drop fetid saliva, presumably because of discomfort in the mouth and pharynx when swallowing.

In cattle infected with strains of high virulence, diarrhea, sometimes preceded by an absence of defecation, starts four to five days after the onset of pyrexia; usually one to three days after mouth lesions become visible. The faeces are initially thin and dark but may progress to contain blood, mucus and shreds of necrotic epithelium. The hind quarters are fouled, and tenesmus with eversion of the rectal mucosa is frequent. The

diarrhea causes dehydration, weakness and prostration, and in severe cases abdominal pain is evident. Recumbent animals face their flanks in typical "milk fever" posture.

The oculonasal discharge becomes increasingly purulent during the course of the disease, and the conjunctivitis causes photophobia. Corneal opacity is rare in cattle, but has been recorded in some species of wildlife such as giraffe, buffalo and kudu (Shanthikumar *et al.*, 1985). Epiphora is evident below the medial canthus (Henning, 1956).

Early descriptions of RP frequently described skin lesions (Nocard & Leclainche, 1896; Sanderson, 1866). Most recent accounts (Curasson, 1942; Plowright, 1968; Scott, 1964) regard these as rare, though they are sometime present in domestic buffaloes, sheep and goat. The lesions appear as maculo-papular rash on areas of soft skin such as the axillae and groin, where the hair may become matted when the rush eventually becomes pustular and breaks open (Bansal, 1986; Curasson, 1932; Joshi *et al.*, 1977; Mohan & Bahl, 1953; Mornet & Guerret, 1950).

Although respiration may be labored during acute clinical disease, pulmonary lesions are uncommon and are mainly due to secondary bacterial infections. Emphysema of the lung may occur terminally. Lymph-adenopathy is not usually evident even on palpation. Milk may become watery or dry up. Pregnant animals frequently abort, sometime weeks or months after clinical disease (Curasson, 1932; Jacotot, 1931; Nocard & Leclainche, 1896; Wafula *et al.*, 1989). Most animals that succumb to RP die 5 to 14 days after the onset of pyrexia.

Convalescence is marked by the rapid healing, within two to six days, of mucosal lesions, starting three to six days after their appearance. Diarrhea diminishes more slowly and may persist in a less severe form for up to two weeks. Complete recovery

from severe RP takes one to two months or more, depending on how much condition the animal has lost on the quality of subsequent nursing.

Since RP virus destroys lymphoid tissues, infected animals suffer from a variable degree of immunosuppression. In Africa this frequently leads to the reactivation of a variety of chronic or latent diseases, especially those caused by haemoparassites (Curasson, 1932; Ghaffar & Ata, 1982; Holmes, 1904; Hornby, 1934; Provost & Borredon, 1972; Réfik-Bey & Réfik-Bey, 1899; Scott, 1964 & 1967).

In endemic areas the disease is milder than the above description (Curasson, 1932; Lowe *et al.*, 1947; Plowright, 1963a; Provost & Borredon, 1963; Robson *et al.*, 1959). Most of the signs are much reduced in severity, and some may be absent. Experimental infections with such strains have shown that they may cause no death, no diarrhea, transient mouth lesions in only a proportion of infected *Bos taurus* cattle, and only transient pyrexia in *Bos indicus* cattle (Plowright, 1963a; Robson *et al.*, 1959; Taylor, 1986).

2.2.5. Pathology

A proportion of infected cattle shows lymphocytosis before the onset of pyrexia. This is followed by marked lymphopenia, caused by lymphoid necrosis, which in most cases lasts throughout the acute clinical stage of the disease (Baldrey, 1906; Heuschele & Barber, 1963; Maurer *et al.*, 1956; Réfik-Bey, 1902; Robey & Hale, 1946; Thiéry, 1956b). During convalescence, lymphocyte levels slowly return to normal over a period of days or weeks. The number of neutrophils remains relatively unaltered, though juvenile forms are not infrequent during the terminal stage of fatal infection. However, a degree of neutropenia that parallels the decline in lymphocyte

levels has been reported (Thiéry, 1956b). Eosinophils may also disappear from the blood during the early stages of clinical disease, returning to normal levels some two to three weeks later. In severe cases the excessive loss of water causes hamoconcentration (Baldrey, 1906).

Serum aspartate transaminase and blood urea nitrogen levels increase during severe cases of disease (Bhattacharya & Chakraborty, 1979; Heuschele & Barber, 1966). Serum chloride levels fall markedly in terminal illness, and other electrolytes may decrease in absolute terms although this can be masked by hamoconcentration. Blood clotting may be impaired in severely affected animals. Serum protein levels may be lowered, especially in fatally infected animals (Curasson, 1932; Dhir *et al.*, 1987; Gibbs, 1933). In cattle recovering from experimental infections a rise in serum globulin was attributed to the specific humoral response to the virus (French, 1936), but since the challenge material was citrated blood, this may need re-interpretation in the light of known responses to heterologous tissue antigens.

The lesions of RP are a direct result of virus-induced cytopathology. There are many descriptions of the gross pathology, (Bansal, 1986; Bristowe, 1866; Curasson, 1932; Gamgee, 1866; Jacotot & Mornet, 1967; Scott, 1964), but those of Maurer *et al.* (1955, 1956) are especially valuable. Generally, the severity of the lesions is directly related to the virulence of the strain of virus involved (Thiéry, 1956a). Complications may arise during convalescence through reactivation of latent pathogens, especially protozoa (Curasson, 1932; Holmes, 1904; Scott, 1967).

The overall appearance at necropsy is similar for most species that die of typical RP. The carcass is dehydrated, sometime emaciated, and usually soiled with fluid faeces.

The eyes are sunken and often encrusted with mucopurulent discharge and cheeks may show signs of epiphora.

Erosions with or without necrotic material may be found throughout the mouth, but predilection sites are the gums, lips, buccal papillae, dorsal and ventral aspects of the tongue and the soft palate. The erosions often extend into the pharynx, anterior esophagus, rumen (especially the pillars), the reticulum and omasum. Necrotic areas, some of which may penetrate the leaves of the omasum, are sometimes present. The folds of the abomasum are congested and edematous and often show necrosis, erosions and hemorrhages along the edges. The fundus of the abomasum may have small discrete erosions which increase in size toward the pylorus, where whole areas of mucosa may become desquamated. The early necrotic lesions are pale-grayish, whereas the erosions are often red as a result of congestion of the underlying lamina propia. Hemorrhages may occur from the raw surface. The abomasoum is almost invariably severely affected, whereas the small intestine frequently shows less involvement. Congestion, edema and erosions may occur on the margins of the mucosa folds of the anterior duodenum and terminal ileum.

The Peyer's patches, being lymphoid tissue, are severely affected and are swollen, dark red to almost black as a result of hemorrhage, and may slough completely leaving deep ulcer-like areas. Large erosions are commonly found on the ileocaecal valves. In the large intestine, marked edema and congestion accompanied by petechiae or large hemorrhages occur, particularly along the crests of longitudinal folds of the mucosa. This can be very striking in the colon and rectum, meriting the description 'zebra striping'. In acute cases, the gut has little content other than

desquamated necrotic epithelium, blood, and fibrin exuding from exposed lamina propria.

The urinary gall bladders are frequently congested and hemorrhagic with occasional erosions. The vaginal mucosa may be congested and have small erosions.

The mucosa of the upper respiratory tract, including the larynx, is congested and usually covered with muco-purulent secretions. Petechiae are frequent and necrotic; erosive lesions may extend from the nares to the larynx. The tracheal mucosa is frequently congested. Congestion and emphysema may be seen in the lungs, while secondary bronchopneumonia may complicate chronic cases. Although RP virus has a predilection for lymphoid tissues, there are usually few visible changes to the superficial and visceral lymph nodes. These may show congestion, edema, and few petechiae. The nodes of animals that die after a prolonged clinical course may be shrunken and may show grayish radial streaks in the cortex, presumably due to hemorrhages (Bansal, 1986). The spleen and haemolymph nodes appear normal or slightly enlarged.

The essential histopathology of RP was first described in infected rabbits (Brotherstone, 1951; Fukusho & Nakamura, 1940). Widespread necrosis of lymphocytes is apparent throughout the lymphoid tissues, together with syncytia and intracytoplasmic and, less frequently, intranuclear inclusion bodies. The histology in cattle is similar (Khera, 1958a; Maurer *et al.*, 1956; Thiéry, 1956b) with lytic destruction of lymphoid tissues that is most evident in the germinal centers, sometimes accompanied by an increase of the number of macrophages. In acute cases lymph nodes are virtually devoid of cells, with just a reticular stroma containing eosinophilic material remaining. The earlier epithelial lesions in the squamous

epithelium of the digestive tract are associated with the formation of syncytia and eosinophilic intracytoplasmic inclusions in the *stratum spinosum* (Khera, 1958b). Infected epithelial cells become necrotic and slough off, leaving clearly demarcated erosions. The erosions heal rapidly unless complicated by secondary infections which may rarely cause them to ulcerate. Changes in other tissues are not remarkable, although Khera (1958c) described small foci of necrosis in the liver. Unltrastructurally, infected cells contain large parallel arrays of tubular nucleocapsid material which are probably the intranuclear and intracytoplasmic inclusion bodies seen by light microscopy (Breese & De Boer, 1963; Provost *et al.*, 1965; Tajima *et al.*, 1967).

2.2.6. Immunity

Cogrossi recorded an example of immunity in cattle in 1711: *"I have just been told that in Montodine, a large village in this territory, there is in the midst of the infected animals a cow which has not suffered at all, and that all her sons, though they are in other infected stalls, have been immune for all illness"* (Cogrossi, 1714). This was the first recognition in any species of the phenomenon of maternally derived immunity, although Cogrossi postulated that if the theory of contagion by invisible animalcules was correct, this could be explained easily by the fact that *"these poisonous insects find in oxen alone food adapted to their subsistence and that in certain oxen they do not flourish for the same reason that in certain men the prurient worms of the itch cannot subsist"*. Brocklesby (1746) observed that *"cattle might develop immunities so that one man's herd would remain whole in the midst of the disease, which, like the plague, moved west, that is from London"*. This statement on immunity preceded by far that of Girard and Dupuy (1816), who reported that an infection conferred

immunity. Galtier believed that the immunity of calves delivered by dams infected during gestation was inherited. This was disputed by Hornby (1926) in Tanganyika, who correctly concluded that immunity was acquired only during an animal's lifetime.

In populations subjected to the virus, most adult cattle are immune, and the calves are protected by maternal antibodies in the antibody-rich colostrums which they ingest within the first few hours of birth. The amount of gamma-globulin in milk falls off rapidly after parturition, and a calf's capacity to absorb the large molecules through the gut wall wanes after a few hours. Protection conferred by maternal antibodies last for varying lengths of time according to species. In cattle, it is effective for 4-8 months with a half life of 48 days, 7% of calves in tests still having maternal antibodies at 8 months, and none after 8 months. After this period has elapsed, the calves become susceptible, so that if the host population is large enough, infection can, theoretically, constantly smolder (Spinage, 2004).

In case of infection, virulent strains of RP virus are excreted from epithelial tissues one or two days before the appearance of fever or lesions (Leiss & Plowright, 1964), but the amount of excreted virus increases dramatically as the lesions develop and only starts to decline when the immune response become detectable some four to six days after the onset of the fever. The virus is usually undetectable by 12-14 days after the start of fever. At the height of virus excretion, three to six days after the start of pyrexia, virus titers up to 10^5 tissue-culture infectious dose ($TCID_{50}$)/swab and up to 10^6 $TCID_{50}$/g respectively can be found in nasal secretions and faeces from cattle infected with virulent strains (Leiss & Plowright, 1964). This copious output of virus explains why the disease can be so contagious despite the fragility of RP virus. The

diarrhea and ocular discharge probably help to increase the transmissibility of the virus by forming infectious aerosol, and by causing greater contamination of the environment.

Infected animals mount a vigorous response against the virus. Interferon is produced within two days of infection, enabling attenuated vaccines to protect cattle very rapidly against challenge by virulent virus (Hussain *et al.*, 1982; Watanabe, 1970; Wilde & Scott, 1961). Viral antigens are produced in large amounts throughout the lymphoid tissues and affected epithelia (Nakamura, 1957; Rossiter & Jessett, 1982b; Scott, 1967; Scott & Brown, 1961) and stimulate an effective antibody response which begins two to five days after the onset of clinical disease in virulent infections, and some six to ten days after infection with mild or avirulent strains (Johnson, 1962; Nakamura, 1940; Plowright & Ferris, 1959; Scott & Brown, 1958; Walker *et al.*, 1946). The earlier response consists predominantly of IgM antibodies (Anderson *et al.*, 1982; Okuna & Rweyemamu, 1974) which can be detected by virus neutralization (VN), ELISA, and also, for a period of a few months, by immunoprecipitation, complement fixation and measles virus hemagglutination inhibition (Rossiter & Wamwayi, 1989; Scott *et al.*, 1986). At the same time IgG antibodies are produced, but these persist for much longer, usually for life (Plowright, 1984; Plowright & Ferris, 1962b; Rweyemamu *et al.*, 1974) and are usually measured by VN or ELISA tests (Anderson *et al.*, 1983; Rioche, 1969; Rossiter & Jessett, 1982b; Rossiter *et al.*, 1981; Scott & Brown, 1958; Sharma *et al.*, 1983). Generally high titers (10^2-10^3 $\log_{10}$ VN_{50}) of neutralizing antibodies are produced within two to three weeks of infection and remain high for several months, after which they may decline slowly, but usually remain at easily detectable levels (in excess of 10^1 $\log_{10}$ VN_{50}) for the rest of the animal's life (Plowright, 1984; Plowright & Ferris, 1962a; Plowright & Taylor, 1967).

Rarely, neutralizing antibodies decline to very low or undetectable levels, but such animals are clinically immune, although limited replication of the virus may occur in tissues, such as the tonsils, before the stimulation of an anamnestic response (Plowright, 1984; Plowright & Taylor, 1967; Provost *et al.*, 1969; Rweyemamu *et al.*, 1974; Zwart & Macadam, 1966). The antibody response of naturally infected cattle and those vaccinated with live virus vaccine are indistinguishable.

A study with ELISA showed the development of serum IgA antibodies in cattle after vaccination (Anderson *et al.*, 1982). However, another study using indirect immunoperoxidase failed to detect IgA antibodies and, in contrast to most other studies in cattle, ascribed most of the IgG antibodies to the IgG_2 subclass. Secretory antibody is found in nasal secretions of convalescent cattle (Provost, 1970), but its persistence is presumably limited to only a few months after recovery, and the role it plays in preventing reinfection is unknown.

The severity of the cytopathology caused by the virus before the onset of antibody development influences the course of the disease. Virulent strains cause severe lesions before being restrained by the immune response, and such animals, if sufficiently damaged, will still die despite high titers of antibody and low or undetectable amount of virus (Scott, 1959; Scott & Brown, 1961). The persistence of immunity in recovered animals and those given by live virus vaccines contrasts with short-lived immunity induced by inactivated vaccines (Jacotot, 1950; Scott & Brown, 1958), thus implying that recovered animals may be continually re-stimulated immunologically by RP virus antigen throughout their life. Circulating or fixed immune-complexes have not been demonstrated in RP but might occur, considering the rapidity with which the mass of viral antigen is eliminated by the specific humoral response. There

is a transient autoimmune response in infected rabbits which produce an IgM (19S) hemagglutinin to homologous erythrocytes and an IgG (7S) anti-nuclear antibody (Fukuda & Yamanouchi, 1981).

Very little is known about the cell-mediated immunity (CMI) in RP. However, there is a school of thought which suggests that CMI is the most important mechanism for long term protection against RP (and other morbilliviruses) reinfection. This is based on the observation that some vaccinated animals that do not develop detectable humoral responses are protected against RP when challenged with virulent virus even after several years. A few experiments (Yamanouchi *et al.*, 1993) have been carried out in order to test the efficacy of the H and F glycoproteins of the RP Virus (RPV) expressed by recombinant vectors as subunit vaccine for RP. Despite the inoculation of the subunit vaccine induced the production of well detectable humoral response the animals were not protected against a challenge with a virulent strain of RPV. The rationale of using subunit vaccine was to reduce the risk of using infectious Recombinant Vaccinia Viruses (rVVs) especially in relation to the concern of the HIV epidemic in Africa. The infectious rVVs for RP were proven to be efficacious in protecting cattle against challenge with virulent strains. Moreover the use of whole inactivated virus vaccines for RP in another experiment produced detectable humoral response but very poor protection. One of the hypotheses that was ventured to explain why subunit vaccine and whole inactivated vaccine were unable to protect the animals against a challenge with virulent strains despite the induction of well detectable humoral response was that these types of vaccine were unable to stimulate an appropriated CMI.

In another experiment (Yamanouchi & Barrett, 1994), a rVVs expressing the N protein of RPV was produced to assess its stimulation of the immune system and its ability to protect against challenge with virulent and a mild strains of RPV. The recombinant vaccine induced low levels of non neutralizing anti-N antibodies but RPV specific cell mediated response. The protective strength of the vaccine was examined by challenging the vaccinated cattle with a virulent and a mild strain of RPV. No protection was observed against the virulent strain, while protection was observed against the mild strain, and all control animals developed RP virus. It was noticed that neutralizing antibodies were produced more quickly in the vaccinated animals challenged with the mild strain than the control group. These results could suggest that the CMI induced by the rVVs – N could stimulate the rapid production of neutralizing antibodies after challenge. However, this response was probably not "quick enough" to protect against challenge with virulent strains.

2.2.7. Transmission

The ease with which the disease is transmitted depends upon the strain of the virus. Infected animals are infectious before the end of the incubation period of the disease although still apparently healthy, and they remain infectious into the start of the convalescence (Scott, 1955). Some infected animals do not sicken but are nevertheless infectious to others. It had long been observed that a damp atmosphere favored propagation, and transmission is believed to be by droplet, either in the breath of an infected animal or in its secretions and excretions, but close contact is required lest the virus is destroyed in passage.

Réfik-Bey and Réfik-Bey (1899) concluded that transmission was by direct contact, and the role of soil and water seemed to be nil, while insects also did not appear to be

involved. At the beginning of the 18th century, Carlo Francesco Cogrossi, a distinguished Italian medical teacher, asserted that the first attacks of the disease had been observed mainly in the respiratory and digestive systems, quoting Lancisi as holding the correct opinion that *"the poisonous fermentation was introduced, more than in other ways, through the fauces and nostril"* (Cogrossi, 1714); over two centuries before Hornby (1926) confirmed that it was most easily transmitted through the nostril and concluded that the most common manner by which the disease was spread was probably by inhalation of infective excretion. This way of transmission is still considered the most common till today.

It has been known for over a century that the virus can retain its infectiveness for a long time if kept from contact with the atmosphere and high temperature. In 19th-century studies in Russia, carefully protected mucous discharge had kept their infectivity for periods of 7 to 11 months. In recovered animals, the virus is excreted in milk for up to 45 days (Curasson, 1932).

Fleming (1875) stated that it had long been recognized that the dung of infected animals was a potent agent in diffusing the disease and appeared to keep its infective properties for several months. While the former would be true, virus being shed in the dung, it is unlikely to survive for long in that environment. Although large amounts of virus are passed in the urine and feces, rarely can transmission be attributed to contaminated bedding, fodder, or water, although urine, which does not become infective up until about the third day of fever, can maintain its infectiveness for 9 days. Instances in which the disease has been propagated by the flesh of diseased animals are well known. Fresh hides are also another common source of spreading infection and thought to be a particular cause in India (Spinage, 2004).

Adam Neale, a physician, introduced the idea of insects effecting the mechanical transmission of disease in 1831, suggesting that both human plague and RP were transmitted by this means. In Galicia in 1846, where the disease was well known, it was believed that it could be transmitted, in spite of quarantine, by flies passing from one animal to another, communicating it to the eyes and tender parts of the sound animal. Gamgee (1866) was firmly of the opinion that flies could transmit it: *"One way in which the Cattle Plague may be carried from diseased cattle or from the slaughterhouse is unquestionably by flies, which, after resting on the carcass or offal of sick animals, fly about, rest again on the animal, especially on any wounded part, and thus produce a direct inoculation ... Unaccountable outbreaks may sometimes be due to this cause"*. Although this was early thinking on the disease-carrying capabilities of the fly, its role in RP transmission has not been demonstrated. A greater likelihood of mechanical transmission is provided by ophthalmotropic moths which are attracted to the eyes of animals for their moisture requirements. In the Sudan, Reid (1954) found that moths of the genus *Arcyphora* were attracted to the discharge from the eyes of cattle suffering from RP.

In 1993-97 in Kenya, the conjunctiva of the lesser kudu appeared to be the point of entry of the virus, possibly occasioned by mechanical transmission of dipterous insects of which there was an epizootic of "head fly" (Kock *et al.*, 1999), presumably *Lyperosia* or *Siphona* species. Once the animal begins to weep, such diptera are attracted to the site and pass from one animal to another. However, Hornby (1926) was unable to transmit the virus by swabbing the conjunctiva of an ox with lachrymal discharge of a beast dying of acute infection, but another, swabbed with liver extract, did react.

Pigeons, rats, dogs, and hares have all been implicated in spreading the disease in Europe. Simonds (1866) believed that it was spread by domestic pigeons, particularly in Yorkshire. Vallisnieri wrote to Lancisi in 1715 that dogs carried the infection from one area to another (Spinage, 2004). But a study in India reached the conclusion that its spread by bloodsucking insects, leeches, vultures, and other carrion feeders was minimal (Minett, 1954).

In Africa, a continent populated with large numbers of potentially susceptible artiodactyls, the wildebeest *Connochaetes gnu* and African buffalo have been shown to be the most important transmitters, due to a potentially high degree of susceptibility and close contact with cattle on grazing lands and at watering points (Wilde, 1953). The importance of other species lies more in their capacity to act as contributors to the disease reservoir.

2.2.8. Susceptible species

RP virus infects a wide variety of vertebrates. Some of these, including rabbits, hamsters, mice, giant rats (*Cricetomys gambianus*), ferrets, and susliks (*Citellus mangolicus ramosus*) are usually only infected experimentally, and then often only by using strains of virus adapted to them (Curasson, 1942; Inoue *et al.*, 1930; Nakamura *et al.*, 1938; Plowright, 1968; Scott, 1964; Scott & Witcomb, 1958). Artiodactyls are naturally infected, although dogs fed with infected meat may develop antibodies to the virus, suggesting subclinical infection (Polding & Simpson, 1957).

Among domestic animals, cattle and buffaloes (*Bubalus bubalus*) are especially susceptible and are more frequently infected than other species. There is a big variation in susceptibility to clinical disease between breeds or races of cattle

(Holmes, 1904; Keylock, 1933; Sanderson, 1866; Scott, 1963; Varnell & Pritchard, 1866). Most European cattle breeds (*Bos taurus*) are more susceptible than *Bos indicus* breeds. African humped cattle, such as the Ankole in East Africa, are notoriously susceptible in comparison to East African Zebus (Brotherstone, 1951; Brown *et al.*, 1955; Cornell & Evans, 1937; Henning, 1956; Mornet, 1948; Scott, 1963).

Because Japanese black cattle reacted so severely to goat-adapted vaccines that were sufficiently attenuated for other cattle, the virus had to be further attenuated in rabbits and embryonated chicken eggs (Nakamura & Miyamoto, 1953; Nakamura *et al.*, 1938). Since a detailed description of RP in cattle has already been provided, this section will focus on the most susceptible species other than cattle.

2.2.8.1. Camels

The camel has proven to be a controversial species but is one which could have played a role in introducing the disease into sub-Saharan Africa. In Egypt in 1861, an immense number of affected camels allegedly succumbed to RP. Verdernikof (1893) in Russia considered that the camel showed the same signs as cattle, with an average 5 days' incubation followed by a rise in temperature to 42 °C, the breathing rate increasing to 50 per min., and an accelerated pulse rate of 90 beats per min. There was intense conjunctivitis, sometime with keratinization. Constipation was followed by diarrhea, irregular breathing, and a cough. The temperature fell near the end, and average mortality was 95%.

Its susceptibility was also allegedly demonstrated by Pease in 1894. But camels were reported as unaffected in an outbreak in 1898 in the Turkish Province of Aïdan. Koch

was unable to infect a dromedary experimentally, but Tartakovskii (1899) in Turkestan inoculated two dromedaries and four Bactrian camels, the two presenting an insignificant reaction and the four symptoms of RP, and one dying.

Lingard (1905a) reported that experiments conducted in five camels of the United Provinces, India, showed them to be susceptible to inoculated bovine RP and vice versa. Haji (1932) described an outbreak in India among camels with a mortality of 20-40%, and Dhillon (1959) reported 60 of 128 camels dying of the disease in India. But in neither case was it confirmed by serology, only assumed from clinical symptoms. Curasson (1932) pointed out that when referring to the camel, workers rarely stated which camel was referred to, whether the single-humped *Camelus dromedari*, or two-humped *Camelus bactrianus*, but there did not seem to be any difference between them in their reaction to RP.

In the outbreak in northern Kenya in 1960, Scott and McDonald (1962) examined sera from 60 camels from outbreak areas but found no trace of RP antibodies. Yet, in 1967 Singh and Ata (1967) did demonstrate antibodies in 8-10% (n=194) of camels slaughtered in the Cairo abattoir from RP endemic areas, which included animals from Sudan (9.7%). These workers found that camels were susceptible to the virus experimentally, developing high levels of neutralizing antibody following inoculation with virulent virus but developed unapparent infections. However, an infected camel failed to transmit disease to a susceptible calf, but that is not to say that infection could not be transmitted given the right circumstances. The course of the disease might be different under the stress to which camels are frequently exposed, such as long desert journeys without water. The only clinical sign of infection was recorded as a transient fever which easily could be missed. The camel must therefore be regarded

as a possible source of transmission. Hamers-Casterman *et al.* (1993) demonstrated that the camel had an "amazing" immune system, producing immunoglobulins unique among mammals. It is this unique capacity which may explain their variable response to RP (Jassim & Naji, 2001).

2.2.8.2. Pigs

Pigs have been infected experimentally through eating infected raw meat. Whether the infection was contracted from nosing the meat beforehand, or from ingestion, is not clear (Plowright, 1968 attributes it to ingestion). European domestic pigs experience unapparent infections. In India, evidence of infection in pigs was considered strong, but inoculation failed to produce a reaction in them (Hallen *et al.*, 1871). Pease (1894) noted that the wild pig contracted it. In Asia, the domestic sway-back pig was affected, and in Sri Lanka, wild pigs are reported to have died from eating carcasses of infected cattle (Mahamooth, 1943). It was not until 1993 that there was the first confirmed report in European pigs in India, when an outbreak among Large White Yorkshire in Tamil Nadu resulted in the death of 40 of 56 infected. Theiler (1897) in South Africa and Angeloff (1917) in Bulgaria failed to obtain any reaction in pigs, although Theiler claimed that the passaged blood was infective to cattle.

Leblanc reported RP in peccaries in 1886 (Carré & Fraimbault, 1898), and Penning (1894) claimed to have infected wild boar. Pease (1894) stated that it had been observed in the wild pigs of Sumatra and transmitted experimentally in them. Carré and Fraimbault (1898) demonstrated in Indo-China in 1896, transmissibility from pig to pig by contact and by blood inoculation, also from bull to pig and pig to bull by inoculation. Pluning observed the disease in pigs in Sumatra, and in Southeast Asia it has been reported in wild pigs (Vittoz, 1954). In the Philippines, it had been noted

from many years prior to 1916 that, simultaneously with the appearance of RP in cattle and water buffalo, in certain localities pigs also become infected.

Pigs exposed to sick cattle in the laboratory in 1914 developed symptoms similar to the disease in cattle. Experiments by Boynton showed that cattle, water buffaloes, and pigs varied but slightly in susceptibility, and the disease could be transmitted practically as readily from one type of animal to the other as among each kind (Boynton, 1916). In Africa, the wild *Suidae* are affected, warthog (*Phacochoerus eathiopicus*), bushpig and giant forest hog (*Hylochoerus mainertzhageni*). Hippopotami (*Hippopotamus amphibius*) develop a subclinical disease (Plowright *et al.*, 1964).

2.2.8.3. Goats

Goats often die from what is attributed to RP in India but elsewhere are affected only sporadically. In some outbreaks, they remain unaffected, while in others, a large percentage may contract the disease. In Germany in 1744, a goat stabled with a cow which died of RP also succumbed (Bucard-Mauchert, 1745). But it was not until 1835 that there was the first confirmed observation of alleged RP in a goat. A Norfolk farmer gave evidence to the Commission in 1865 that his children's pet goat died of RP contracted from his oxen. The goat died within 3 days of being reported sick. Scott and Brown (1961) confirmed the spread of the virus from cattle to goats under field conditions in East Africa, and in the 1980s, antibodies were found in goats and sheep in southern Tanzania. Later, experiments conducted in Chad also transmitted the virus from goat to cattle and vice versa (Bidjeh *et al.*, 1997).

2.2.8.4. Sheep

Fracastro (1546) wrote of the plague sweeping not only *"the wretched cattle, but also nearly the whole of the unhappy flocks of sheep"*, which probably prompted Gamgee (1865) to allege that people had known *"from time immemorial"* that sheep would communicate the disease. Several observers in the 18th and 19th centuries, namely Sauvages, Jessen, Sergejew, and Paschkewitsch, had noted the susceptibility of sheep, and one writer in 1747 asserted that the disease could be spread by their wool. Dr. Kreutzer had first described the symptoms in sheep inoculated with virus from cattle (Spinage, 2004). In 1863, Professor Röll of the Veterinary Institute in Vienna, who, in his veterinary handbook published in 1856 had stated that only the ox and buffalo could be infected with RP (Röll, 1856), studied the disease in sheep and conducted experiments demonstrating its transmission from sheep to cows and showed that passage of the virus through sheep did not render it less virulent. In Sicily, 20,000 sheep and goats died of RP in 1863 and two months after the eruption in Britain in 1865, at least two outbreaks among sheep had occurred.

Various inoculation experiments showed that sometimes sheep took the disease and at others showed no apparent effects. Successful experiments were carried out in England and at the Inoculation Institute at Cherson, in Russia, where infected sheep and goats were found to be just as infective to cattle as if the virus had been transferred from cattle themselves (Spinage, 2004).

In 1973, there was an alarming increase in RP among sheep and goats in Kamataka and Tamil Nadu, India. Whereas there was some spread to cattle, it mainly affected sheep. Although this lack of spread to cattle might be partially accounted for by

vaccination programme, antibody tests showed that only some 31 – 68% of cattle were immune (Spinage, 2004).

Goats inoculated with an isolate once passaged in sheep showed a reaction with fever and diarrhea, but all survived. But after three passages in sheep, mortality in goats was total. In sheep, its course was rapid, possibly leading to low levels of replication of the virus and thus low levels of virus excretion; but although highly pathogenic to sheep, the strain was less so to buffalo and grade zebu cattle. Serial passage of the virus in sheep created an enhanced pathogenicity with a shortened period of incubation and an extension of the infective phase (Ramani *et al.*, 1974).

2.2.8.5. Other ruminants

Deer were reported to have died in the woods in Germany in an epidemic in 1744 (Bučard-Mauchart, 1745). Pease (1894) reported RP in India in the banteng, gaur, buffalo, muntjac, goral (*Naemorhedus goral*), "gazelle" and "stag". In south Asia, it has been reported also in banteng, gaur, buffalo, and smaller antelopes and deer (Vittoz, 1954). In 1923, Japanese workers demonstrated that sika deer (*Cervus sika*) were susceptible, noting that the period of inoculation in natural infection did not exceed 10 days, while in the laboratory it was 3-6 days. Serum was found to be curative when administered at the onset of fever and repeated 4 days later, the virus disappearing from the circulation within 6 days of recovery (Ono & Kondo, 1923). Jacotot (1927) obtained a very benign experimental infection in the sambar in Indo-China.

Dieckerhoff (1890) attributed the decline of the European bison (*Bison bonasus*) to RP. Buffaloes are particularly susceptible, whether wild or domestic, Asian, African

or European. Several African artiodactyls are also susceptible to the disease at different extent. The involvement of African wildlife in the RP African pandemic is described in detail in Section 2.3.5.

2.2.9. Epidemiology

Infected animals excrete infectious virus in their ocular, nasal, oral, vaginal secretions and faeces (Curasson, 1932; Hall, 1933; Hornby, 1926; Scott, 1964). Highest titers of virus are excreted during the earliest stages of clinical disease when epithelial lesions, especially those in the mouth, are developing to their maximum extent. Subsequently the titers of excreted virus wane as antibodies develop (Leiss & Plowright, 1964; Scott & Brown, 1961). Recovered cows may abort an infected fetus weeks after apparent recovery, with virus excretion in their uterine and vaginal discharges (Curasson, 1932; Jacotot, 1931; Wafula *et al.*, 1989). The fragility of the virus ensures that most infectivity survives for only a few hours outside the host, though some may persist under favorable conditions for up to two to four days (Shilston, 1917; Todd & White, 1914). Carcass decomposition inactivates the virus within one to three days (Curasson, 1932).

The spread of RP virus, therefore, is effected almost exclusively by contact between infected and susceptible animals. Transmission by infected aerosol probably occurs only under ideal conditions of close proximity and gentle air current (i.e. among housed animals) (Idnani, 1944). Nevertheless, experimentation has proven that airborne transmissions is a theoretical possibility over several hundred meters (Hyslop, 1979), and circumstantial evidence for such transmission occurred during an epidemic in Nigeria.

The high levels of antibody that are maintained in recovered animals for years after infection, usually for life, suggest persistent infection with RP virus. A recent preliminary study has gone some way to confirm this by demonstrating viral RNA in the peripheral blood lymphocytes of cattle for up to three months after the cessation of viraemia and recovery from clinical disease (Barrett, 1987). There are a handful of chronic cases of RP (Curasson, 1932; Datta & Rajagopolan, 1932; Delply, 1930; Gibbs, 1933), but such cases are exceptions. As a rule, however, there is no "carrier" state in RP, and recovered animals do not excrete infectious RP virus and are not involved in the maintenance and transmission of the disease. The virus is not transmitted by arthropods (Sen & Salam, 1937), and the potential for transmission through abortion is limited (Wafula *et al.*, 1989).

Consequently, RP virus has a short direct cycle of infection and is spread by close contact (Cooper, 1932; Idnani, 1944; Taylor *et al.*, 1965). As originally noted by Lowe (1942), the spread of RP is like a bushfire; it continually moves on the new uninfected animals (unburnt bush) leaving behind dead or immune animals (burnt bush). The disease stops when it runs out of susceptibles (firebreaks), and vaccination is a technique of rendering animals non-susceptible (backburning). Therefore, RP is maintained by large, heterogeneous populations of animals with a sufficient supply of susceptible animals. Stress, malnutrition and high concentration of animals in localized watering points during periods of prolonged drought can be favoring factors in the initiation of spectacular outbreaks (Anderson & May, 1982). In Africa in recent times, the endemic areas have been those with large cattle populations belonging to nomadic or semi-nomadic people, which ensure good mixing of the population, especially when restricted by the availability of water during dry season.

One of the most important epidemiological features of RP was summarized by Huytra *et al.* (1946): *"Rinderpest causes enormous losses among cattle in regions previously free from infection and may lead to the almost complete extinction of animals in large areas. On the other hand, in permanently infected regions it might assume a milder form and cause only a slight loss".*

In populations comprised mainly of susceptible animals RP behaves in epidemic fashion with the virus infecting virtually all of the susceptibles and causing severe clinical disease in most age groups. Endemic RP, however, is much milder and is maintained by young animals usually less than two years old that have lost their maternal immunity. In the classical course of the endemic disease in cattle, once born, a calf has immunity for up to 8 month. It then contracts the virus and is infected and infective for a period of approximately 1 month. But the only susceptible animals are the calves themselves, the adults being immune, for if they had not attained immunity, they would not be able to pass on antibodies (Spinage, 2004). In this situation the mortality is low and the infection sub-clinical and a new generation of susceptible calves is always needed to maintain the disease. This implies that only a large, mixing population can maintain RP in an endemic way. Intermediate patterns also exist. The "plain" cattle of India have a high resistance to the disease, and Edwards (1930) considered there to be an evident periodicity with 3-years periods of abatement, when outbreaks were few, mortality was low, and the disease tended to become readily extinguished spontaneously or sub-clinical. This alternated with 3-year periods of recrudescence that marked numerous outbreaks, high mortality and a tendency of rapid spread. This was attributed to the fact that after 3-year period, the number of highly susceptible stock became almost exhausted, and propagation of the infection could almost no longer take place until a succeeding susceptible generation of cattle

became available. Kock *et al.* (1999) suggested that, in East Africa, the Lineage II virus has a cycle of infection approximately every decade, while Mariner & Roeder (2003) and Mariner et al. (2005) identified a cycle of 5 years.

Examples of epidemic situations in Africa were the enormous losses reported in southern Africa during the Great Pandemic (Section 2.3.), compared with the mild and frequently sub-clinical infections seen in endemic areas, such as Maasailand later in the last century (Henning, 1956; Mornet, 1948; Scott, 1963). However, selection of milder strains of the virus may be an even more important mechanism that could explain different epidemiological behavior of RP, particularly as the generation time of the virus is so much shorter than that of cattle.

Computer modeling provided insight as to how this might occur through selection of strains with differing transmissibility (James & Rossiter, 1989; Rossiter & James 1989). The model shows that during an epidemic RP virus is transmitted rapidly to susceptible animals and the most transmittable strains eventually predominate. For a strain to be highly transmissible it must cause the release of large quantities of virions from damaged epithelia. Hence transmissibility and virulence are directly related. The selection of increasingly transmittable strains in an epidemic involves an increase in virulence. In endemic areas such highly transmittable strains tend to die out because of the lack of susceptible hosts. However, milder, less transmissible strains of virus which are continually produced, presumably through mutation, can persist for longer in the limited supply of susceptible individuals, thus being given the opportunity to become dominant strains (Rossiter & James, 1989). When the less transmittable strain is re-introduced to an unconfined supply of susceptibles, it will again transmit freely and potentially revert to virulence. This ability of the virus to render itself less

clinically obvious in endemic areas may explain those outbreaks of disease that have occurred in areas long thought to have been free of the virus (Lowe *et al.*, 1947; Taylor, 1986; Taylor & Watson, 1967).

Other domestic species such as small ruminants play only an accessory role in the maintenance of the disease in Africa, helping it to smolder for a while after epidemic have occurred in the cattle population (Zwart & Macadam, 1967a & 1967b). They are, however, insufficiently susceptible to maintain endemic strains of RP virus. In India this is not the case. Strains of RP virus, clearly distinguishable from PPR virus have become established in sheep and goats, which could pass the disease back to bovines (Bawa, 1940; D'Costa & Singh, 1933; Narayanaswamy & Ramani, 1973; Orr, 1945). The origin of such strains is unknown, though the use of insufficiently attenuated goat-adapted vaccines has been implied (Crawford, 1947).

Clinical RP was commonly diagnosed in pigs in southern India, but it was not established whether pigs could maintain RP virus independently from cattle, even though circumstantial evidence do not support this hypothesis (Ramani *et al.*, 1974). European pigs, which can be infected by diseased cattle and by eating contaminated meat, suffer only sub-clinical infection but are a potential source of infection for other pigs and cattle (De Lay & Barber, 1962; Scott *et al.*, 1962).

In Asia wildlife species have been described with clinical disease, and such infected animals can transmit infection to other susceptible species, including domestic stock (Bansal *et al.*, 1987; Datta & Rajagopolan, 1932; Lingard, 1905b; Pease, 1894; Singh & Murty, 1979; Vittoz, 1963). However, the size and density of wildlife population are low, and they were not considered to be involved in the maintenance of the virus in Asia.

The role of wildlife in Africa has received much closer attention. This is due to the greater population size and densities, the larger number of susceptible species, and the frequency with which the disease used to be reported in wildlife (Carmichael, 1938; Cornell & Reid, 1934; Wilde, 1948). Until the 1960s a widely held view in East Africa was that wildlife, particularly the vast population of game on the plains in Maasailand, could maintain the virus independently of cattle (Libeau & Scott, 1960; Lowe, 1942; Plowright 1963; Reid, 1949) though some authorities considered cattle to be the main reservoir of infection (Daubney, 1929; Hornby, 1932). However, when cell-culture attenuated vaccine led to the eradication of the disease from cattle in Maasailand in the early 1960s, clinical disease also disappeared from wildlife (Stewart, 1968). The absence of antibodies in wildebeest and other species born after 1963 supported this (Plowright, 1987; Plowright & McCullough, 1967; Taylor & Watson, 1967) and as a consequence opinion changed to the view that wildlife could not maintain the virus (Plowright, 1982).

Subsequent events have indicated that the role of wildlife may not be so straightforward. A study of wildlife sera collected in Kenya between 1970 and 1981 detected significant levels of specific antibodies to RP virus in 8% of the samples (Rossiter *et al.*, 1982). Coincidentally, RP was diagnosed in buffaloes in the Serengeti National Park in 1982, which was the first confirmation of the disease in Tanzania since 1965 (Nyange *et al.*, 1985). The 1982 outbreak then expanded to involve wildlife and cattle throughout a large area of Tanzania north of the central railway line before being controlled by the vaccination of cattle in 1983 and subsequent years.

Serological surveys showed also that most of the buffalo population in the area of Kenya immediately north of the Serengeti also had high levels of antibody to RP

virus, despite having shown no sign of clinical disease. Analysis of the birth dates of sampled animals indicated that new sero-conversion had occurred during two-to-three year period (Rossiter *et al.*, 1987), but unfortunately, no virus was recovered.

Since no RP was reported in cattle in this area, it appeared that wildlife may have assisted in maintaining RP virus for a period of one year and that the infection was unapparent or mild enough to escape notice by wildlife authorities. This has encouraged a more cautious reappraisal of the role of wildlife. At present it seems probable that wildlife do not maintain RP virus indefinitely, but that a sufficiently large population may occasionally maintain mild or sub-clinical RP virus for periods of a few years.

2.2.10. Diagnosis

Several handbooks and reports detail the diagnosis of RP (Bansal, 1986; Bansal *et al.*, 1983; Provost, 1974; Provost & Joubert, 1973; Scott, 1967; Scott *et al.*, 1986). A presumptive diagnosis of RP can be made on the basis of the clinical signs and gross pathology. However, in areas where the disease is not prevalent, and especially in regions dependent on livestock exports, it is essential to obtain laboratory confirmation of the diagnosis as soon as possible.

Countries where RP is either endemic or at high risk should treat any syndrome resembling RP as such until proven otherwise. This will allow immediate steps to control the disease and restrict losses.

2.2.10.1. Differential diagnosis

All conditions that cause stomatitis and / or enteritis in domestic stock may be clinically confused with RP. In cattle, difficulties arise in distinguishing RP from Mucosal Disease (MD), Malignant Catarrhal Fever (MCF) (Theiler, 1909), Infectious Bovine Rhinotracheitis (particularly when caused by strains that induce diarrhea) (Hassan & El Tom, 1985; Hassan *et al.*, 1987), Bovine Papular Stomatitis (Nagington *et al.*, 1967), Jembrana Disease (Teusher *et al.*, 1981), Vescicular Stomatitis and Foot-and-Mouth Disease.

In small ruminants, Peste des Petits Ruminants (PPR) and Nairobi Sheep Disease can resemble RP. Infection with *Campylobacter* spp., *Serpulina* (*Treponema*) *hyodysenteriae* and *Salmonella* serovars need to be considered when investigating possible RP in pigs.

In practice, only MD in cattle and PPR in small ruminants regularly present a problem (Brown & Scott, 1957; Scott, 1964). The clinical signs and gross pathology in cattle with MD can be indistinguishable from RP and diagnosis requires laboratory confirmation. However, MD usually affects very few animals in a herd, whereas morbidity rates in RP are much higher. Agar-Gel Immunodiffusion applied to tissue suspension can rapidly differentiate the two diseases (Darbyshire *et al.*, 1961). Immunohistochemical techniques can be used on frozen sections of mesenteric lymph nodes or on formalin-fixed tissues to distinguish between RP and MD (Gajendragad *et al.*, 1983). Failing this, virus isolation with subsequent virus identification must be attempted, with follow-up studies to detect rising antibody titers. The differentiation of PPR from RP is more difficult. Useful epidemiological evidence is provided by the absence of disease in cattle. The virus cross-reacts serologically with RP virus and is

difficult to differentiate with hyperimmune polyclonal sera. Recent works have produced monoclonal antibodies and cDNA probes that clearly distinguish between PPR virus and RP virus, at least for the limited number of strains tested to date (Diallo *et al.*, 1989; McCullough *et al.*, 1986). In African countries which have previously been free of PPR, it is unwise to assume that a RP-like syndrome in small ruminants is not PPR.

2.2.10.2. Laboratory diagnosis

The collection of adequate quantities of appropriate specimens greatly increases the chance of an accurate laboratory diagnosis. A thorough clinical examination should be made in animals in suspected herds, and six to seven animals in the early acute stage of the disease with fever, mouth lesions and lachrymation should be selected for sampling. Animals that are dead, moribund or have had diarrhea and mucopurulent discharges for more than three days are less reliable sources of virus or antigen as the level of these declines with the onset of antibody development (Scott, 1967).

Blood for serum antibody assay, blood collected in anticoagulant for virus isolation from leukocytes, a lymph-node biopsy, debris from oral lesions, and ocular and nasal swabs should be collected from each selected animal for virus isolation and antigen detection. If possible two or more animals should be killed for necropsy examination, and collection of up to three universal bottles of spleen, mesenteric lymph-nodes, prescapular lymph-nodes, tonsils and ileocaecal entrance should be carried out. All specimens should be collected and bottled aseptically, kept cool on ice (but not frozen) and transported as rapid as possible to a diagnostic laboratory (Scott, 1967; Scott *et al.*, 1986; Wafula *et al.*, 1986). Glycerol should not be used as a preservative

because it inactivates RP virus. The use of antiproteases increases the survival of RP virus antigens in tissue suspensions (Rossiter, 1985).

At the laboratory, suspensions of solid tissues are prepared in physiological saline or cell-culture medium, the buffy coat is removed from the whole blood and the serum separated from the clotted blood. Thirty percent tissue suspensions (w / v) for antigen detection can be prepared by most techniques, including grinding with sand in a mortar, but 10% suspensions for attempted virus isolation can best be prepared in Ten Broeck or similar grinders.

The first procedure carried out is to detect viral antigen using specific rabbit hyperimmune serum against RP virus. The most commonly used assay is the agar-gel Immunodiffusion test (AGID) (Scott & Brown, 1961; White, 1958) which is simple to use, easy to read, highly specific and can be performed in the field with swabs and gum debris. A micro-version is more economical and gives results within 2 hours (Forman *et al.*, 1983). Counter-immuno-electrophoresis is quicker and more sensitive than AGID but requires more sophisticated equipment (Ali & Lees, 1979; Rossiter & Mushi, 1980). Immunofluorescence and immunoperoxidase staining are very sensitive, but also need more equipment than AGID (Babu *et al.*, 1984; Gajendragad *et al.*, 1983; Provost, 1970; Rossiter & Jessett, 1982a; Sharma *et al.*, 1986). Although once widely used, complement fixation and conglutination complement absorption tests are too complicated in comparison with more recently developed tests. Various hamagglutination assays are sensitive but not widely applied (Bansal *et al.*, 1987; Singh, 1972) though latex bead agglutination is giving encouraging results (Mohamed, 1983) and, if combined with monoclonal antibodies, could prove very sensitive. A positive result in any of these tests confirms RP. If classically prepared

rabbit hyperimmune sera (Scott, 1967) are unavailable, sera can be prepared using other immunizing techniques in rabbits (Mohammed *et al.*, 1977; Mullich *et al.*, 1982; Rossiter, 1985) or in goats or cattle (Scott, 1962; Scott *et al.*, 1963).

If cell cultures are unavailable, the specimens can be inoculated into known immune and susceptible cattle, as long as these are isolated from other susceptible animals. If cell-culture facilities are available, attempts should be made to isolate the virus. Suspensions prepared from swabs, gum debris, buffy coats or lymphoid tissues are inoculated onto growing monolayers of primary or secondary bovine kidney cells in tubes (Plowright & Ferris, 1957; Plowright & Ferris, 1962a). Vero cells are also suitable, while culture systems such as microplates can also be used (Mirchamsy *et al.*, 1970; Wamwayi & Wafula, 1987) but may be less sensitive. After 12-24 hours adsorption the tubes are washed, re-fed with maintenance medium and rolled at 37 °C. Typical cytopathic effects develop within 3 to 14 days, occasionally longer, and consist initially of round and refractile cells with cytoplasmic processes and small syncytia, followed by generalization throughout the monolayer with distinct syncytium formation. Negative test cultures should be passaged at least once. The virus can be identified by inoculating sample materials into tubes containing antiserum to RP virus or by examining fixed monolayers using immunofluorescent or immunpperoxidase techniques (Krishnaswamy *et al.*, 1981; Leiss, 1963; Prabhudas & Sambamurti, 1976).

If antigen detection and virus isolation are negative, then convalescent animals should be bled again two to four weeks later. Assays for serum antibodies should demonstrate a four-fold or greater increase in antibody titer in recovered cases. Virus neutralization in microplates is most commonly used for this (Anderson & Rowe,

1982; Rioche, 1969; Rossiter & Jessett, 1982b), although several other techniques such as measles virus haemagglutination inhibition, indirect immunofluorescence, ELISA and counter-immuno-electrophoresis are alternatives (Joshi *et al.*, 1984). Serum during the early antibody response contains significant levels of IgM to RP virus (Anderson *et al.*, 1982; Okuna & Rweyemamu, 1974), the detection of which should confirm the diagnosis. Histopathology is not sufficiently specific to confirm a diagnosis of RP, but demonstration of syncytia and viral inclusions is supportive.

Nucleic acid techniques, including hybridization with probes and polymerase chain reaction are capable of detecting minutes quantities of RP virus RNA in tissues and secretions (Barrett, 1987) and are nowadays the method of choice for confirmation. A complete sequence of the RNA can also be obtained so as to assist epidemiologists in drawing conclusion on the origin of the outbreaks under investigation.

As for the Manual of Standards, the tests recommended for RP by the OIE are:

a) Identification of the agent:

i. Agar Gel Immunodiffusion

ii. Counter Immunoelectrophoresis

b) Differentiation between RP and Peste des Petits Ruminants

i. Differential Immunocapture

ii. Polymerase Chain Reaction

c) Serological Tests

i. Competitive Enzyme-Linked Immunosorbent Assay directed against the RP H-protein (the prescribed test for international trade).

ii. Virus Neutralization

2.2.11. Control

In countries where RP is exotic, confirmed outbreaks are controlled by the slaughter and disposal of all affected and in-contact animals, as well as by the imposition of rigid quarantine and animal-movement control. Such measures eradicated the only RP outbreak in Australia within two months (Weston, 1924) and halted the 1866 outbreak of RP in Britain within six months (Turner, 1906).

Virtually all outbreaks in virgin areas (i.e., areas known to be without reported RP cases) have been due to the importation of live animals (Scott, 1957). Prevention in such areas is therefore largely dependent upon vigilant control of live animals from potentially infected areas. Importation of fresh carcasses or meat products constitutes a minimal threat, although at least one epidemic has been attributed to this source (Ademollo, 1958; Claverie, 1931; Curasson, 1942), and outbreaks in endemic areas have been traced to fresh infected meat. In the Great African Pandemic, the virus is reputed to have crossed the Orange River in this way. In frozen meat the virus persists for much longer than in fresh meat and is therefore a risk to swill-fed pigs (Receveur, 1957; Scott, 1957 & 1959). Infectivity disappears rapidly from adequately dried infected hides (Beaton, 1932) and from decomposing carcasses held at ambient temperature in tropical regions for more than two or three days (Curasson, 1932; Edwards, 1925).

Contaminated areas should be physically cleaned of all animal waste and soiled bedding and treated with disinfectant solution of high (>10) or low (<3) pH containing solvents to destroy the virus envelope. Most disinfectants have some activity against RP virus, but it has been shown that solution of caustic soda and lysol have the highest virucidal activity against virus contaminated with organic matter

(Wamwayi, 1989). Such premises could be restocked after a week but the customary caution of veterinary authorities usually results in the period being longer.

Control is more difficult in endemic areas. Clinical diagnosis should be sufficient to instigate control procedures and all outbreaks of RP-like disease should be treated as RP until proven otherwise. Slaughter of affected and in-contact animals remove the source of virus, but may be counter-productive where owners have either a close cultural bond with their animals or are unlikely to receive compensation. In such areas quarantine and strict movement control must be enforced immediately and the support of local cattle owners is essential in this. Countries bordering high risk zones must channel the import of domestic animals through border quarantine stations for vaccination and three weeks of observation (Libeau & Scott, 1960).

All susceptible animals in affected herds should be vaccinated immediately with subsequent ring vaccination of surrounding districts. The importance of vaccination in the control of RP in endemic areas cannot be overemphasized, and most early research concentrated on the development of effective vaccine (Curasson, 1932; Mornet & Gilbert, 1958; Scott, 1964).

Following the recommendation for the inoculation of infected bile by Koch (1897) in South Africa and the simultaneous administration of convalescent serum and virulent virus developed by Danyz and Bordet, Theiler and Watkins-Pitchford, and Kolle and Turner (Hutcheon, 1902) neither of which were very satisfactory, Kakizaki (1918) developed a glycerol-inactivated tissue vaccine that induced a solid but short-lived immunity. This encouraged the development of many other inactivated vaccines, but these required frequent administration. Continuing research, aimed at a more permanent solution, led to the development of a series of increasingly attenuated

vaccines (caprinized, lapinized, and avianized) which were produced by passage in host other than cattle (Edwards, 1930; Jenkins & Shope, 1946; Nakamura *et al.*, 1938; Stirling, 1932).

Eventually repeated passage of the virulent Kabete 'O' strain of RP virus in bovine kidney cells produced a totally safe and effective attenuated virus (Johnson, 1962; Plowright & Ferris, 1962a) which is now the principal vaccine used to combat RP throughout the world. It is cheap and easy to produce and test, stable in freeze-dried form (Plowright, 1972) and highly effective even in animals with severe infections, such as East Coast Fever and trypanosomiasis (Rurangirwa *et al.*, 1980; Wagner *et al.*, 1975). It induces life-long immunity.

Rarely, vaccinated animals may be infected by experimental challenge, but they have not been shown to re-excrete the virus (Plowright, 1984). The only drawback to the vaccine is that after reconstitution it has a "working-life" of only a few hours in the high ambient temperatures common in regions where RP occurs (Plowright, 1972; Scott, 1985). There is some evidence that the vaccine ought not to be used more than one hour after reconstitution at ambient temperatures around or above 37 °C (Ramachandran & Scott, 1985). Research to improve vaccine thermostability has been based on the selection of heat-resistant clones of vaccine virus and improvement in freeze-drying procedures (Languet *et al.*, 1985; Mariner *et al.*, 1990; Provost & Borredon, 1972; Scott, 1985).

Several laboratories are attempting to remove the problem of the "cold chain" by pursuing the current trend of genetically engineered vaccine in which genes expressing RP virus-immunizing antigens are incorporated into vaccinia virus or some other vector (Hsu *et al.*, 1988; Tsukiyama *et al.*, 1987 & 1988). These recombinants

have proven effective against experimental challenge (Belsham *et al.*, 1989; Yilma *et al.*, 1988). The use of freeze-dried vaccine in subcutaneous implants (Brown & Glossop, 1965) or eye powder would do away with the 'diluent's problem' in hot, isolated areas.

Prevention of RP in isolated areas requires annual vaccination of all calves up to two years of age. Maternal antibodies from immune dams interfere with active immunization and may persist for nine to ten months (Brown, 1958a & 1958b). In practice, however, few animals over six months of age have antibody, and it is sensible to immunize the 6 to 12-month-old animals rather than only those which are over a year old (in an effort to economize vaccine).

Where vaccination is difficult, infrequent or inefficient, and where cattle populations are continually mobile, annual blanket vaccination of all cattle will produce the highest levels of herd immunity (Plowright *et al.*, 1981; Rossiter & James, 1989). In such areas some serological monitoring of herd antibody levels is also advisable. The results from this procedure indicate whether vaccination campaigns have been successful or need to be repeated.

Since relatively high herd-antibody rates may be prevalent in endemic areas (Plowright & McCullough, 1967; Rossiter & James, 1989) serological monitoring can pin-point areas in which herd immunity may be too low to prevent endemic strains from establishing themselves. The successful eradication of endemic RP from Maasailand in 1962 and 1963 was accomplished by very efficient vaccination campaigns which raised overall herd immunity levels to 90% or more. Values in excess of this have been calculated to be necessary for the eradication of measles virus from human populations (Anderson & May, 1982).

In endemic areas sero-surveillance of unvaccinated animals, such as small ruminants and wildlife is also important. These species are sentinels since any antibody they possess must have been induced by field strains of RP virus.

The OIE oversees the zoosanitary approach necessary to achieve the eradication of RP using a practical moving pathway, known as the OIE pathway, which every country has to follow culminating in a declaration of freedom from RP infection (OIE, 2005). FIGURE 2.1 presents a schematic of the above mentioned pathway.

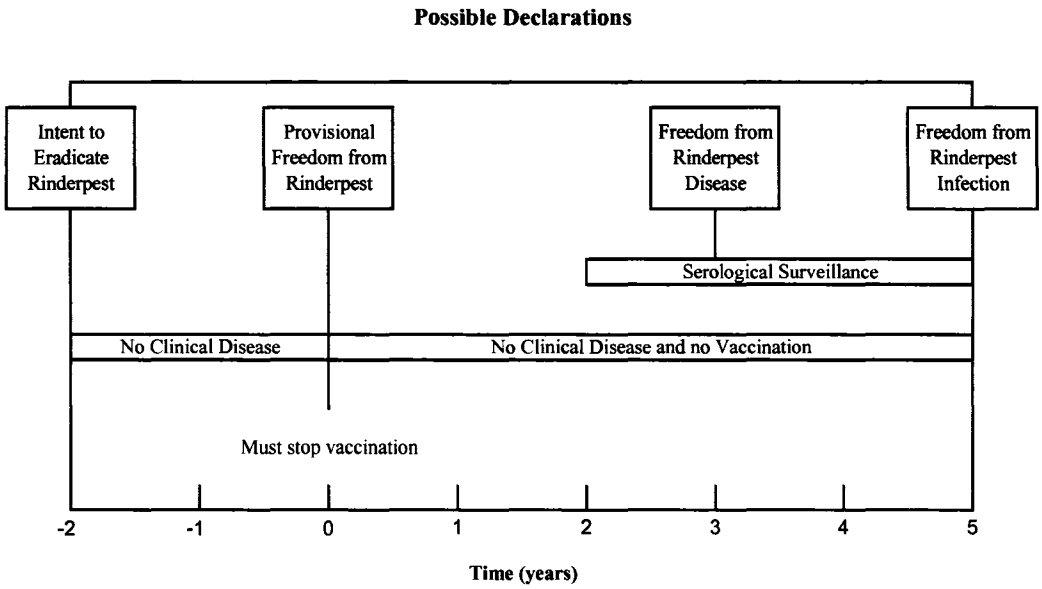

FIGURE 2.1: OIE requirements for the declaration of freedom from RP disease and infection.

As discussed previously the OIE was created in response to an unexpected outbreak of RP that occurred in Belgium in 1920 due to uncontrolled animal movements. "Thanks" to RP also, the first Veterinary Schools were created to tackle the ravages that the disease was causing in Europe in the 18th century, and significant studies in vaccine developed subsequently to the need of controlling the pandemics of RP in Asia, Europe and Africa. RP is also the only infectious disease of veterinary importance for which a Global Eradication Program has been conceived and pursued.

It appears that today the only remaining foci of RP infection are situated in the Somali nomadic areas of southern Somalia, north eastern Kenya and possibly Zone 5 of Ethiopia (the so called Somali Eco-System). In these areas RP expresses itself in a very mild or unapparent form in cattle, and it does not create any significant constraint at production level. However, it still represents an important obstacle for the cross-border cattle trade and oversea export of the Region, without considering the potential of reversion to virulence of the current RP strain, which still represent a great concern at national and international levels.

The final eradication of RP, if accomplished, will eliminate the risk of further spreading of the disease in Africa and the rest of the word and will represent the first animal disease and the second disease in absolute terms (after Small Pox) to be eradicated globally.

2.3. Rinderpest in Africa

2.3.1. The great African Rinderpest pandemic: from 1841 to 1905

2.3.1.1. The introduction of Rinderpest in Africa

It is considered that RP was first introduced into Africa through Egypt from a ship at Alexandria in 1841 with infected Russian cattle offloaded for treatment after others had died. With no effective treatment known, the animals were marketed and dispersed. In a short time, the disease covered the whole of Egypt; allegedly killing 665,000 head of cattle, but by 1843 had apparently burnt itself out. Toward the end of the year, large numbers of cattle passed from Nubia to Egypt to replenish the losses, and the disease erupted again, also killing buffaloes and large numbers of sheep and goats. In the next 3 years, a further 350,000 cattle are alleged to have died,

representing 90% of the herds. Since then the disease remained endemic with further upsurges in 1863-64, 1881 and 1903, and it was reported as endemic again from 1926.

In 1889, when many parts of Africa were in the grip of a serious drought which lasted several years, devastation struck its ungulate fauna. A great RP pandemic swept the length and breadth of the continent in the space of 7 years, 1889-96, destroying populations of many species of artiodactyls both wild and domestic, and contributing to the worst famine Ethiopia had known within living memory. But not Ethiopia alone, for there was wholesale starvation and death among many livestock-dependent tribes such as the East African Wahima and Maasai. In southern Africa, the plague sparked off the Matabele rebellion.

Eliminated south of Limpopo by 1903, it remained endemic in the tropical belt south of the Sahara. Widespread immunization, largely by goat-attenuated virus, was practiced from about 1943 to 1963, so in many of the accessible areas, the only susceptible cattle were young, having lost the maternally derived antibodies and had yet to be vaccinated. But war and governmental mismanagement eventually led to a resurgence of epidemics after the 1970s which continued foci of infection to the present (Spinage, 2004).

2.3.1.2. Rinderpest in Ethiopia

The Ethiopians traditionally blamed the Italian reinforcements landing at Massawa in November 1887, accompanied by cattle from India, that could well been infected, to have imported RP into the country. The importation was allegedly conducted by an Italian called Andreoli. This was asserted at the time by the Ethiopian ruler's Swiss private adviser, Alfred Ilg, and a German traveler, Conrad Keller. Many Ethiopians

believed that the Italians had spread it deliberately, a once widely held view being that they had inoculated three animals with the disease (Pankhurst, 1966). Zonchello (1917) reported that RP appeared to have been first imported into Eritrea in 1987-88. Its spread occasioned by the incompetence and dilatoriness of the Italian authorities in the administration of elementary prophylactic measures. Instead of slaughter, specific measures of treatment were attempted on a large scale with unsatisfactory results. Ferraro (1917) stated that this was the "caudal inoculation" method practiced as a method of vaccination (presumably copying the Willems method for the pleuropneumonia), but the outbreak did not spread widely and was of short duration. In 1888 the outbreak had assumed dramatic proportions. Finding its way through the plateau, it exterminated the vast majority of cattle in Ethiopia. On January 19, 1889 Mother Reeygasse (a Roman Catholic Missionary) wrote from Massawa: *"... M. Picard wrote to me that the people, having nothing more to eat, to feed themselves boil the skin of cattle destroyed by the epidemic"* (Reeygasse, 1889). Herds of 500 – 1,000 were reported dying in 1 or 2 days, and at Bulga, Wurtz reported that all cattle died within 8 days, while of the Emperor Menelik's several thousand, not one survived. Menelik is alleged to have lost about 250,000, while some of the richer Galla lost 10,000 – 12,000 (Wurtz, 1898). The year 1888-89 was excessively hot and dry in Ethiopia, conditions considered to favor the spread of RP through the closer associations of animals at fewer watering points. At the same time RP struck, there was a harvest failure from drought, followed by outbreaks of locusts and caterpillars. The net result was allegedly the worst recorded famine in Ethiopia, lasting from 1888 to 1892.

During the Italian campaign of 1895-96 RP remained circumscribed in the Agrodat District, reappearing along the sea coast in 1897, and, owing to a repetition of

attempts at caudal inoculation, assumed a widespread character. In 1903, Zonchello states, it was decided to adopt the serum-simultaneous method of Kolle and Turner. It is probably from the "caudal inoculation" method or the serum-simultaneous method that the rumor of the Italians deliberately spreading the disease arose. Remaining endemic in many parts of the country in 1897, the disease spread to the Somali coast from Addis Ababa were kudu, hartebeest and cattle suffered severely especially in the Juba River Region (Wurtz, 1898). By 1898, the cattle in Ethiopia still had not recovered their former number.

2.3.1.3. The spread West

From Ethiopia RP spread rapidly west as well as south. Monteil (1895) first encountered RP in West Africa at Dori in Burkina Faso at the end of May 1891. Monteil wrote that a *"year"* ago, every Peulh in *"all the region to Sokkoto"* had at least 100 head of cattle, but now herds of 500 – 600 were reduced to, at most, 10, 20 or 50 head. It was stated categorically that the outbreak *"came from the east"*. In 1892 the slave trader Rabih invaded Bornu from the east, and in 1893, Maistre (1895) reported numerous cases of an epidemic amongst cattle herds near the Tabara River in the Adamawa region of eastern Nigeria.

Chevalier (1908) stated that an epidemic occurred throughout Chad in 1893 destroying a great part of the cattle. Wild buffaloes and antelopes were equally affected. In the following year, ship and goats were ravaged by an epidemic.

2.3.1.4. The spread East

Pankhurst and Johnson (1988) quote a report (Kolle, 1901) of an extensive outbreak of cattle disease between Wadi Halfa and Khartoum (Sudan) in 1888, and stated that

RP appeared in scattered but isolated areas in 1889-90, causing far less damage than in Ethiopia. We know from Slatin Pasha that famine was ravaging in Omdurman at the close of 1889. In March that year, when the epidemic was rampant in Ethiopia, the Mahdists invaded the western part of the Sudan, so if the disease was not already present, it could well have been carried there by the Mahdist forces who may also have initiated its westward passage. About this time, they also ravaged the Dinka tribe and brought back immense herds of cattle (Slatin, 1895).

About 1890-91, the pandemic swept along the south-eastern border of southern Sudan apparently following the Pibor and Sobat Rivers courses. According to Littlewood (1905), the eastern Sudan was affected in 1898 by cattle from Eritrea passing down the valley and the River Atbara.

In Uganda and Kavirondo (Kenya), the RP epidemic began to show its effects in the summer of 1890 and, by autumn of the year, had just reached Unyoro and Ankole, apparently spreading west-wards. About July – August of the following year, it had swept off "every ox" at Kavalli, on the western side of Lake Albert. Emin received information of RP in Ankole in January 1891, and in early April of that year, he found that it had reached the Kagera River via Mpororo. In June, it had reached Bukoba, where it was estimated that 400,000 head of cattle were reduced to some 20,000 in 1891-92 (Ford, 1971). Presumably the plague had begun to affect the Nkore herds in 1890, and the chief then raided Mpororo and Rwanda to replenish his stocks. Tucker (1908) recorded in January 1891 at Kampala that a cattle disease had swept away almost every head of cattle in the country. The disease probably moved south in 1890 through the coastal Galla herds into those of the Wakamba, whence it spread to other tribes, particularly the Maasai. It had not reached Maasailand in the spring of 1890 but

had done so by autumn of that year, apparently first appearing among herds of the Loitokitok Maasai at the base of Mount Kilimanjaro possibly carried there with infected cattle raided from Malindi. Chanler (1896) reported that the disease had destroyed many thousands of cattle in the vicinity of Witu and Lamu in 1889, and in 1890, the Laikipia Maasai raided the Maasai near Nairobi, carrying off infected stocks. As a result most of the Laikipia cattle died. By March, it had reached the Kinangop on the slope of the Aberdere Mountains. Hobley (1929), during his exploration up to the Tana River, first came across evidence of the disease in August 1891 below Mount Meru on the Tana River. Isolated regions, such as Mount Marsabit and Niyro in the north of Kenya, and some parts along the coast apparently escaped infection, later supplying cattle for restocking elsewhere.

The German Vice-Consul at Zanzibar stated in 1892 that the disease had been prevalent in the district around 2 years. On February 7, 1893 the German Consul-General wrote that the Governor of German East Africa reported on December 9: *"the last great havoc had been done of late, not only on the shores of Lake Victoria and Nyassa, but also on the coast and through the intermediate part of the country"*. Barber (1968) placed the first outbreak in Karamoja in 1894-96. In 1897, a renewed outbreak began at Nzawi in the southern part of Ulu, Ukamba Province, which, *"despite all possible measures of isolation"* (Ainsworth, 1905) killed some thousands of head of cattle in various parts. On November 1, 1897 from his headquarter at Machakos, the Commisioner of the East African Protectorate, Sir Arthur Hardinge, submitted to Prime Minister Lord Salisbury a set of proposed regulations which he had drafted on October 26 for Ukamba, to prevent the spread of RP *"now present among the Maasai cattle"* as follows:

"Whereas under the East African Order in Council 1897, Her Majesty's Commissioner has power to make regulations for [the] *peace* [,order,] *and good Government* [of the Protectorate (?) and for the regulation of all matters relating to agriculture and public health within the Protectorate,] *and whereas it is expedient to prevent the spread of cattle plague it is hereby notified that the Commissioner has in the Exercise of the powers aforesaid made the following regulations:*

These regulations apply to the Province of Ukamba only.

From and after this date and until further notice all persons within the province either owning or having cattle in their charge, must at once report any case of sickness to the nearest Government Officer who will use his discretion as to whether or not the animal should be slaughtered.

All transport riders will report their arrival in any district and intended departure from same to the District Officer and they must account for any cattle lost en-route.

Any Government Official may at his discretion order the examination and detention of any cattle where-so-ever found provided of course that he has reason to believe that such cattle has any sickness or have come from any of the proscribed Districts notwithstanding that the owner or drivers may have a pass for such cattle from the Officer in Charge of the District which they have left.

Any person convicted of having concealed sick cattle or of committing any other breach of these regulations shall be liable, to a penalty which may extend to one month's imprisonment.

These regulations may be-as the Cattle Plague Regulations (Ukamba) 1897."

In his annual report for 1898 – 1900, the veterinary officer for British East Africa and the Uganda Protectorates, Robert Stordy, made brief reference that the disease had existed for the last *"five or six years, during which much work has been done by way of prevention and suppression"*. So far, the disease had not been recorded beyond the Gilgil River, 16 Km from Naivasha, and it was hoped that the regulation in force would keep the disease within its circumscribed area. Bullock transport was now operating from Nakuru Railway station (Stordy, 1901). If RP appeared among their cattle, the Maasai were advised to construct special pens at a distance from any habitation and remove all sick cattle to them, which proved successful to limit further outbreaks up until 1899, one sector placing a strong cordon of warriors around the

grazing grounds to prevent communication. But it broke out again among their cattle in 1900, according to Hinde and Hinde (1901) *"through no fault of their own"*.

2.3.1.5. The spread South

From the Kilimanjaro region, the plague moved relentlessly south through Tanganyika. In August 1891, Vrithoff (Alexis, 1894) reported that at Mpwapwa, the camp was surrounded by the carcasses of 2,000 cattle taken by a disease. On August 24, at Mouhalla, a day march from Kilimatinde, he reported that there were a few herds through Ugogo, a disease having killed 14,000 cattle in Ugogo alone. Lieutenant Prince, writing in 1897, estimated that 90% of cattle and half the game had died. Cattle were reported to have survived in only a few places such as on Mafia and other offshore islands, in east Sukumaland and further east, in the higher reaches of Mount Kilimanjaro, but not at Moshi, and possibly in part of the mountain districts of Buha. Shape (1983a) reported that practically all cattle of the north Nyasa country were *"cleared out"*, mortality being over 90%. Lugard (1893) noted that the Wankondé north of Nyasa possessed vast herds of cattle until RP destroyed them at this time. Overall, it moved south from Hamasen to the Zambezi River at an average rate of some 825 Km per year.

The Zambezi River had proven an effective barrier to further natural transmission in initial expansion of the disease, but in February 1896, Rhodes's private secretary Robert Coryndon, who had been to a visit to the Luangua River, reported to the authorities in Bulawayo that the disease had crossed the Zambezi. One year later the French missionary François Coillard wrote:

> *"But a far more general plague than the small-pox, and a much more terrible scourge than the locusts, suddenly made its appearance, and dogged our steps. This was the* ***Rinderpest****.*

No one who was not lived in Africa can form the least idea of this awful calamity. It moved down the whole bovine race in its passage. Hundreds of carcasses lay here and there, on the road-side, or piled up in the fields. In vain did the natives gorge themselves, careless of the consequences. In vain did legions of vultures and beasts of pray gather to devour them. They could not overtake the quantity, and the carrion lay there, putrefying everywhere. More than nine hundred wagons, loaded with merchandise, without teams or drivers, stood abandoned along the Bolawayo road. In a few weeks – a few months, let us say – I am assured that eight hundred thousands head of cattle – some say nine hundred thousand – perished in Khama's tribe alone.

Never within the memory of man has such a thing been seen. The Government grasped the situation from the beginning. But in spite of all the sanitary cordons, and the severest preventive measures, the scourge pursued its course relentlessly ...".

In fact, as soon as the disease was reported to have crossed the Zambezi River, the British authorities of the Bechuanaland Protectorate and the Cape Colony were alerted, two prominent Veterinary Surgeons, Hutcheon and Theilor, were sent to investigate the situation and, after their report, a "RP Commission" was instituted. A slaughtering policy was recommended, where all infected and in-contact animals had to be slaughtered and buried and the owners compensated for the losses. However, the compensation policy could not be fully applied due to lack of funds and the reluctance of cattle owners, Europeans and Africans, to have their animals shot. Moreover, the Boers looked upon the disease as a decree of Divine providence, which *"it would be impious to arrest"*.

At that time small ruminants were not considered to be able to transmit the disease, even though they were dying in great number, so no preventive measures were applied for them. Species of game reported as most affected in the north of Botswana were eland, buffalo, gemsbok, and reedbuck, and in the south, steinbuck, duiker, springbuck, and kudu. But Hutcheon makes no reference to the possibility of RP spread by game and clearly did not think that game was a significant factor. As a result the disease rapidly moved south and westward.

In response, quarantine and movement control were instituted. No horned animals, pigs, dogs, donkeys and mules could move from the infected areas to the clean ones and it was decided to build a double fence to isolate the epidemic zones from the rest of the country. Also this time the instituted control measures failed. The police forces were not in sufficient number to enforce the movement control policy, and the disease moved too quickly to permit the completion of the fence. Hutcheon (1897) reported: *"Within a period of 25 days* (of the first reported occurrence in Rhodesia) *therefore, RP has traveled southward at a rate of 20 miles per day, and had reached a point 16 miles north of the colonial border* [i.e., Cape Colony] *on the 31st of March".*

The construction of the fence was also delayed by the lack of oxen transport. In fact, oxen were dying in great number and the supply of material for the construction of the fence could rely only on mules and donkey, these being almost unusable during the season of the African horsesickness. As a result in May 1896 the disease broke out in the area between Pretoria and Johannesburg, and in July of the same year 299 oxen died in Zeerust District. In the same month, President Kruger held a conference in his farm, and this was followed up in Pretoria at the end of August by an International RP Congress to review the situation. Several proclamations were issued by the South African Government in the attempt of preventing further spread of the disease. These included again a strict slaughtering and movement control policy, but the disease continued its course.

RP erupted in the Kimberley District close to the border of the Orange Free State and the Transvaal in January 1897 as a result of two cows being shot on different farms which all the surrounding farmers came to see and assist to the autopsies, declaring the disease to be gall sickness. They then left without disinfecting themselves, and the

disease appeared on adjacent farms. RP continued to spread and in all, the disease appeared in 16 farms where 29 animals died and 2,951 were shot.

The disease was contained by slaughter in Griqualand west until the end of March, when slaughter was abandoned generally throughout the Colony and Koch's bile inoculation adopted (Spinage, 2004).

When RP appeared in Basuto African reserves of Herschel in that month, cattle owners refused their cattle to be shot, demanding inoculation. This provided the first opportunity to try Koch's method on a large scale; 20,000 cattle of 60,000 were inoculated, of which 14,000 survived. But outbreaks of RP continued in the vicinity, tending to discourage others adopting inoculations. The logic of shooting sick animals eluded the logic of native Africans, and as inoculation sometime spread the disease, they began to wonder why RP always followed government officials, asking how it was that: *"first comes the special with his bottle, and then – after, not before – comes the rinderpest?"* (Anon., 1897a). In May 1897, the Cape Government enacted legislation empowering compulsory inoculation (Act No. 2 of 1897). During a debate of June 3, a Member of Parliament urged removal of the cordon along the Orange River, *"that the frightful expenses be stopped, and the farmers be encouraged to attend to their cattle themselves. Let them be provided with the best bile for inoculation"*.

As there was some distrust of inoculation, it was not enforced but it was left to local RP Committees to decide whether to adopt it in their particular area. But Hutcheon believed that the abandonment of slaughter in favor of inoculation was premature. At Berlin, in the King William's Town District, a demonstration of bile inoculation was arranged for a number of native Africans the recipients dying, and as a results, further

inoculations were refused. Acceptance varied widely in Transkei and Pondoland; at Umtata the Chief inoculated its own cattle and did everything he could to persuade his people to follow his example, but most refused and lost their cattle, while at Eliotdale, two thirds were inoculated, and 75% survived the epidemic. At St. Marks, inoculation was opposed, and 85% of cattle died. In Basutoland in 1897, 34,000 head were inoculated with bile in one district alone saving 50% of the total. In a district where cattle were not inoculated 30,000 died.

Scully, Resident Magistrate at the time at Nquamakwe, Transkei, close to the east coast, relentlessly advocated for the bile inoculation. As a result 20,000 animals had been saved, but where Magistrates in the surrounding districts had obeyed Government's instructions not to pressurize people to adopt inoculation, all the cattle were killed by RP.

Despite all protective measure, RP crossed the Orange River and entered in the Cape Colony. On November 12 the disease was reported at Mowbray Park near King William's Town in the Eastern Cape, and also among Cecil Rhodes's captive elands at Groot Schuur, Rondebosch. During 1896 – 98, 575,864 (35%) of 1,639,435 cattle died of RP in Cape Colony proper (Spinage, 2004).

By June 1896, some 856 Km of fence had been erected along Natal's borders at an average cost of £80 per 1.6 km Despite that RP did break out in September, and the first endeavor was to stamp it out, but owing to so many outbreaks occurring directly after and in so many different parts of the country, such a procedure was found impractical (Verney, 1898). The Department of Agriculture employed 883 men to serve in the campaign against the disease, the country being divided into 21 districts, each with an inspector backed by a total of 127 European guards and 679 native

Africans. The Europeans were armed, and both black and white employees were sworn in as special constables with authority to quarantine infected herds, shoot infected animals, and arrest peoples contravening regulations by bringing cattle across the border into Natal. Mountain passes between Natal and Basutoland were dynamited to make them impassable to cattle and ox wagons.

By May 1897, the Burgesdorp and Aliwal north areas of the Northeastern Cape Province were riddled with RP, and the Transvaal had been depleted of stock. Almost a year after the first alarm, in late June or early July 1897, the disease broke out among trek oxen at Ladysmith. Legislation was already prepared and enacted immediately, declaring infected areas and placing restriction upon cattle movements. After it appeared, in 3 months it had spread over almost two third of the country, taking aback the authorities by its rapidity of spread. Of the 26 administrative divisions 24 were infected between July and October.

A stamping-out policy was then pursued; slaughtering all infected and suspected animals. Because of the discontent this policy aroused, within 10 days bile inoculation was introduced. In September, serum inoculation replaced bile inoculation and it had a better success rate, but by the time extensive programmes were under way, the majority of stock had been lost, and there was a scarcity of "salted" cattle from which to obtain the serum (Spinage, 2004).

In July 1897, the *Times of Swaziland* wrote at length on the responsibilities of the Pretoria government in the event of RP entering the country, appealing for the greatest vigilance in watching the course of the disease (Phoofolo, 1993), but nothing was done, and when it did break out in August, it spread rapidly over the country such that the *Times of Swaziland* reported: *"In no part of South Africa has the rinderpest*

appeared with so much virulence as in Swaziland and in no part of the continent will more difficulty be experienced to fight it" (Anon., 1897b).

Before reaching Swaziland, it had been in Mozambique, killing unknown number of cattle and wild animals. It was first reported there on April 10, 1896 at Chimoio, introduced from Rhodesia by transport drivers.

In February 1896, RP broke out in the Pongola Game Reserve where many wild ungulates died. Kudu was the most susceptible, together with the nyala, and the mountain reedbuck. The impala seemed little or completely unsusceptible as well as bushbuck, waterbuck, blue wildebeest, hartebeest and tsessebe. In Natal, in the game reserve on the other side of the Lebombo River, kudu and other species died near blue wildebeest, but he had not heard that any of the latter had died (Van Oortd, 1898b). The reserve seemed the last place in the Transvaal to be affected (Van Oortd, 1898a).

On October 11, 1899, the second Boer war was declared, nullifying any regulations in force restricting movements of oxen or stock. Isolated cases probably still existed in 1900, and in July of that year, RP was apparently encountered on the Zeerust road. The last case of RP in Cape Colony had been in August 1899, but on March 1901, it was reported to have become epidemic in German South West Africa. In May 1901, it broke out at Mefikeng and Maseru in Basutoland [Lesotho]. In 1902 many parts of the country were once more overrun with RP.

The last South African case was at Gopani Location in the Marico District on August 30, 1903, 7 years 5 months after its first appearance there. In March 1904, the disease, believed to still exist in Zululand, reappeared in two districts in Natal where it was quickly contained. Contrary to the rest of the continent, where RP remained endemic,

the disease was finally eradicated throughout South Africa in 1905, never to reappear, although there were vast quantities of susceptible wild ungulates in the region.

In April 1897, the first case of RP was reported in the German South West Africa, just south of Windhoek, near the Shaf River. The disease then spread through the country in a matter of weeks and was finally eradicated in 1903.

It seems that only a small part of Angola was unaffected, although it did not reach western Angola until late 1897 or early 1898. The disease spread also to the Island of Sao Tomé. In Angola RP was mainly controlled through inoculation and movement control and since then, as far as it is known, the disease never reappeared there apart from a localized outbreak in slaughter cattle in 1972 (Spinage, 2004).

2.3.2. Rinderpest in Africa in the 20th century

2.3.2.1. Egypt

Despite the precautionary measures in place at Alexandria Quarantine Station controlling imports from Asia Minor and Russia, in June 1903 the disease was again introduced into Egypt with cattle from Asia Minor originating from the Baghdad District, affected with a mild form of the disease which did not display typical symptoms, thus escaping detection on import. The disease killed more than a third of Egyptian cattle in less than 1 year, the outbreak lasting until 1905. No sheep and camels were affected.

Whenever the disease appeared in a district, there was much indiscriminate slaughtering, butchers and dealers visiting villages and urging people to dispose of their animals before they were affected by the plague. Healthy animals worth £10 were sold for £3 or £4. Also, large numbers of recovered or "salted" animals were

sold to abattoirs as they were weak for some time afterwards and unable to work. Controlling movements of animals, necessary to prevent spread of the disease, was a new form of legislation, and of such magnitude that the authorities were unable to cope with it, an army of trained officials being required, while the people at first did not understand or believe in its importance. After initial closure of markets, in September 1903, two were reopened as a tentative measure, followed by others. While not immediately apparent, later the mistake became obvious as the disease spread widely.

In 1911, a total of 1,450 animals were reported infected. Up to 1912, the serum alone method of inoculation was used but found to be a failure and replaced by the serum-simultaneous method. A general programme of eradication was then interrupted by the 1914 war, allowing the disease to reestablish itself. In 1917 it spread over almost the whole of Lower Egypt. After the war, recovery of the situation was effected and in 1920 – 21, no deaths were reported from RP while 41,676 serum-simultaneous inoculations were carried out. But imports from enzootic areas of the Sudan were to mean that a low, sporadic incidence was still recorded in the 1980s. In early 1982, there was an outbreak spread by cattle imported from southern Sudan, 4,000 animals dying before it was brought under control (Spinage, 2004).

2.3.2.2. West Africa

Allegedly free from RP since 1893, in 1913, an epidemic of an attenuated form in Chad, supposedly spread from Darfur, killed only young animals and persisted till 1914. In 1917, another epidemic crossed the frontier of Massalit at the same time as entering Zaghaoua, becoming endemic (Pécaud, 1924). Many cattle had been immunized in 1914, and it spread only slowly westwards, killing an estimated

200,000 animals in French Equatorial Africa compared with some 500,000 in 1914. According to Delpy (1931), from 1915 the disease became endemic in French West Africa. In 1918 it again reappeared from the Sudan in a virulent form at first restricted to Ouaddai, but migratory movement then spread it further. From 1920 to 1923, the disease became endemic, with isolated outbreaks appearing here and there and further epidemics in Chad in 1928. Then from 1928 to 1964, outbreaks were reported almost annually, peaking in 1937 and 1951. Delpy (1931) reported that in French West Africa, more than 80,000 animals died each year of RP when the animals concentrated near the Niger River during May to July.

At the end of 1933, a vaccine production laboratory was set up at Fort Lamy to make formalized vaccine, as had been used in West African French colonies by Curasson and Delpy since 1926, when serum production centers were set up at Bamako, Saint Luis and Niamey. Two other centers were opened in Chad in 1936 and 1938. The establishment of these laboratories enabled vaccination campaigns to be undertaken. From 1950 to 1953, production was switched to formalized aluminized vaccine leading to minimal losses, by 1953 half of it being caprinized. In 1962, a coordinated campaign was launched, with cattle in the western part of Chad being vaccinated each year and almost 2.5 millions vaccinations being conducted between 1962 – 63, and 2.7 millions between 1964 – 65 (Thomé, 1964). From 1970 into the 1980s, Chad was to be without proper veterinary care due to internal troubles.

Nigeria was engulfed with major epidemics in 1913 – 14, and again in 1919 – 20. Among the Fulani, the 1919 – 20 outbreak was known as *Docchal*, a remnant, because it left only remnants of herds, there being so many deaths that even the hyenas did not eat the carcasses.

In French West Africa, after the 1891 – 92 outbreaks, there had been no RP until 1915, when it spread from the east, probably Chad, into Niger. It then continued westwards toward the coast with local trade cattle. In Niger, certain areas were declared infected, and as a result of measures taken, Upper Senegal, Mali, and Upper Volta were preserved from it until 1916, when Upper Volta was infected. It then spread throughout the country by the trade routes and infected animals sent by rail to Kayes in western Mali and from there to Senegal. In Senegal it appeared to be assuming a more virulent type, destroying about four fifth of the country's cattle up to 1919. Then the epidemic moved backwards from Mali to Guinea and the Ivory Coast in 1917, finally reaching Togo. Thus it spread first through the areas of dense Sahel cattle populations (there was an estimated 6 millions cattle in French West Africa in 1918; at Timbuctoo, large herds were kept by Tuareg tribes and Fulani) and then returned eastwards through the more sparsely populated forest-savanna mosaic herds (Ford, 1971).

RP was reported again in Upper Volta in 1925 till 1928 ravaging the Peulh's cattle population. Since then it was not reported again till March 1932, when the region was declared totally infected (Marchal, 1980). By 1983, the disease was again widespread in Nigeria, and outbreaks were reported in Niger, originating from the Sudan endemic zone. It had also been endemic in Upper Volta and it also appeared in Cameroon.

2.3.2.3. Sudan and Central African Republic

In 1903, RP broke out in Eritrea and the following January passed to Kassala in eastern Sudan, infected cattle from Eritrea apparently passing down the valleys and the River Atbara as in 1898. An army Veterinary Surgeon, Lieutenant Head, therefore began a program of inoculation using the serum-simultaneous method after checking

the blood for protozoal infections. Between Kassal and Atbara he inoculated about 12,000 animals. The disease was stopped in the area by the end of the same year (Head, 1906).

The Dinka at Wau in the Sudan had thousands of cattle at the beginning of the century, but Landor (1907) reported that RP had frequently played havoc with them, especially further east. In February 1971, there was an outbreak among sheep and goats in the eastern Sudan near the Rahad and Dinder Rivers causing 60–70% mortality among young animals and 20-25% among adults, but it did not spread to the wild animals in the Dinder National Park, and the cattle in the vicinity were vaccinated (Ali, 1973). In 1980 – 81, there was an outbreak among the cattle between Nyany and Malakal, but no wild animals were seen affected. Owing to an inability to maintain annual vaccination of young stock, the disease spread countrywide, 20,000 head dying in the northern region up to June 1982. By August 1983, the outbreak was apparently under control, but in 1983 – 84, RP was present in cattle at Wau, and there were unconfirmed reports of its presence in buffalo and giant eland north-west of Wau near to the border with the Central African Republic.

In the lesser-known Central African Republic, formerly French Equatorial Africa, although it must have mirrored the Chari Region outbreak in Chad, RP was reported first in 1939, then in 1946, 1956 and 1968, the latter two outbreaks apparently killing only game. Chad was infected in 1983 from the Sudan, the disease spreading rapidly south, and nomad cattle from Chad suffered severe mortality in the Central African Republic in 1983, some 2,000 dying. In 1984, some giant eland, a few waterbuck, and warthog were seen to be affected. It then spread south along the stock route and eastwards into the Bamingui-Bangoran National Park, apparently increasing in

virulence and badly affecting buffalos, elands, and warthogs. In 1985, buffalos were reporting dying on the Ourra River in the south-east, the disease reaching Djéma in the east by July, buffalo and Lelwel's hartebeest being the species most affected. In 1984, it also broke out among cattle in the west but was brought under control by vaccination. In 2002, the western part of the country was declared provisionally free from RP (Spinage, 2004).

2.3.2.4. Ethiopia

In 1905 – 06 RP was endemic in most part of Ethiopia; however, mortality progressively decreased from 1905 to 1914 and rarely exceeded 50%. Local people reported eight different outbreaks between 1905 and 1914, with control attempted by sero-vaccination. In many instances, although results were satisfactory, treated animals created new centers of infection with a considerable spread.

At the beginning of September 1918, there was a small outbreak among 135 Somali cattle at Bir in British Somaliland (Adams, 1919). Maydon (1925), hunting in Ethiopia in 1924, reported in eastern Arusi, near the Webbe Shebeli River, one place where the ground was white by the bones of cattle due to a disease which practically wiped out the stock in the neighborhood *"some time ago"* so that now villages appeared to barely exist.

2.3.2.5. Uganda

Presumably, the Karamojong of northern Uganda replenished their herds after 1897 initially through raiding and in all part of the country large numbers of animal were found. But despite the movement and trade control measures in 1911 an outbreak killed half of the stock of both Karamojong and Suk tribes (Melland & Cholmeley,

1912). The disease reappeared in the Mengo District of Buganda Province in August 1913 and during the next 2 years extended into other districts. In 1915 – 16, an outbreak killed large number of Uganda kob. Between May and July of the same year 5,007 cattle were inoculated. In Eastern Province, the disease was prevalent since being introduced from Kenya in 1910, losses apparently confined mainly to young stock. After March 1916, there were no outbreaks in cattle or game in Buganda, the disease eradicated by quarantine measures and inoculation. Between 1913 and 1916, 12,000 uninoculated cattle are estimated to have died.

In 1916 – 17, numerous outbreaks were reported in Eastern and Northern Provinces. In 1916 it struck the cattle in Karamoja again. The following years, 1917 – 18, it spread to Bunyoro, where it was believed to have been introduced by game. From there the outbreak passed to the Northern Province killing cattle, buffalos and warthogs in great number (Duke, 1919). The outbreak reached to within 45 km of Kampala, and Duke (1919) reported *"... it is almost certain that, in their panic of the rinderpest, considerable numbers* [of buffalo] *must have trekked south into Buganda ... the main agent in this spread is beyond doubt the game, especially buffalo"*. In August 1919, Roscoe encountered an epidemic in Ankole where it was *"carrying off the cattle by the thousands"* (Roscoe, 1922).

Ruanda-Urundi and the region of Lake Kivu were infected in 1920. Within 6 days, 2,800 animals were infected or in contact. The disease was spread further south by porters who brought supplies and serum to the Belgian veterinary team combating the outbreak. Vaccination of all animals at risk, totaling 18,000 serum-simultaneous vaccinations, limited the spread, and by June no further cases were reported (Carlier, 1920). From the Lake Albert region, it probably passed through the area of today's

Queen Elizabeth National Park and into the Belgian Congo's Albert National Park, where buffalos were reported to be greatly affected in 1920-21. In 1920, Carlier remarked that the disease remained endemic in Uganda. There was another virulent epidemic in West Nile District in 1925 and from there the disease rapidly extended into the Teso District, but by the end of the year, all outbreaks in cattle had been eradicated (Carmichael, 1938). By 1926, the disease existed only in the Eastern Province on the Kenya border where 7,000 cattle died on this year (Carmichael, 1973). But in 1928 a fresh wave came from the north, 150,000 – 180,000 cattle dying in the Eastern Province.

Kennedy reported another outbreak in West Nile District in 1929, buffalo being heavily affected. In Buganda and the Western Provinces, the cattle population increased from 220,000 in 1921 to 677,000 in 1929 when another major epidemic broke out. In 1930 entered again Ankole and Masaka from the north, where it is estimated to have killed 15,000 head of cattle. In 1931 there were only two outbreaks reported among cattle. In 1933, it was very virulent in south west Ankole, where 15,000 cattle died. RP then persisted among cattle and game in Ankole and Kigezi Districts in 1934, and in 1936 was widespread throughout the year. A further outbreak is reported in Ankole in 1937; and then in 1942, another wave entered Uganda from the Sudan, spreading once more to the Lakes Edward and George region, which appeared to have been free from outbreaks since 1934. It reached Ankole in 1943 having spread almost throughout Uganda, but was never out of control as far as the veterinary authorities were concerned, over 250,000 cattle being inoculated, now with the much more efficient KAG vaccine. According to Carmichael (1973), by the end of the year it existed only in game.

Another outbreak occurred in the Albert National Park in 1945 where a lot of game succumbed. Limited incursion from Sudan occurred again in Uganda in 1948. In 1950, it was present in scattered cattle herds in Karamoja with a major outbreak in Teso early in the year. In 1954 the disease in buffalo spread again from the Sudan border south as far as Murchison Falls National Park and Lake Albert and invaded Ituri Province in Belgian Congo, which had been free of it for 10 years. Further minor outbreaks occurred in the Belgian Congo in 1956 and 1960. Uganda had then become free until 1966 when vaccination campaign ceased and cattle movements became uncontrolled. In 1979, 27 outbreaks were recorded in which more than 5,000 animals died, and by the end of the year 1.5 millions had been vaccinated, but vaccination was not maintained. In September 1988, there was an outbreak in Murchison Falls National Park of northern Uganda in which several hundreds buffalos died (Spinage, 2004).

2.3.2.6. Kenya

In 1903 the disease reemerged in Kenya, and in 1904 an outbreak in Naivasha was extinguished by veterinary teams. In 1907 a new epidemic threatened Kenya from the north and in 1908 an outbreak occurred in Borana (southern Ethiopia and northern Kenya) in which thousands of cattle were reported dying daily (Pankhurst, 1968). At the end of 1908 there was an outbreak in Nakuru, but with low mortality. Montgomery examined sick cattle in the coastal areas of northern Kenya and at Kisumu in 1910. Passing rapidly through the Maasai herds on Laikipia in 1910 and causing heavy mortality in the Kavirondo District, Montgomery saw a new serious disease outbreak in Njoro and on the Nakuru-Naivasha road confirming RP by successfully transmitting it with 2 cc. of blood. In 1910 – 11, the position was

considered serious, the epidemic having spread to Uganda and German East Africa [Tanzania].

A second major epidemic began in 1913 continuing until 1921 although, until 1918, Kenya had a notified outbreak only in the Nandi District. A test of hyperimmune serum in Nyanza had proven successful, and Kabete began producing serum by 1912, nearly 24,000 doses being issued and outbreaks in Ukamba and the Rift Valley then being treated with success. This second outbreak was said to have been fanned by World War I military campaigns with their transport oxen. In 1914 – 15 there were 12 outbreaks on European farms, the disease being endemic throughout most of the African reserves (Spinage, 2004).

In 1915 – 16, outbreaks occurred in several areas: Lumbwa, Molo, Londiani, Njoro, Naivasha, Kedong, Nairobi, Tika, Machakos and Samburu in the Northern Frontier District. By the use of serum-simultaneous methods most of the outbreaks of RP were contained, but a great incidence of redwater was observed. In fact the virulent blood that was injected was taken from native beasts with endemic redwater, almost all native cattle having been exposed to the disease as subadults. Once this was realized, the laboratory built up a herd of redwater-free cattle to provide a reservoir of RP serum (Huxley, 1935).

The years 1917 – 18 witnessed 13 outbreaks (Stordy, 1918). In 1922, Mallett (1923) reported an outbreak at Bardimat in the Narok District of Kenya, where a lot of wildlife died. At this time the Veterinary Department started a wide vaccination campaign, where the cattle owners were requested to pay for the cost of the inoculation. In 1925, 180,000 vaccinations were carried out with 2% mortality.

In 1934, widespread outbreaks occurred in game in Kenya, but only 64 were reported on Europeans' farms and 54 in African areas. These were brought under control with some 215,652 inoculations.

2.3.2.7. Tanganyika

Two German veterinarians had been present in German East Africa since at least 1905 when they made the first report of a RP-like disease in Ugogo in 1905, but it was not apparently until 1912 that the Germans established that certain districts were definitely infected with RP. As a result an Animal Disease Institute was set up at Mpwapwa in 1912.

Tanganyika came to be regarded as the pivotal area for the maintenance of RP because of the vast herds of susceptible Serengeti antelopes in the region adjacent to Mwanza, representing the largest concentration between the southern Sudan in the north and the Kalahari Desert in the South.

While susceptible short-ranging species, such as warthog and bushbuck, were considered to represent little danger, it came to be realized by the late 1920s that the disease could be spread quickly by migratory species, such as buffalos and elands.

The persistence of RP in Tanganyika has been well documented by Branagan and Hammond (1965), who showed that successive outbreaks in the 20th century were apparently not the results of fluctuations in virulence or oscillation between immune and non-immune wild populations, but varying degrees of control maintained by the authorities through inoculation programs.

According to native Africans reports, RP in German East Africa had remained present since the 1890 – 92 outbreaks (Ostertag, 1916). In 1905, a disease was observed among cattle near Lake Victoria which, in the next few years, spread to Mpwapwa, the Meru and Usambara Regions, Kilimatinde and Tabora, Mangati, Irangi, Uhehe, Ugogo, and also Usuwi in Rwanda. In 1909, it was found in the Island of Ukerewe in Lake Victoria. This disease was the object of much controversy, the "catarrhal fever – RP dispute", still unresolved in 1915. Wölfel considered that RP was introduced from British East Africa. In 1909, it had spread south from Kenya to German East Africa and west to Uganda the following year. In 1911, it was present in Sotik, and in June 1912, spread into the settled area near the Uganda road. When it appeared in Uganda in 1910, the German Governor Rechenberg decided to create a 50-km game-free zone along the German East Africa border, employing the army to shoot out the game, a policy apparently widely considered as folly (Koponen, 1994). It did indeed fail to prevent advance of the disease.

Numerous new outbreaks at the end of 1912 and during 1913 affected both young and old animals, cattle and wildlife. The report of 1912 and 1913 showed a very variable clinical picture, and no disease was apparent in sheep and goats. Immediately upon receipt of the reports in 1912, the infected districts were isolated, but there was great difficulty in carrying out the policy. Twelve thousand doses of serum were on hand from Egypt, and a further 15,000 doses were obtained from Kabete. Two centers were immediately erected for the production of serum, but the inoculation program ceased soon after its introduction due to the outbreak of World War I. The disease spread then almost throughout Tanganyika and southwards into Northern Rhodesia.

The first case south of the Central Railway occurred 50 km north of Iringa in November 1914 but was speedily eradicated. The disease broke out again south of the railway at Iringa in January 1917 caused by military supply cattle collected from north of the railway. From Iringa, infection appeared to have been carried south to Njombe following the British lines of communication. In April 1918, few infected cattle were moved to Ukonde. At the end of October 1918, General von Lettow-Vorbeck crossed the border with his German troops, traversing the immune and cattle-free belts near Fife into Rhodesia, pursued by British troops. At least half of the herds came from RP endemic country in Wassanga and Wabena. These infected cattle had traveled through Rhodesia, by way of Fife, to Chambers River, South Kasama, and thence to Abercorn. These troops were met at Abercorn, and as soon as the surrender had taken place, a quarantine station was formed. In November an outbreak occurred in Isoko among some animals which had been brought from Nyasaland. By December 1918, the southern areas were considered clean (Spinage, 2004).

In 1921, there were 86 outbreaks among cattle, which Curasson (1932) attributed to troops from the Belgian Congo. The outbreak reached the Rukwa Valley in southern Tanganyika, killing elands, bushpigs and warthogs. By 1922, the disease was restricted to the north of the Dar-es-Salaam to Tobora railway lines by immunization, but a widespread outbreak broke out in southern Dodoma District. In 1923, Tanganyika witnessed 70 fresh outbreaks in addition to 23 which had begun the year before, involving a total of 374,000 cattle of which 42,894 (11.4%) died.

Generally mild in virulence with a declining mortality averaging only 4.3% in 1926, the disease remained widespread in the northern part of the territory. Although Iringa had remained free for 5 years, this year witnessed the first recorded occurrence of

very mild RP at Tabora. Then in 1927, a number of Kenya Maasai with 45,000 head of cattle, some of which were infected with RP, moved to Loliondo in northern Tanganyika. From this focus, the buffalos of the Ngorongoro highlands became infected, and these spread infection to Mbulu and Mbuguwe to the south, and the southern end of Lake Manyara. At the same time cattle carried it to Lolkisale in the southern Maasailand Region.

In 1929, its progress was aggravated by drought, spreading RP rapidly among the dense concentration of cattle around watering points. It then spread north, affecting the whole of northern Maasailand, Arusha and Kilimanjaro Regions. By 1930, the authorities had eliminated it east and west of the Rift Valley escarpment by means of quarantining and serum inoculation, reducing it to 11 circumscribed foci in the Northern and Lake Provinces. But it then reappeared in October of that year in wildebeest in the western Serengeti (Hornby, 1931), believed contracted from Maasai cattle originating from Kenya, and in 1931, the disease spread rapidly again in the Northern, Western, Lake, and part of Central Provinces.

Owing to difficulties of storage and transport of serum, research was being undertaken into inactivated tissue vaccines, the first field trials of which were conducted in 1929 with some success. In 1932, the highly susceptible Ankole cattle of Bukoba District suffered mortalities of 80%, sometimes 100%. Administrative difficulties, caused by lack of funds, allowed the disease to overwhelm control. The years 1933 – 40 saw a slow loss of containment with the southwards spread of the disease. In 1933, there was an outbreak at the southern end of Lake Jipe in eastern Tanganyika brought in by the Kenya Maasai, spreading to 12,000 head of cattle. In January 1934, it also became established among buffalo, eland and lesser kudu.

Moving south and west, buffalo and eland are said to have carried it to the area of Some, whence it spread to Naberera, and by November, 42,000 cattle were infected.

Meanwhile, in the triangle formed by Tabora, Singida, and Nzaga, a "shuttle movement" of RP was carried on by illicit livestock marketing and seasonal migration of WaTaturu and WaSukuma herds, but was largely controlled by the end of the year. The disease was then reintroduced into Bukoba District by buffalo from Ruanda-Urundi, causing heavy mortality among Ankole cattle; and the following year, 1935, it spread extensively in Lake Province with many wild animals' deaths in the western Serengeti. Near Ngorongoro Crater, this disease was *"almost an annual occurrence, though in recent years it does not appear to do much harm"* (Teare, 1935). In 1936, many deaths were reported in the western Serengeti near the Seronera River.

Wild animals were regarded as the decisive epidemiological factor which spread RP southwards in 1936, and in the southern part of the country the greater kudu was often implicated in linkages. Most domestic cattle outbreaks were among zebu cattle which had not been exposed for 10 years and were highly susceptible, but mortality was generally about 20%. The following year, drought once more aided its further spread as both game and cattle assembled from long distances at watering points, but also its low mortality led to illicit stock movements as an important disseminating factor. By October of that year, it had crossed the Great Ruaha River, which was very shallow due to the drought, but by the end of the year, the disease had been eliminated in the southern Highlands Province.

In January 1938, an international conference was called at Nairobi which agreed to create a 64-km-wide belt of triple immunized cattle to the south of the infected area in Tanganyika. But in May, low level of the Ruaha River allowed it to be crossed again

by infection as in 1936. The special campaign began in June 1938, and by December, 128-km-wide belt of immune cattle had been created. In December the disease was found in cattle and game in Mbeya District and emergency meetings were called at Lusaka and in Tanganyika, where it was decided to create again a belt of triple-vaccinated cattle along Tanganyika's southern border between Lake Nyasa and Tanganyika. Nevertheless, the infection continued to spread, and by the beginning of 1940 it was only 60 km from the southern border with Northern Rhodesia (Spinage, 2004).

With £55,000 guaranteed by South Africa and Southern and Northern Rhodesia, approximately 1 million cattle south of the Central Railway were immunized by the end of the year. In the same year, it was found that the danger of disseminating the diseases by means of the new attenuated goat vaccine developed in Kenya (KAG) was negligible, and it was decided that all cattle behind the vaccinated border herds and as far north as the Central Railway should be inoculated with the goat vaccine (Smith, 1941). Increasing efficiency in the use of this vaccine proved to be the decisive factor in the future control of RP (Branagan & Hammond, 1965). In June 1941, infected cattle and game were identified to the southwest of Lake Rykwa. Another international conference was held in southern Tankanyika, which recommended construction of 3.6-m-high elephant-proof wooden palisade across the 48 km of the Rukwa Valley, later extended to cover 255 km from the northern end of Lake Nyasa to the southern end of Lake Tanganyika, with a 32-km-wide game-free strip on the southern side. As soon as the Lake Rukwa outbreak was discovered, another 42-km-long game-proof fence was constructed in the Saisi Valley where game was infected and cattle both sides of the Northern Rhodesia and Nyasaland borders immunized. At the end of the year the only known focus was in game at the northern end of Lake

Rukwa. The following year 2.7 million inoculations were carried out, and the disease was brought under control. By 1942, the disease was under control in all areas except north Mara, and it was thought that eradication would soon be completed. But in 1944, owing to wartime staffing exigencies, control was lost once again, and infection appeared at the Central Railway at Tabora where it was of such mild character that it was feared that it could spread southwards undetected.

Further outbreaks occurred, 1945 witnessing an extremely virulent strain at Lake Jipe which spread among cattle and game in Tanga Province. A year later its effect on calves was so mild as to be almost undetectable. At the beginning of 1947 the disease was confined to Loliondo, Ngorongoro, and north Maswa. By the end of 1948 it had spread over a wide area aggravated by severe drought conditions of 1948 – 49 and continued to extend slowly among cattle despite inoculation with KAG vaccine. The disease became further widespread in the Central Region in 1949 because quarantine measures could not be enforced due to drought, cattle in many areas being widely dispersed in search of grazing and water. The disease remained prevalent in the area from 1949 to 1953. In 1948, the Director of Veterinary Services concluded that the disease would probably die out where game concentration was small, but persist among large concentration of susceptible species, as in the Serengeti, unless contact between game and cattle could be prevented.

The policy of *ad hoc* mass inoculation in outbreak areas as and when outbreaks occurred was clearly ineffective in eliminating the disease. Mild forms could pass unnoticed until it was well established in the area, permitting game to become infected and spreading the disease further. Thus, beginning in 1950, *all* susceptible cattle in important game areas north of the Central Railway were to be inoculated

annually. Much of the failure to achieve the degree of expected herd immunity was due to the numbers of calves that escaped inoculation on account of the laxity of owners and local authorities, such that in 1954, it was found that as many as 25% were unvaccinated in some areas, and by 1960, there was strong opposition to inoculation (Branagan & Hammond, 1965).

In 1953, the situation deteriorated and some 6,500 Ankole cattle died. In 1955, increased supplies of vaccine resulted in almost 3 million cattle being inoculated, and the year 1956 became the first year since 1919 in which no outbreaks were confirmed. But the following year game was dying in great number (Swynnerton, 1958). By the end of the year, the disease had died out without spreading to cattle. In 1948, it appeared again in the Serengeti's game, and in January 1959, it broke out in young cattle in Engaruka and by March, it spread again among the Serengeti's buffalo. Minor outbreaks occurred near the Ngorongoro Crater in 1960, in the west of the Serengeti, and in Arusha and Moshi Districts. In 1961, illicit stock movements during drought took the disease south to Monduli Juu, whence it extended further along illicit stock routes, necessitating a fresh immunization campaign.

By September the disease was considered eradicated in cattle. From September 1961, Tanzania was generally considered free of the disease. But in December 1965, the disease was confirmed once more in cattle in Tanzania's Loliondo District. The only other known focus at this time was of a different strain, 320 km to the north in Kenya's Isiolo District, suggesting that the Loliondo outbreak was evidence of an endemic infection which had persisted in isolation since 1961 in an undetected form. In March 1966, a fresh focus was confirmed in the same District, where resident and

migrant Serengeti wildebeests were concentrated, but there was no indication that these were infected.

Tanzania was almost free of RP since 1966, but then stolen cattle from the Sudan spread the disease into Kenya and Uganda in 1982, and infected cattle looted in Uganda by Tanzanian troops carried the disease into Tanzania where it spread to wild animals in the Serengeti and Ngorongoro areas, and then to cattle further south (Spiange, 2004). Peaking in 1983, the outbreak was checked by local vaccination campaigns, cattle in Malawi and Zambia along the borders with Tanzania being vaccinated to form an immune belt to prevent southward spread. With an estimated total now at 14.5 million in 1984, sampling in 1983 – 84 showed that five cattle tested positive from Mtwara District, four of which had been recently purchased from the north. Of 136 goats sampled in Mtwara, 14 in Mbeya and 15 in Iringa, only one from Mbeya was positive (Rossiter *et al.*, 1987).

2.3.2.8. Continuation of the infection in Kenya and Uganda

In 1955, several small outbreaks were reported in Kenya from pastoral areas, some of which were places frequented by buffalos. The next confirmed case in a wild ungulate was in an eland in an area near Kinna in Eastern Province, then in 1978 in buffalo in the Narok District of southern Kenya. In 1981, a high prevalence of antibodies was found in small ruminant samples in western Kenya. Positive samples were also detected in November 1983, January and December 1984, and February, May and June 1985, indicating that sero-conversion had occurred frequently and possibly continuously rather than in an outbreak (Rossiter *et al.*, 1987).

A major wave of infection swept over Karamoja and Uganda's Northern Province in 1960 – 61, killing large numbers of wild animals. After the 1979 war in Uganda, the disease broke out again in Karamoja, and there were continued reports of small localized outbreaks in several parts in 1983 – 85. Although controlled by vaccination, in March 1985, it was reported that the disease had moved south to Luwero, north of Kampala, almost certainly through uncontrolled movements of market cattle. In early 1986, the disease was reported in western Uganda around Tororo and Mount Elgon. Then it spread to Jinja in 1987and Arua in West Nile in 1989 (Spinage, 2004).

2.3.2.9. The 1990s Rinderpest outbreaks

In 1986 – 89, an outbreak passed unnoticed, reported in the Nairobi area as MCF and confused by the presence of Theileriosis and other infections (Wamwayi *et al.*, 1992). A severe outbreak was contained in the Tsavo and other parts of eastern Kenya in 1994 – 95, the outbreak killing many wild animals. Initially the virus was thought to be of Type 1 lineage originating from the southern Sudan and neighboring areas, but it was found to be completely different, confirmed as the Type 2 lineage found in West Africa up to 1983 and active in East Africa since the 1940s, related to previous virus which affected game north and east of the Tana river in the 1960s. This last one was isolated from a reticulated giraffe in Kenya shot near Garissa in 1962, suspected to have originated from Somalia.

Over the previous 10 years, only virus of Type 1 African lineage had been detected in Kenya, Sudan and Ethiopia, and it was thought that the giraffe lineage (RGK/1) had become extinct. Thus a second main focus in East Africa was revealed after remaining undetected for 32 years, two distinct lineages coexisting for many years. The exact

location of the focus was uncertain but was believed to be north eastern Kenya and southern Somalia (Barrett *et al.*, 1998).

The genetic stability of the RGK/1 is not unique. If environmental conditions remain stable within an ecological niche, the virus could remain stable at a peak of genetic fitness, and as a result, a relative genetic stasis would be observed despite the high polymerase error rate characteristic of such viruses. The virus was distinct from those of the Sudanese and Ethiopian Afar foci. Its outbreak resurrected the question of a possible game-adapted strain only eradicated by killing game, but the pattern was clearly epidemic and the game sampled in adjacent areas appeared negative, an unlikely occurrence if RP was endemic in the region. The endemic source is uncertain, as it is considered that such a virulent strain for wild animals could not have persisted unnoticed in Kenya game for over 30 years, undetected in the JP15 campaign and 8 year of PARC, and surveillance of game in north eastern Kenya has not implicated game in maintenance of the virus in a silent form. Reversion to a mild form could be a means whereby the virus escaped detection for many years, and cattle in eastern Kenya, Mandera, Wajir, Garissa and Lamu Districts as far as west Meru and adjoining Somalia were considered likely endemic foci. Hence, often in combination with drought, it probably caused periodic reduction in game animals over the last 40 – 50 years. Each epidemic can last several years followed by gradual recovery of game populations given adequate rainfall.

Serological surveys indicated that it had not been active in north-central Kenya, from Samburu-Buffalo Springs northwards through Losai to Marsabit. However, the mid 1990s epidemic spread westwards from Tsavo to Amboseli National Park and north to Kajiado and Nairobi National Park. It also spread south into Tanzania from Loliondo

to west Kilimanjaro and to Lake Manyara, with Ngorongoro and Serengeti clearly under treat (East, 1997).

The source of the Tsavo outbreak was not determined but believed to be connected with illegal movements of large herds of cattle from Garissa along the eastern border of the Park. One rumor blames Somali refugees moving south to safety with their families and livestock, some of them being infected. The Meru outbreak was likely to be similar because there are no longer contiguous game populations between Tsavo and Meru. After apparently disappearing from Tsavo and Meru in 1994, RP re-emerged in Nairobi National Park at the end of 1996. The outbreak was effectively controlled through emergency ring vaccination of all herds in the southern districts, imposition of quarantine, and strict livestock movement control.

At that time, there was no evidence to suggest a major outbreak among cattle in Kenya before the Tsavo outbreak, but in 1997 the disease was confirmed in northern Tanzania. Mild in cattle, RP might have passed unrecognized elsewhere. Kock (2000) considers that, in vaccinated or partially vaccinated cattle, a strain of this lineage causes only mild disease, which is often missed by herders and is of no economic importance to them.

In February 1998, continuing presence of RP was established in the southeast Sudan endemic focus in an area of civil strife and uncontrolled livestock movements, Torit County, close to the Uganda border in an area between Torit and Laifon, involving the intensive pastoral herding system which spread into the neighboring areas of Ethiopia, Kenya and Uganda. The virus was shown to belong to African lineage 1 previously associated with outbreaks in southern Sudan and adjoining regions. Approximately 80% of animals of 6 – 12 months of age and 10% of adults showed symptoms with

50% mortality. Although the origin of the infection was not confirmed, it was thought possibly to be infected cattle brought in from a neighboring area, although there were no reports of outbreaks elsewhere; but for security reasons, the suspected area could not be surveyed until in 2001 it was established that the virus had probably been present since 1998 in animals of the Jie and Murle tribes in Eastern Equatoria and Jonglei. An outbreak of serious stomatitis-enteritis disease in the vicinity of Pibor in 2000 – 01 was identified by the pastoralists as RP. Veterinary opinion was mixed, and investigations attributed the disease to schistosomiasis, but the Operation Lifeline Sudan (OLS) Livestock Program, which FAO assumed responsibility for in late 2000, seized the opportunity to mount an intensive vaccination campaign among the almost 1 million head of Jie and Murle cattle between May 2001 and May 2002. This was the culmination of an effort which had begun in the early 1990s. The FAO campaign was completed successfully by the end of June 2002, and vaccination ceased throughout the whole of the Sudan. Checks up to November 2002 have shown no evidence of the disease, and all indications are that the African lineage 1 virus could now be extinct (Anon., 2002a).

Historically, in north eastern Kenya – southern Somalia ecosystem, RP reappeared periodically showing a cycle of about five years:

- 1980-1983: a moderately severe epidemic of RP entered Mandera and spread to extensive areas of Southern Somalia.
- 1985-1988: a second wave of RP affected the Middle and Lower Juba Regions of Somalia.

- 1991-1993: coincident with the onset of drought in 1991, two waves of RP spread out from Wajir District, Kenya. The first in April traveled through Simper Fatima in central Mandera District to cause moderate mortality in eastern Mandera District. The second wave passed Liboi, Kenya to enter Lower Juba causing moderate to severe mortality (30 to 70%) at Tabta, Bilis Qooqaani, Afmadow, and Badhade in Somalia.

- 1994-1996: RP in Mandera District persisted and assumed a mild form. From Mandera the disease spread to no-mans-land between El Wak, Kenya and El Wak, Somalia where it was sighted by Somali veterinary personnel in mid-1994.

Subsequently, low to moderately severe outbreaks occurred in border regions on both sides of the border until the onset of the rains in early 1996. At that time the furthest known eastern extension of the focus was at Fafadum in the western Gedo Region, Somalia. Clinically mild RP was observed in numerous herds in the Fino, Hashino, Lafey, Alunga, and Warengara areas of Mandera District. Ocular and nasal swabs from affected cattle at Fino and Hashino were positive for the presence of RP antigen in AGID tests conducted by the Government of Kenya at NVRC, Muguga. No clinical disease was observed in Somalia, and no first-hand reports of active clinical disease were received from Somalia since the onset of the rain in April 1996 (Flanagan & Mariner, 1996).

Until then, in Somalia, endemic foci of African type 2 lineage were considered to be confined to the Trans-Juba Region (south from the Juba River). However, more recent investigations have detected RP antbody presence also in unsuspected areas of central Somalia (Terra Nuova, 2001b).

In 1998 – 99 clinically mild cases of RP were detected in several locations of Afmadow District. In Lower Juba, Middle Juba and Gedo Regions of southern Somalia 1,693 serum samples were tested using a RP cELISA H test and 152 were detected antibody positive (8.9%) (Terra Nuova, 1999).

In April 1998, a boat from Somalia carrying infected cattle was turned away from the port of Dubai and is believed to have unloaded the animals at Kisimayu in southern Somalia. In early 1999, there were reports of an outbreak among cattle in southern Somalia and high mortality among warthog. In the Lower Juba Region, there was evidence of clinical symptoms related to RP of a benign form, which usually did not cause mortality in cattle. For security reason, control and investigative work in the area had to be suspended, and RP was not definitely confirmed (Terra Nuova, 1999).

In 1999-2001 serological investigations carried out on unvaccinated young stock showed positive results (using the RP cELISA H) in various locations of Hiran, and Galgadud Regions of central Somalia (Terra Nuova, 2001b)

In October – November 2001 an outbreak of mild RP was detected and confirmed on buffaloes in the Meru National Park, but no evidence of RP virus circulation was found in domestic animals (MoARD, 2001).

At present, it is believed that RP remains in the Somali cattle herds of central and southern Somalia and north-eastern Kenya where it has reappeared periodically in cycles of about 5 years.

In November 2002, an intense international effort was required to focus on this area of endemic maintenance before the virus could break out through the movements of nomadic herds and the export of cattle. The southern part of the Somali pastoral

ecosystem could re-infect nearby areas, but trade could also carry it across the Red Sea to the Arabian Peninsula (Anon. 2002b).

2.3.3. The Rinderpest eradication effort in Africa

2.3.3.1. Conflicting views in control

There were two conflicting viewpoints concerning inoculation. One school of thought was that the disease could be eradicated only by immunization programs if it were not for the large concentrations of highly susceptible game animals which coexisted with cattle and migrated freely in northern Tanganyika and into Kenya. An opposing viewpoint was that, if eradicated in cattle, RP would disappear because it "burned itself out" rapidly in game (Plowright, 1963b). This view reflects the successful eradication of RP in South Africa at the beginning of the 20th century, where the disease was eradicated through its control in the cattle population, despite the presence of large number of wild animals.

Whereas there is ample evidence of the occurrence of RP in sheep, goat and domestic swine, there was no evidence for these species contracting it in Tanganyika, and they were never included in any immunization campaign.

Provost (1980) considered it puzzling that veterinary authorities had embarked upon the original JP15 campaign, for if, as was generally believed, wild ungulates were the reservoir of the virus, there was no hope of eradicating RP by simply immunizing cattle. But the campaign was a success where applied, and eradication of the virus in cattle led to its spontaneous disappearance in sheep, goats and wild ungulates. It is now accepted that the absence of reservoirs in wild ungulates means that vaccination campaigns can eradicate the disease (Anon., 1997), but recently Kock (2000) has

suggested that *"it may be the very effort at eradication through vaccination of cattle that is encouraging the evolution and persistence of this particular strain* [i.e., the game-virulent lineage 2 strain]*"*.

2.3.3.2. The Pan-African campaigns

Until 1950, when vaccination campaigns were increased, some 2 million head of cattle were dying annually of RP in Africa, Asia, and parts of Europe; and in some part of Africa, it still remains endemic.

In 1962 an internationally founded and coordinated vaccination campaign, JP15, was initiated. It began operations in part of Cameroon, Chad, Niger, and Nigeria; in Phase II extending to Dahomey, Ghana, Ivory Coast, Mali, Upper Volta and other parts of Niger and Nigeria. In 1965, the project was extended to Gambia, Guinea, Liberia, Mauritania, Sierra Leone, other parts of Ivory Coast and Mali. In 1968, it was extended to East Africa, to include Ethiopia, Kenya, Somalia, Sudan, Tanzania and Uganda. In Ethiopia, 60 million vaccinations were carried out, but follow-up measures were inadequate, the disease remaining in highland areas. In Nigeria the number of cases decreased from several hundreds annually prior to 1962 to two outbreaks in 1963 – 64 and was allegedly eradicated entirely in 1972. In 1970, it broke out in 74 places in Upper Volta but was contained by 1974. At the termination of the campaign in 1976, over 70 million head of cattle in 22 countries had been vaccinated at a cost of US$ 51 million (Anon., 1983a), but internecine difficulties in some countries, particularly Sudan and Ethiopia, meant that complete coverage could not be achieved, and the disease remained endemic in some areas.

Beginning in 1975, RP spread progressively from a focus in southeast Ethiopia to cover the entire country once again and cross into the Sudan in 1977. From 1976 to 1979 in West Africa, RP was reported only in Mali, Mauritania and Senegal; but after 1979, the disease started to spread once more in Nigeria, Niger, Benin and Upper Volta. The origin of the outbreak was probably from the huge mass of cattle which congregates in the dry season on the border of Mali with Mauritania near the Niger River.

Then, provoked by drought conditions in 1981, the largely unvaccinated focus in southern Sudan erupted, a virulent strain moving rapidly west to Nigeria producing arguably, according to Scott (1993), the biggest cattle disease disaster of the 20th century. One third of the cattle belonging to the Fulani died, and many Fulani herdsmen committed suicide, while their dependents were forced to seek famine-relief camps.

In East Africa it was reported in the early 1980s in cattle in eastern Ethiopia near the Somali border, in Tanzania where it was eradicated in 1983, and in Uganda where it remained endemic in the northeast.

Following resurgence, some US$ 2 million were spent on a further campaign in 1981 covering 10 West Africa countries. This was followed up by an international conference in Kenya in November 1981, at which a Pan-African Rinderpest Campaign (PARC) was proposed under the aegis of the OAU and IBAR, which would be conducted by FAO and the OIE (Anon., 1983b).

The aim was the eradication of RP in Africa with simultaneous campaigns in all countries with endemic foci. A 4-years project, followed by a 6-years follow-up phase to eliminate residual foci, was proposed, commencing December 1984; but by June

1983, the finance required, calculated at US$ 83 millions, had failed to be raised, reducing the campaign to emergency assistance. It was to begin field operations eventually in 1987 in 34 countries, and by the year 2000, donor financed operations had cost nearly US$ 300 million largely funded by the EEC (Kock, 2000). According to Scott (1993) the initial results were spectacular, RP not having been seen in West Africa since 1988, but it remained in the southern Sudan and neighboring areas of Ethiopia, Uganda and Kenya provoking several outbreaks in 1993 – 94.

In 1994, FAO established a new priority program, *Emergency Prevention System for Transboundary Animal and Plant Pests* (EMPRES), of which the initial thrust would be again RP with the *Global Eradication Programme* (GREP) in conjunction with the OIE, and a target date for the worldwide eradication by the year 2010. The United Kingdom's Pirbright Laboratory was designated the FAO *World Reference Laboratory* (WRL) for RP. As a result of this program, the following countries declared themselves provisionally free from RP: Gambia in 1990; Togo and Egypt in 1996; Senegal, Ivory Coast, Ghana, Niger and Mali in 1997; Burkina Faso, Nigeria and Tanzania in 1998. By the end of 1999, Eritrea, Mauritania and Cameroon were added to the list.

In 2000, the absence of infective activity in both Ethiopia and Uganda was established, and active control measures demarcated and selected off the pockets of probable infection in southern Sudan (possibly eradicated at present) and the so called Somali ecosystem, which includes central and southern Somalia, southern Ethiopia and northeastern Kenya.

For a country to become officially free from RP, it must adopt the "OIE Pathway". That is, it first undergoes intensive clinical surveillance. If no RP is detected for at

least 2 years, it applies for "provisional freedom from disease" and must stop using vaccines.

Clinical surveillance continues for a further 2 years, and if no infections are detected, intense serological surveillance of unvaccinated animals is undertaken for at least another 2 years. If no sero-positives are detected, the country is declared "free from disease".

Active serological surveillance continues, and if no evidence of RP is found, the country will be declared "free of RP infection" after 2 more years, that is, 7 years from the declared intention to follow the "OIE Pathway" (OIE, 2005). Thus, to achieve the target date of global eradication by 2010, final foci must be made extinct by the end of 2003.

In the middle of the year 2000, PARC was replaced by the Pan-African Control of Epizootics (PACE) giving wider coverage of animal diseases. PACE is funded by the EEC.

2.3.4. Economic effect of the Rinderpest pandemic in Africa

Cattle are not the recent introduction to Africa that historians of a few decades ago supposed. We know now that in the African Iron Age, from about 200 BC to AD 400, sheep were kept in both Namibia and Zimbabwe, and cattle were present on the edge of Botswana's Okavango Delta slightly before AD 300. In the central Sudan, cattle were present by 3500 BC and in Kenya possibly by 4000 BC, perhaps 5000 BC (Gowlett, 1988). Import of humped-back Asian zebu cattle is believed to have occurred 2000 – 3000 years ago, and this would have been via northeastern or eastern Africa. The impression is that pastoralists arrived in East Africa after the onset of

drier climates to the north and spread to the south as drier climatic conditions permitted. Predating Bantu entry into the region, cattle are known to have been present between central Africa and the Cape from before AD 300, and many have been present at the Cape by the 4^{th} – 5^{th} Centuries (Smith, 1992). Herskovits (1926) has evaluated in detail the former significance of cattle in African cultures in eastern and southern Africa, where, above all, they signified wealth. Providing milk for consumption, cattle were rarely used as a source of meat, and their loss following the RP pandemic had its effect primarily upon the social order; however, the economic losses due to the first incursion of RP were great.

It would be difficult to assess which region in Africa suffered most from the pandemic as our knowledge depends upon reports made at the time, and these are sporadic. The greatest human suffering seems to have been in Ethiopia. However, we find that in many countries, RP only compounded the problems of wars, drought, famine, locust plagues and fatal human epidemics, such as cholera and smallpox, all taking place at the end of the 19^{th} century. Serious drought was experienced from north to south of the continent, and RP was only one factor in the devastation that wrought the African economies. Waller (1988) points out that there is little evidence from Maasailand to support the view that the pandemic precipitated a general ecological crisis, stock epidemics being less harmful in their long-term effects than either endemic diseases or drought.

There are conflicting accounts of the number of cattle which died during the pandemic, but the most accepted one estimates the havoc incurred because of RP at 2.5 million in South Africa and 5.3 million in Africa as a whole. However it is

impossible to separate the mortality that occurred directly because of RP and the one related to other diseases and environmental and climatic factors (Spinage, 2004).

2.3.5. The effect of the Rinderpest pandemic on African game

Over 40 species of wild animal in Africa are potentially susceptible to RP, but the response varies widely from sub-acute to peracute reactions terminating fatally, while blindness contracted by giraffes and both species of kudu is rare in cattle. The most peracute infections other than in cattle are found in buffalo, eland, kudu and warthog. At first in the pandemic, only cattle are alleged to have suffered, although there is some doubt concerning sheep and goats in Ethiopia, but it was soon seen in the *"then huge herds of buffalo"* (Percival, 1918). Eland showed it early, and subsequently all ungulates in East Africa were affected, with the exception of gazelles, zebra, rhinoceros, elephant, and possibly hippopotamus.

Eland was thought to be almost exterminated, but by 1928, Percival could report that in Kenya it was as plentiful as ever, as in *"the old days"*. The greater kudu had been widely but thinly distributed in Kenya before 1896 but was then almost exterminated. It was only 10 years later that it started to recover. A common species in South Africa, Stevenson-Hamilton (1929) considered that, although nearly exterminated, enough were left for the species to become, in less that 20 years under protection, the most numerous of all bush-loving antelopes next to impala. Warthogs were very susceptible, according to Percival dying before there was any sign of the disease among antelopes. Giraffes sometime died in large numbers. Wildebeest were said to be last to become infected in the pandemic according to the Maasai, only succumbing after almost all cattle had died. Hartebeest were not reported as affected at that time, but were so in later outbreaks.

In Uganda waterbuck were seldom infected. Jackson (1894) wrote that buffalo had been almost destroyed. In South Africa, warthog, bushpig, buffalo, bushbuck, kudu, eland, duiker, reedbuck, waterbuck, gemsbok, blesbok, bontebok, springbok, and steenbok, were affected, but not wildebeest, hartebeest or impala (Theiler, 1897). The last great springbok trek in South Africa took place in 1896, and it has been suggested that RP may have played a part in this species never again recovering its former numbers.

It is impossible to state with any degree of certainty how many wild herbivores succumbed in the initial pandemic, but there could have been some 500 million susceptible herbivores in a 10 millions km^2, with the possibility of 50% liable to an acute reaction. At a conservative estimate of 30, ranging to 90% mortality overall, this would indicate between 75 and 225 million deaths, compared with the estimated 200 million cattle death in 18-century Europe, an area about half the size (Spinage, 2004).

2.4. Spatial analysis techniques in epidemiology

Over the years the inclusion of a spatial component in the epidemiological studies has taken progressively more importance. Nowadays spatial epidemiology is referred as *"the description and analysis of geographically indexed data with respect to demographic, environmental, behavioral, socioeconomic, genetic, and infectious risk factors"* (Elliot & Wartemberg, 2004). It is part of a long tradition of geographic analyses dating back to the 1800s when maps of disease rates in different countries began to emerge to characterize the spread and possible causes of outbreaks of infectious diseases, such as yellow fever and cholera (Walter, 2000).

Over the ensuing decades, it grew in complexity, sophistication and utility. Spatial epidemiology extends the rich tradition of ecologic studies that use explanations of the distribution of diseases in different places to better understand the etiology of diseases (Doll, 1980; Keys, 1980).

Spatial epidemiology makes use of spatial analysis techniques. Largely due to computational difficulties, spatial analysis is an area that only recently has become more easily accessible for epidemiologists. Spatial epidemiological analysis includes hypothesis-driven as well as non-hypothesis driven investigations. The latter fit well within the new field of data mining, which has emerged as a result of the availability of large databases (Pfeiffer, 2000).

The complexity of spatial analysis is mainly the result of proximity inter-relationship between observations over space. This causes a problem, because conventional epidemiological analysis typically focuses on the attributes of observations, and makes the assumption that they are independent, while in spatial analysis observations are considered to be spatially auto-correlated. Temporal analysis introduces consideration of one- or multi-dimensional dependence between repeated observations on the same subject. Spatial analysis deals with dependence in two- or three- dimensional spaces (Pfeiffer, 2000).

The last 20 years have seen a significant development in the statistical methods that can deal with analysis of interdependent observations. Spatial analysis is a very challenging field as it introduces a whole new set of techniques in the context of data storage as well as statistical methodology, which are different from what epidemiologists have used traditionally (Pfeiffer, 2000).

2.4.1. Spatial data

Any data with a spatial reference, either relative or absolute, should be considered spatial. In the simplest case, it can represent the geographic coordinates of a location where an outbreak of a disease has occurred. More generally, the spatial reference associated with a single observation describes a spatial feature such as a point or a polygon (Pfeiffer, 2000).

As an example, a point could represent the location where an animal was found dead, and a polygon might describe the boundaries of a farm. In addition to the spatial reference, each observation can also have attribute information associated with it. For example, in the case of the location where a rabid animal had been found, attributes could be the animal species, its age as well as any diagnostic examination results. If the geographical data represent farm boundaries, its associated data may for example record the name of the farmer and the tuberculin test history of the herd. Spatially referenced data can be obtained through direct data entry, digitizing or remote sensing. Particularly the latter has over the last years become a more cost-effective source, for example, for digital vegetation data (Pfeiffer, 2000).

Coordinate point locations can be easily stored using standard databases and displayed using scatterplot graphs. But particularly in the case of lattice structures, Geographical Information Systems (GIS) provide much more effective tools for input, storage, manipulation and presentation of spatial data (Pfeiffer, 2000).

One of the most important decisions that has to be made during collection of spatial data is the choice of the appropriate level of spatial aggregation. It has a strong impact on the cost of data collection, the power of the analysis and the level at which inferences can be drawn. Farms are often presented as point locations as this is the

quickest and cheapest method for obtaining spatial reference data, and this may often be sufficient. But it becomes difficult to analyze neighborhood relationship with this kind of data, as it does not take into account the shape and the size of the properties (Pfeiffer, 2000).

Polygon (i.e. area) data could be presented at different levels of aggregation in that the numbers of infected animals are summarized by herd, district, or country. Inappropriate aggregation can lead to effects, such as the ecological fallacy, and the modifiable areal unit problem (MAUP). The first relates to the differences between estimates at the aggregate and the individual level. The second, the MAUP, stems from the fact that areal units typically do not represent "natural" but rather arbitrary constructs (Haining, 1998). This may result in a fixed number of areal units of the same spatial extent, but varying numbers of animals per unit. Alternatively, the number of areal units can be reduced, resulting in increased spatial extent of each unit. When analyzing for presence of association, variability typically is underestimated, and therefore measures of association may increase. Ideally, one would choose the scale revealing most details, but this is likely to result in substantial cost, loss in data quality, and in data quantities that cannot be processed. Therefore, a sensible compromise has to be found which still allows meaningful observations to be made and sensible inferential conclusions to be drawn (Pfeiffer, 2000).

The accuracy and precision of spatial data is an issue, which deserves special consideration. GIS often combines data from many different sources, which may well be of different accuracy and collected at different scale of precision. Often the error associated with the base maps is not documented. Combining such maps to generate new maps, one of the fundamental function of GIS, can produce unpredictable results.

This can result in error propagation particularly when these output maps are used as input for other operations.

2.4.2. Spatial data analysis

The analysis of spatial data can focus on the relationship between attribute variables, or on the spatial and space-time dimensions or a combination of attribute and space/space-time. The method used in spatial data analysis can be broadly categorized in those concerned with ***visualizing data***, those for ***exploratory data analysis*** and methods for the development of ***statistical models*** (Bailey & Gatrell, 1995).

Analysis can be hypothesis driven or they are used to trigger alarms if something unusual from a statistical perspective has been recorded. The first approach fits nicely into the classical framework of statistical analysis, whereas the second has resulted in controversy amongst scientists, as the risk of Type 1 errors can be high. Many studies will require post-hoc hypothesis formulation and involve multiple comparison analyses.

In spatial analysis, effects have to be distinguished between those which result in long-range spatial trends (i.e. first order effects) and those which produce localized dependence (i.e. second order effects). Often both effects will be present, which complicate spatial analysis as most procedures make the assumption that only one of the two effects is present. First-order effects can be modeled relatively easily using regression models, whereas second-order effects have to be specifically incorporated in the error terms (Bailey & Gatrell, 1995). During most analyses, a combination of techniques will be used with the data first being displayed visually, followed by exploration of possible patterns and possibly modeling.

As previously mentioned, analytical approaches can be divided into visualization, exploration and modeling. Visualization of the data should be the first step in a spatial data analysis. It involves displaying the actual data values as two-, three- or more-dimensional maps. Data are presented as points, colored points or continuous surfaces/lattices. It allows detection of data errors, as well as generation of hypotheses. Exploration of spatial data is aimed at description and quantification of spatial structure. During this phase, hypothesis testing is limited to detection of clusters and spatial dependence (Pfeiffer, 2000).

Methods for quantifying spatial autocorrelation, variograms and specific tests for detection of spatial clustering are applied. Modeling is used to explain and predict spatial structure. Testing for cause-effect relationships is the main purpose of these techniques, which include statistical and simulation modeling, and also multi-criteria/multi-objective decision modeling (Pfeiffer, 2000).

2.4.2.1. Visualization techniques

Visual analysis methods are extremely useful as they allow a process called visual thinking, and they eventually lead to visual communication through presentation of the data, for example, in map format.

If the emphasis is purely on spatial occurrence, such as whether a herd is infected or not, this can be shown using dot or polygon maps depending on whether point or polygon data have been used as the spatial reference. With dot maps any pattern becomes difficult to detect, as soon as the density of points increases. In this case interpolation techniques can be used to generate continuous surfaces of the underlying point density.

i) *Kernel Smoothing:*

It has become the standard method for these interpolations. The resulting surface represents the probability of the occurrence of a case and is estimated using a bivariate probability density function (i.e. Kernel), which is symmetric about the origin. The amount of smoothing is dependent on the chosen bandwidth, which can be fixed or adaptive. The images improve presentation of point data, but the effect is strongly influenced by the parameters of the Kernel estimator and its algorithm. It is also possible to estimate the ratio of Kernel estimates and, thereby, adjust for differences in the population at risk (Bithell, 1990; Kelsall & Diggle, 1995).

The Kernel Density Estimate $\hat{f}(x)$ for a sample $x_1, \ldots x_n$, is given by:

$$\hat{f}(x) = \frac{1}{nb}\sum_{i=1}^{n} w\left(\frac{x_i - x}{b}\right) \qquad [2.1]$$

where n is the number of observations, b is the *"Bandwidth"* and $w(\,)$ is the *"Weight Function"* or *"Kernel Function"* satisfying:

$$\begin{cases} w(x) \geq 0 \\ \int_{-1}^{1} w(x)dx = 1 \end{cases} \qquad [2.2]$$

That is, $w(x)$ is a probability function on [-1,1].

In the case of a bivariate probability function the estimate is given by:

$$\hat{f}(\underline{x}) = \frac{1}{nb^2}\sum_{i=1}^{n} w\left(\frac{\underline{x}_i - \underline{x}}{b}\right) \qquad [2.3]$$

where $w(\,)$ is the *"Weight Function"* satisfying:

$$\begin{cases} w(\underline{x}) \geq 0 \\ \iint w(x_1 x_2) dx_1 dx_2 = 1 \end{cases} \quad [2.4]$$

The intensity function $\lambda(x,y)$ is similar to a probability density function, but its intergral needs not to be 1. Considering $\underline{x}_1$, ..., $\underline{x}_n$ to be the n points of a point process, a Kernel estimate of λ is given by:

$$\hat{\lambda}_b(\underline{x}) = \frac{1}{b^2} \sum_{i=1}^{n} w\left(\frac{\underline{x}_i - \underline{x}}{b} \right) \quad [2.5]$$

This estimate ignores edge effects. To account for them, the following correction must be utilized:

$$\hat{\lambda}_b(\underline{x}) = \frac{\sum_{i=1}^{n} w\left(\frac{\underline{x}_i - \underline{x}}{b} \right)}{p_b(\underline{x})} \quad [2.6]$$

Where:

$$p_b(\underline{x}) = \int_A w\left(\frac{\underline{x} - \underline{u}}{b} \right) d\underline{u} \quad [2.7]$$

With polygon data, differences in denominators can be taken into account through cartogram presentations or density equalized map projections (Merrill *et al.*, 1996), where the size and shape of the polygon is basically re-scaled to represent differences in denominator values, such as for example population density.

ii) *Standardization:*

Standardization was used to be the main technique for mapping of disease rates and ratios. In the case of case events their spatial distribution can be described in terms of spatial variation of intensity of the cases $\lambda(x)$ at any spatial location x.

In order to provide an estimate of the "at-risk" population at spatial locations, it is first necessary to choose a measure which will represent the intensity of cases "expected" at such locations. This is normally defined as the background population hazard function *g(x)*. The ratio *R(x)* of the two estimates is given by:

$$R(x) = \frac{\lambda(x)}{g(x)} \qquad [2.8]$$

By mapping this ratio it is possible to compare $\lambda(x)$ and *g(x)*. However, the standardization of rates and ratios has the disadvantage that cannot take into account the increased uncertainty about true estimates resulting from small counts and denominators.

iii) *Empirical or Fully Bayesian Estimates:*

Empirical or fully Bayesian estimates are more appropriate as they use the overall or local risk as a prior, with the effect being that the local estimate is shrunken towards the overall or the neighborhood mean risk particularly if it results from small numbers of observations in that particular location (Clayton & Kaldor, 1987; Langford, 1994). In fact, in many epidemiological problems, it is natural to regard some or all of the model parameters as random variates which are governed by a probability distribution, which leads to a Bayesian analysis. The attribution of random effects to groups or individuals is the subject of frailty models (Clayton, 1991), and in principle the inclusion of such effects requires the addition of a random component in the specification of the probability of a case event $\lambda(x)$ at some location x. In this case, for an individual random effect, it is possible to define:

$$\lambda(x_i) = g(x_i)\xi_i m(F\alpha) \qquad [2.9]$$

where ξ_i is the random effect for the i^{th} individual. For this model there are a variety of approaches to the examination of posterior information provided by the sample found.

A full Bayesian analysis evaluates the full posterior distribution of parameters and associated summary measures, if required. This approach provides general information on parameter variability and the correlation among parameters. The fitted values are usually defined as an estimated expectation, i.e. $\hat{y}_i = \hat{\mu}_i = E(y_i \mid \hat{\theta})$. Under a Bayesian model there are a number of possible values for θ under the posterior distributions. The posterior distribution of θ given the data y, where $g(\theta)$ are the prior distributions for θ, can be defined as follows:

$$P_0(\theta \mid y) \propto L(y \mid \theta) g(\theta) = \prod_{i=1}^{n} f(y_i \mid \theta) g(\theta) \qquad [2.10]$$

The predictive distribution of an observation y^* can be defined as:

$$p(y^* \mid y) = \int f(y^* \mid \theta) P_0(\theta \mid y) d\theta \qquad [2.11]$$

Hence, a sample of y^* values could be obtained from (2.11) and this could be compared with the y data. In a full Bayesian analysis, (2.10) can be used to provide a sample or samples of parameter values. Usually, the joint and marginal posterior distributions of parameters are of interest, and these can be estimated from these sample values.

It is also possible to avoid this full exploration by attempting to find the *modal* values of the posterior distribution of parameters. This form of analysis is known as Empirical Bayes (EB) as it uses conventional frequentist estimation in the posterior

distribution (Besag, 1986; Ripley, 1988). In the case of case event data it is possible to assume (2.9) but with $m(\cdot)=1$, and $\xi_i \sim G(\alpha,\upsilon)$, then the posterior distribution is equal to:

$$\prod_{i=1}^{n} \xi_i g(x_i) e^{-\xi_i \int g(u)du} p(\xi_i) \qquad [2.12]$$

where $p(\cdot)$ is the prior distribution for the random effect. In this case the EB method would lead to the estimation of α and υ, and the substitution of these estimates in a functional of the posterior distribution, such as the expectation.

Examples of the use of EB estimators for census tract data are numerous. In the case of count data observed in census tracts, a number of authors have proposed estimators of tract relative risk based on different random-effect assumptions. For example, if observed counts are defined as n_i with expected count e_i and relative risk θ_i, then, with $n_i \sim Poisson\ (e_i\ \theta_i)$ and $\theta_i \sim G(\alpha,\upsilon)$, the posterior distribution of θ_i is $G(e_i + \alpha,\ n_i + \upsilon)$ conditional on n_i. The posterior expectation of θ_i reduces to:

$$\frac{n_i + \upsilon}{e_i + \alpha} \qquad [2.13]$$

Note that the crude relative risk estimator, the standardized mortality/morbidity ratio (SMR) is just n_i/e_i and this is the maximum likelihood estimator for the ordinary saturated model. Hence, a Full Bayesian Analysis of this model would require sampling of θ from the above conditional gamma distribution, whereas an EB approach could estimate θ from the conditional expectation with suitable estimates of α and υ substituted.

2.4.2.2. Exploratory analysis

Exploratory analysis of spatial data is aimed at describing spatial patterns using inferential statistic, and it is used for the development of a hypothesis. With data on the occurrence of disease, it is mainly about whether diseases occur randomly in space or not.

It is complicated by often having to take account of the spatially clustered distribution of the underlying population at risk. One effective method for dealing with this problem is the use of case-control data, where the cases of particular disease are selected as usual and the controls are selected randomly from the non-diseased population, and therefore should represent the spatial distribution of the underlying population at risk. The control could be matched to cases with respect to confounding factors other than spatial locations (Lawson & Waller, 1996).

This approach requires exact point locations for cases and controls to be available. If the data source is routine surveillance, it is likely to be aggregated at some administrative level such as veterinary districts.

As exploratory spatial data analysis involves statistical hypothesis testing, with the availability of fast computer technology bootstrap and permutation methods can be used to deal with the multiple testing problem (Kulldorf & Nagarwalla, 1995).

In the statistical assessment of spatial clustering of point and polygon data, global or local statistics can be generated. Global statistic will indicate if there is clustering somewhere in the area of investigation. Local statistics will also indicate where the likely clusters are, and this will be particularly useful if the analysis is aimed at triggering an alarm (Pfeiffer, 2000).

i) *Nearest-Neighbour Statistic:*

Cuzick and Edwards (1990) developed a method which is based on nearest-neighbour distances. This aggregated test statistic compares the number of case-case pairs for a given number of near neighbors and tests for detecting non-random patterns of the observed mean distance to the expected mean distance of a randomly distributed population of the same density ρ. To do that the ratio R is calculated in order to detect the degree to which the observed distance distribution depart from random expectations and it is defined as follow:

$$R = \frac{\bar{r}_A}{\bar{r}_E} \qquad [2.14]$$

where R is the Nearest-Neighbor Index, $\bar{r}_A$ is the average distance between randomly selected points to their nearest-neighbor, and $\bar{r}_E$ is the expected mean distance between nearest-neighbor under the randomness assumption. To test the hypothesis that the observed distance distribution is from a randomly distributed population, the null and alternative hypothesis are formulated as follow: H_0: $\mu = \mu_0$ against H_A: $\mu \neq \mu_0$. Since R is a point estimator of μ *(Lawson, 2001)*, the test statistic is given as:

$$z = \frac{R-\mu}{\sigma_{\bar{r}_E}} = \frac{R-1}{\sqrt{\frac{(4-\pi)}{\pi n}}} \qquad [2.15]$$

The null hypothesis will be rejected for $|z| \geq z_{\alpha/2}$.

Considering two populations of cases and controls i and j respectively with μ_i and μ_j being the true mean distances between nearest-neighbor for the two populations, it is also possible to test the null hypothesis that the means distance distributions are equal.

In this case the null and alternative hypothesis are formulated as follow: H_0: $(\mu_i - \mu_j) = 0$ against H_A: $(\mu_i - \mu_j) \neq 0$ *(Lawson, 2001)*, and the test statistic will become:

$$z = \frac{(R_i - R_j) - (\mu_i - \mu_j)}{\sqrt{\frac{\sigma_i^2}{n_i} + \frac{\sigma_j^2}{n_j}}} \qquad [2.16]$$

Where σ_i^2 and σ_j^2 are the respective population variances.

Other more recently developed techniques for the analysis of case-control point data for presence of spatial clustering are the K-function and the spatial scan statistic.

ii) *The K-Function:*

The K-function produces an aggregated statistic. It expresses the mean number of cases with increasing distance from a given case scaled by the density of points in the area. The procedure is performed for cases and controls separately, and the difference between the two resulting K-functions can be plotted against distance. The K-function is the cumulative function of distances from points in *A* (the study area) to other points in *A* and it is expressed as follows:

$$\hat{F}(d) = \frac{\sum_{i \neq j} I_{ij}(d)}{N(N-1)} \qquad [2.17]$$

where $I_{ij}(d) = 1$ if the distance d_{ij}, between points i and j is less than some specified distance, d, and 0 otherwise, and N is the total number of points in the population. The function $F(d)$ is subject to a downward bias if no allowance is made for boundaries.

To take into account edge effect, Ripley replaces $F(d)$ with:

$$\hat{K}(d) = \frac{A\sum K_{(i,j)}}{N(N-1)} \qquad [2.18]$$

For a Poisson process, the expectation of $\hat{K}(d) = \pi d^2$. By taking the square root of $\hat{K}(d)$, and plotting it as a function of distance, *d*, the plot should be linear:

$$\hat{L}(d) = \sqrt{\frac{\hat{K}(d)}{\pi}} \qquad [2.19]$$

It is therefore possible to obtain a random expectation statistic that can be then generated by randomly permuting cases and controls at least 100 times and estimating K-functions and their differences for each permutation. If the observed curve extend beyond the simulation envelop, then significant clustering of cases relative to controls can be assumed (Bailey & Gatrell, 1995; Jones *et al.*, 1996). For a Poisson process $\hat{L}(d)$ has an approximate mean of *d* and an approximate variance of $1/(2\pi N^2)$; and for a unit square, $\hat{L}(d)$ is an estimate of the proportion of the population within *d* units.

This techniques assumes that only second-order effects are present and that these are isotropic (i.e. not dependent on direction). The disadvantage of the K-function is that it does not describe the location of the clustering. It is also quite common with animal diseases to have first-order effects present, such as variation in climatic conditions, as result of different levels of elevation. (Pfeiffer, 2000).

iii) *The Spatial Scan Statistic:*

In contrast, the spatial scan statistic is a local clustering statistic. The procedure involves generating ever-increasing circles around every point and calculation of relative risks based on disease risks within and outside the circle and the statistic is generally based on Bernoulli or Poisson models.

Assuming a Bernoulli model, there are cases and non-cases represented by a *0/1* variable. These variables may represent individuals with or without a disease, or individuals with different types of disease. They may reflect cases and controls from a larger population, or they may together constitute the population as a whole. Assuming a Poisson model, the number of cases in each census area is Poisson distributed. Under the null hypothesis, and when there are no covariates, the expected number of cases in each area is proportional to its population size.

A likelihood ratio test is then calculated to assess for statistical significance, and the distribution of the test statistic is estimated using Monte-Carlo sampling (Kulldorf & Nagarwalla, 1995).

For each location and size of the scanning window, the alternative hypothesis is that there is an elevated rate within the window as compared to outside.

Under the Poisson assumption, the likelihood function for a specific window is then proportional to *(Lawson, 2001)*:

$$(c/n)c([C-c]/[C-n])(C-c)I() \qquad [2.20]$$

where C is the total number of cases over the whole area, c is the number of cases within the window, and n is the covariate adjusted expected number of cases within the window under the null-hypothesis.

For the Bernoulli model the likelihood function is *(Lawson, 2001)*:

$$(c/n)c(1-c/n)(n-c)((C-c)/(N-n))(C-c)(1-((C-c)/(N-n)))(N-n)-(C-c)I() \qquad [2.21]$$

where c and C are defined as above, n is the total number of cases and controls in the cluster, N is the total number of cases and controls in the data set.

The spatial scan statistic is one of the more robust techniques for exploratory analysis of spatial clustering.

iv) *Moran's I and Geary's C*

For aggregated data such as disease rates, Moran's *I* and Geary's *C*, which are global statistics, and are used as estimators of spatial autocorrelation (Geary, 1954; Moran, 1948). The Moran's *I* is defined as follow:

$$I = \frac{n}{2A} \frac{\sum_{i=1}^{n}\sum_{j=1}^{n}\delta_{ij}(Z_i - \overline{Z})(Z_j - \overline{Z})}{\sum_{i=1}^{n}(Z_i - \overline{Z})^2} \qquad [2.22]$$

where *I* is like a correlation coefficient between "neighboring" Z_i's (observed value), which can be defined as δ_{ij} equal to *0/1* for non-neighbor and neighbor, respectively, or can represent proximity matrix (generally inverse distances, $\frac{1}{d_{ij}}$, where d_{ij} is the distance between pairs of points). *A* is the total number of joints and is defined as:

$$A = \frac{1}{2}\sum_{ij}\delta_{ij} \qquad [2.23]$$

The Geary's *C* is defined as:

$$C = \frac{(n-1)\sum_{i=1}^{n}\sum_{j=1}^{n}\delta_{ij}(Z_i - Z_j)^2}{4A\sum_{i=1}^{n}(Z_i - \overline{Z})^2} \qquad [2.24]$$

The Geary's *C* provides information similar to the Moran's *I*.

To allow a local description of spatial dependence for this type of data, the generic concept of Local Indicators of Spatial Association (LISA) has been developed

recently (Anselin, 1995). It embraces Moran's *I*, Geary' *C* and the Getis-Ord G_i statistics under a single mathematical framework. This methodology can be complemented by visualization of the resulting statistics as maps presenting spatial lag pies or bar charts (Anselin *et al.*, 1993).

v) *Space-Time Cluster Analysis:*

Testing for clustering in space and time may be of interest. This is usually done using the point locations of the cases, and most statistics available will work on all possible pairs of time-space distances between the points. The Knox test (Knox, 1964) and Mantel method (Mantel, 1967) have been the classical techniques used for this analysis. More recently, the space-time scan statistic (Kulldorf *et al.*, 1998) and the K-nearest neighbor test (Jacquez, 1996) have been developed. All four techniques use permutation statistics.

They make the assumption that population sizes do not change in time, but the statistics are not affected by spatially heterogeneous populations. The K-nearest neighbor test of space-time interaction allows an assessment of the statistical significance of a potential space-time interaction process. The test statistic indicates the number of case pairs which are K-nearest neighbors in time and space. The statistic is based on an approximate randomization of the Mantel product statistic.

vi) *Variogram Analysis:*

An exploratory analysis of the spatial dependence of continuous type data involves the use of techniques such as the variogram method. This type of data is usually collected at sample point locations where some attribute, such as density of disease vector populations is being measured. Spatial moving averages are used to represent first-order spatial effects that are global trends in a particular geographical area,

whereas variograms describe localized effects, i.e. second-order effects. The variogram function $\gamma(h)$ is given by:

$$\gamma(h) = \frac{1}{2N(h)} \sum_{(ij)|h_{ij}=h} (z_i - z_j)^2 \qquad [2.25]$$

where h is the distance separating sample locations i and j, z_i is the variable of interest at location i, and $N(h)$ is the number of data pairs separated by distance h.

The presence of second order effects would result in positive covariance between observations at small distance apart, and lower covariance or correlation if they are further apart.

The covariogram describes the function of the covariance for varying distances h between sample points and the correlogram the corresponding correlation. The semi-variogram is a graphical representation of the variation between sampling points separated by a given distance and direction.

For a stationary spatial process all three analysis describe similar information. Estimates of the semi-variogram are considered to be robust to departure from stationary represented as a general trend in the spatial process. A continuous process without spatial dependence will result in a horizontal line.

A stationary process will reach an upper bound, referred to as the still at a distance h called the range. Theoretically, the intercept with the y-axis is called the nugget effect. Variograms which do not reach an upper bound suggest non-stationarity in the data (Pfeiffer, 2000).

2.4.2.3. Modeling

Models derived from spatial data can be used to identify risk factors or they can remain within the spatial domain if these models are used for interpolation or smoothing. Particularly the latter are aimed at visual presentation.

The most basic models are based on map modeling (Bonham-Carter, 1994). These involve overlaying different geographical layers of information using, for example, Boolean logic, whereas more advanced map modeling will apply fuzzy logic or Bayesian methods.

i) *Spatial Autoregressive Models:*

A major area of continuing research development is the field of spatial risk factor modelling. The objective of these analyses in the context of epidemiology is to generate risk surfaces taking into account underlying geographical risk factor patterns. In epidemiology parameters of interest are very often counts or proportions which should be modeled using generalized linear modeling techniques rather than ordinary least-squares regressions. The most significant problem has been to determine mathematically sound techniques for taking into account dependence in the spatial data structure.

Bailey and Gatrell (1995) suggest introducing covariates into the regression model, such as spatial coordinates or a variable representing broad regions to adjust for the effect of spatial dependence. Glass *et al.* (1995) developed a risk density map for Lyme disease based on a multiple logistic regression model, but they did not attempt to remove spatial dependence from the data. Williams *et al.* (1994) compared a number of different predictive modeling approaches for spatial data. They used linear and non-linear discriminant analysis, tree-based induction and neural networks to map

tsetse distributions in Zimbabwe and concluded that while the simpler methods (linear discriminant analysis and tree-based induction) were less precise, they were easier to interpret. They also did not explicitly take account of spatial dependence. Pfeiffer *et al.* (1997) used a logistic regression analysis for prediction of *Theileria parva* presence in Zimbabwe. The regression model includes eight different environmental and land use variables and is based on information collected at randomly sampled locations throughout the country. The model was used to generate a risk map representing the probability of *T. parva* presence at a particular location given a number of risk factors included in the model. The Receiver Operating Characteristic Curve (ROC) characterizing the predictive accuracy of the model was used to adjust the decision making cut-off for the prediction probability balancing sensitivity and specificity as required. In this analysis the possible presence of spatial dependence was taken into account using a categorical variable representing region as a random effect.

Important developments have now provided more appropriate solutions to the problem of incorporating spatial dependence in regression models. The advent of multi-level modeling provided a statistical framework for modeling spatial dependence in covariance structures (Langford *et al.*, 1999). Spatial dependence of residuals in regression models can be assessed by the use of, for instance, a Moran's *I* test (see [2.22]) or a Lagrange multiplier test, the significance of which is determined as follows:

$$\left[\frac{n\hat{\varepsilon}'W\hat{\varepsilon}}{\hat{\varepsilon}'\hat{\varepsilon}tr(W^2+W'W)^{\frac{1}{2}}}\right]^2 > \chi^2_{1,.95} \qquad [2.26]$$

where ε is the vector of the model residuals, and W a spatial weight matrix. If the residuals are spatially autocorrelated, then the regression model should account for this lack of independence. This is done by adding a covariance term in the model which accounts for the spatial correlation of the residuals and is based on a spatial weight matrix. The model can be defined as follows:

$$\underset{\sim}{Y} = X\underset{\sim}{\beta} + (I - \lambda W)^{-1}\underset{\sim}{\varepsilon} \qquad [2.27]$$

One particular approach for representing spatial dependence with binary outcome variables involves the use of autologistic terms which were originally described by Besag (1974). This term can be used as a covariate additional to other risk factors in a logistic regression model to reflect the dependence of the local risk of disease or the level of disease in the neighbors (Gumpertz *et al.*, 1997; Augustin *et al.*, 1998).

These authors applied Gibbs sampling for parameter estimation. A Bayesian hierarchical spatial modeling has been described by Xia *et al.* (1997) who used a mixed prior, one conditionally autoregressive and the other unstructured. This approach of including non-spatial, as well as spatial random effects in the model, which was first suggested by Besag *et al.* (1991), is now commonly adopted when performing spatial regression modeling (Wakefield & Elliott, 1999; Langford *et al.*, 1999).

Other techniques use the prior in Bayesian statistics for representing the spatial dependence during the estimation of model parameters. In addition, the computation technique Markov Chain Monte Carlo (MCMC) method using Gibbs sampling procedures robust simulation-based estimates of the likelihood or the posterior distribution are used in the case of Bayesian inference (Lawson *et al.*, 1996).

ii) *Trend Surface Analysis:*

A number of approaches can be used to model or predict spatially continuous data. For the first-order processes, trend surfaces can be generated with ordinary polynomial least square regression and can be defined as follows:

$$Z(\underline{x}) = f_0(\underline{x})\beta_0 + ... + f_p(\underline{x})\beta_p + \varepsilon(\underline{x}) \qquad [2.28]$$

where $Z(x_1)$, ..., $Z(x_n)$ are "observations" at certain locations x_1, ..., x_n, $f_0(\underline{x})...,f_p(\underline{x})$ are known functions, $\varepsilon(\underline{x})$ is an "error" process sometime referred to as random field and β_0, ..., β_p are parameters of the model. The Least Squares Estimate of $\underline{\beta}$ is given by:

$$\hat{\beta}_{LS} = (F'F)^{-1}F'Z \qquad [2.29]$$

Results have to be treated with caution, because the standard regression assumptions of independent random errors and homoscedasticity are likely to be violated. Lessard *et al.* (1990) applied this type of approach when using an inverse distance weighted mathematical algorithm to interpolate climatic measurements between sample points.

Most trend surface models may be able to describe an overall trend, but are not useful for local prediction. In the presence of weak first order, but strong second order effects it is more appropriate to use models fitted to variograms. Such models can be defined "by eye" and are most commonly based on a spherical, exponential or Gaussian model fitted to the variogram. The fit of a particular model can be assessed through cross-validation based on a comparison between the observed and interpolated values.

iii) *Kriging:*

A variogram model itself does not allow prediction of values. This can be achieved with Kriging. This is a weighted moving average technique for estimating the value of a spatially distributed variable from adjacent values while considering interdependence expressed in a variogram. It allows the interpolation error to be mapped and from a statistical viewpoint is considered, according to Olivier and Webster (1990), an effective technique for interpolation of continuous type spatial data.

Webster *et al.* (1994) used Kriging to describe the risk of cancer in children for the West Midlands of England. The resulting maps showed that child cancer in this region clearly had a patchy distribution, in that areas of high risk were near to each other as was the case amongst those of low risk. The authors emphasized that with low incidence disease such as in their analysis it was important to have large amounts of observations available. They expressed concern about the validity of the confidence limits they had estimated for their binomial data, as the technique is more appropriate for prediction of continuous type variables.

Carrat and Valleron (1992) applied Kriging to generate a map of weekly influenza cases for France. They concluded that Kriging had the advantage that it was not constrained by geographical boundaries and that it can be used to satisfactorily replace missing values.

Pfeiffer (1994) used ordinary Kriging to produce a surface of possum population density based on possum capture data at sample points.

iv) *Multivariate Analysis:*

A number of multivariate methods can also be used for modelling of spatially continuous data. Principal components combine the information from multiple variables into a small number of components, each of them representing a particular combination of variables and explaining a particular proportion of the variation in the data. Eastman and Fulk (1993) used the technique to analyze the information contained in a time series of NDVI map for Africa, thereby conducting a space-time analysis of continuous type variables.

This technique could be used to assess the relative importance of spatial variation in comparison to temporal variation; for example, the pattern of tick-borne disease incidence across a country could be separated into spatial and seasonal/cyclical variation.

Cliff *et al.* (1995) discussed the application of multidimensional scaling (MDS) to spatial epidemiological data. They used the technique to map geographical information about measles mortality in Austria and New Zealand as a disease space where points with similar disease risks are closer to each other on the MDS map even though they are far removed geographically. Bailey and Gatrell (1995) discuss a range of other multivariate analysis techniques for spatially continuous data.

Recently, the use of spatial data for optimization of resource allocation has been more and more explored. Methods include multi-criteria and multi-objective evaluation techniques which have been adapted for spatial problems. They can take account of the uncertainty in the underlying input data, as well as of the risk of making the wrong decision (Eastman *et al.*, 1995). Systems are being developed which take data from various spatial input data sets, for example, define optimal types of animal production

for specific geographical regions. With this methodology, it could be possible to define optimal disease control strategies given various spatially defined constraint variables.

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CHAPTER 3:
STUDY DESIGN AND IMPLEMENTATION

3.1. Introduction

Nowhere in Africa is nomadisme, of such great significance as in Somalia, where an estimated 60% of the population is engaged in mobile livestock keeping, with degree of variability in the location of animal production and residence over time. The remaining 40% of the population comprises urban dwellers and farmers, but they usually maintain considerable contact in a variety of ways with the rural and nomadic areas (Schawartz, 1993). It has become even more difficult to differentiate between the various groups in clear statistical terms because, as can be readily observed, there is a rapid unfolding process involving the formation of numerous transitional and combined forms of nomadism, semi-sedentary and sedentary ways of life in the rural sector (Abdullahi, 1990).

Livestock production and trade represent one of the most important sources of livelihood for pastoralist and ago-pastoralists in the Great Horn of Africa (Jankhe, 1982). Moreover, taxation of livestock marketing and export is considered to be one of the primary sources of revenue for Somali local authorities. With the collapse of the Somali state in 1991 and the introduction of more stringent health codes regulating international animal trade and certification, such as the WTO Sanitary and Phytosanitary Agreement, Somali pastoralists have experienced a dramatic

deterioration in terms of trade for their animals and severe disruption in livestock export toward the Arabian Peninsula. This has seriously affected the Somali economy in general, impaired coping mechanisms developed in the last centuries and increased risk of relegating the back bone of the Somali economy to a more modest role (Ahrens, 1998; Dietz *et al.*, 2001; Steffen *et al.*, 1998; Holleman, 2002). It is still too early to assess whether Somali pastoralists will be able to regain their past dominance in the livestock industry. Especially the export sector will require strong assistance in terms of animal health services to strive again and regain the role that it had occupied in the very recent past.

Among several factors hindering livestock trade and export toward the very lucrative markets of Saudi Arabia and other Gulf States, the limited knowledge on transboundary diseases due to the lack of animal disease surveillance systems, seems to be one of the major challenges to be addressed.

In fact, the development of reliable animal disease surveillance systems will not only contribute to the reduction of risks associated with livestock trade, but may improve access of products of animal origin from developing countries to more lucrative markets (e.g. developed countries), thus fostering fairer trade and contributing to poverty alleviation.

However, establishing reliable animal disease surveillance systems in pastoral areas poses additional challenges because of: (i) the greater mobility of animal populations, (ii) poorly tested surveys systems, (iii) general remoteness, (iv) poor infrastructure development, and (v) the general paucity of public resources to fund reliable and well designed veterinary services.

Furthermore, surveillance techniques have been mostly developed on the assumption that certain conditions apply to a specific system being investigated. For instance, the random concept is at the base of a representative estimation of the level of a disease, meaning that a thorough knowledge of the individuals composing the study population is available. This condition is seldom applicable in most of the highly dynamic production systems in Africa, such as the pastoral nomadic ones.

This constraint has posed numerous challenges in the generation of reliable data that could guide the final RP eradication effort from the Horn of Africa and therefore from the world. In fact to date it is believed that the only remaining foci of RP worldwide are located in the cattle rearing areas of Somalia and the neighboring Somali inhabited areas of Kenya and Ethiopia. In order to provide reliable information on the extension of such foci and the epidemiology of the disease in the country, a cross-sectional sero-survey was designed and implemented in 10 administrative regions of central and southern Somalia. The representativeness of the study was guaranteed by the randomization of the primary sampling units through the use of random map coordinates.

This study was carried out within the framework of the Somali PACE Project (Section 1.5) as an effort towards the final eradication of RP (Sections 2.3.5.).

3.2. Study area

The criteria utilized for the delineation of the area of intervention were: (i) historical and recent data on evidence of RP circulation in Somalia (Section 2.3.2.9); and (ii) high concentration of cattle and susceptible wildlife species in the country.

The cattle density distribution for Somalia, which refers to the last available livestock census for the country (Anon., 1989) is depicted in FIGURE 3.1. The highest cattle densities are found in the southern and central Regions (e.g. Lower & Middle Juba, Lower & Middle Shabele, Gedo, Bay, Bakool, Hiran, Galgadud and Mudug).

Secluded cattle populations are also encountered in the north-west of the country (e.g. Awdal and Wakooyi Galbed). However these populations are quite isolated from the other cattle areas of Somalia as well as the neighboring Ethiopia.

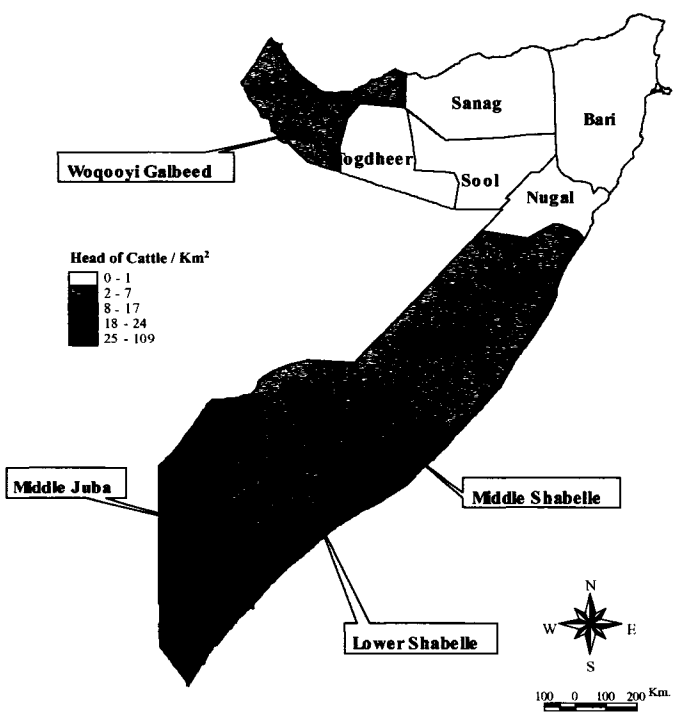

FIGURE 3.1: Cattle population density by region for Somalia.

Moreover, no evidence of circulation of RP virus has been reported from these areas for the past 20 years, and the adjacent areas of Ethiopia are considered free from RP disease (OIE Declaration of freedom from disease on a zone basis). On the other hand, indication of viral activity has been detected at different times over the past two decades in the southern and partially the central Regions of Somalia (Flanagan & Mariner, 1996; Terra Nuova, 1999 & 2001; see also Section 2.3.2.9).

No historical or recent wildlife data are available for Somalia. Nonetheless, it is generally accepted that, after the strike of the civil war when wild animals were

widely hunted for survival or poaching, patchy buffalo, kuku and gerenuk populations have "survived" in a few regions of southern Somalia (e.g. Lower Juba, Middle Juba and Lower Shabele river areas).

Warthogs are still widespread in the semi-arid areas of the country (e.g. central and southern Somalia) since the Islamic religion, dominant in Somalia, does not allow the consumption of their meat.

Other wildlife species still exist in Somalia, but they have not been considered here, because they are not relevant for RP transmission and maintenance.

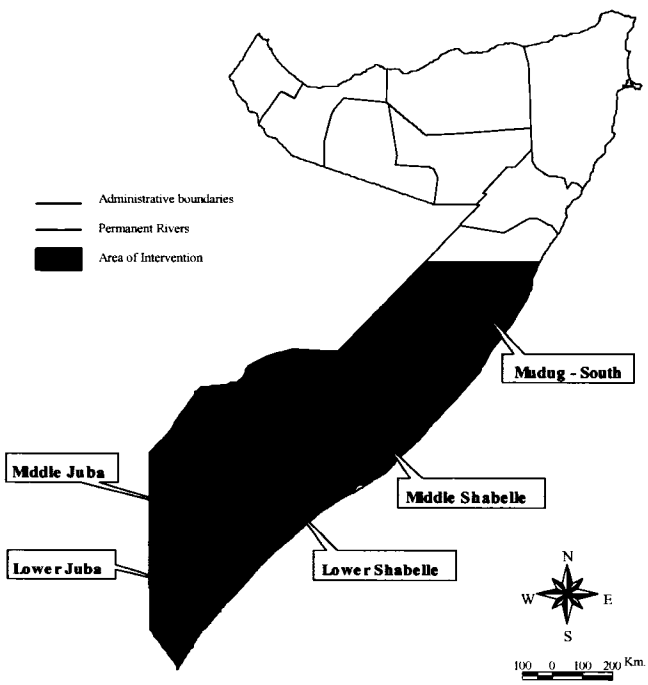

FIGURE 3.2: RP survey – Study area.

Considering the above mentioned factors, the survey was designed to cover 10 administrative regions of the former Republic of Somalia, namely: Lower & Middle Juba, Lower & Middle Shabele, Gedo, Bay, Bakool, Hiran, Galgadud, and Mudug south of Galkayo (FIGURE 3.2).

3.3. Survey design

The investigation consisted of a cross-sectional sero-survey based on a two-stage cluster sampling design.

The sample size was calculated at the level of administrative region. This had the advantage that if one or more regions was inaccessible (e.g., due to open conflict), the rest of the survey could still be implemented without compromising the results of the investigation in other areas.

Serological data from previous RP serological investigations carried out in central and southern Somalia in 1999 – 2000 (Terra Nuova, 1999; Terra Nuova 2001) were used to calculate the between cluster variance (V_c) in the area. TABLE 3.1 summarizes the data of the above-mentioned investigations.

TABLE 3.1: Summary of data deriving from RP serological investigations carried out in central and southern Somalia in 1999 – 2000.

Total Num. of Samples	*Total Num. of Sampling Sites*	*Average Num. of Samples per Site*	*Observed Prevalence*	*Observed V_c*
2185	69	31.5	9.7%	0.0063

The sample size for the actual survey was calculated for expected prevalences equal to or greater than 20%. This was done to cope with the possibility of having a sero-prevalence higher than the previously observed one. The observed V_c (0.0063), (which refers to an observed prevalence of 9.7%) was then adjusted for the new expected prevalences (Thrusfield, 1998) and then used for the calculation of the sample size of the survey. The sample size for each administrative region was computed for a two-stage cluster sampling design using the adjusted V_c (Thrusfield, 1998). The total sample size for the 10 administrative regions was obtained by multiplying the sample size of each region by the number of regions to be covered by the survey.

TABLE 3.2 shows the sample sizes for: (i) Expected Prevalence between 20% and 50%, (ii) 95% Confidence Interval, and (iii) 5% Desired Absolute Precision.

TABLE 3.2: Sample size for an expected prevalence of 20 to 50%, 95% confidence interval and 0.05 desired absolute precision.

Expected Prevalence	*Adjusted V_c*	*Num. of Samples / Site*	*Average Num. of Sites / Region*	*Num. of Regions*	*Total Num. of Samples*	*Total Num. of Sampling Sites*
20%	0.014	13	40	10	5200	400
30%	0.018	15	50	10	7500	500
40%	0.021	13	60	10	7800	600
50%	0.022	15	60	10	9000	600

The sampling size computed for a 50% expected prevalence was selected for the actual survey. This choice guaranteed a better accuracy of the prevalence estimates for any observed prevalence different than 50%. Furthermore, a high number of observation points (e.g. sampling sites) was considered to be the preferential choice for the foreseen spatial analysis of the survey data. The total number of sampling sites (e.g. 600 for an Expected Prevalence of 50%) was then proportionally allocated according to the cattle population density of the regions of interest (FIGURE 3.1).

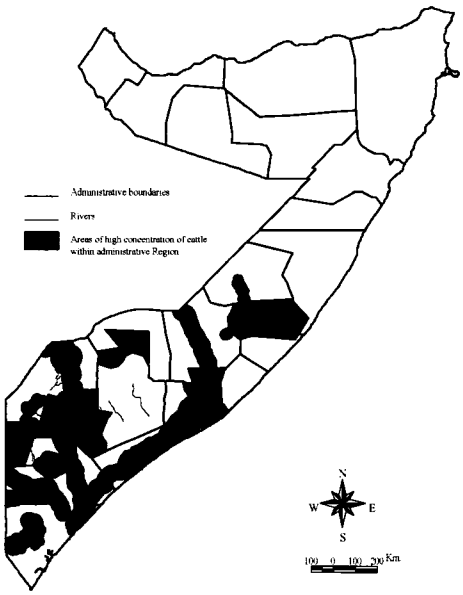

FIGURE 3.3: Areas of high concentration of cattle within administrative region.

Within each region the number of sampling sites was further proportionally allocated according to the identified areas of high and low concentration of cattle. These areas were identified from the available literature and empirical knowledge (FIGURE 3.3). This was done to obtain a better prevalence estimate at the level of administrative region as well as in the survey area as a whole.

TABLE 3.3 shows the number of sampling sites by region for an expected prevalence of 50% after proportional allocation.

TABLE 3.3: Proportional allocation of sampling sites by region (areas of high and low concentration of cattle) for a 50% expected prevalence design.

Region	*Cattle Population*	*Cattle Density (Head of Cattle / Km²)*	*Number of Sampling Sites*		
			Areas of Low Cattle Density	*Areas of High Cattle Density*	*Total*
Bakool	116,080	4	15	35	50
Bay	269,000	6	21	31	52
Galgadud	282,310	6	15	37	52
Gedo	612,900	13	17	41	58
Hiran	200,750	6	15	37	52
Lower Juba	999,450	21	21	49	70
Lower Shabele	443,940	17	13	51	64
Middle Juba	424,860	23	15	58	73
Middle Shabele	443,420	24	26	48	74
Mudug	239,628	7	33	22	55
TOTAL	**4,032,338**	**11**	**191**	**409**	**600**

The sampling sites were obtained by randomly generating the needed number of geographical coordinates according to the breakdown provided in TABLE 3.3.

When using this sampling procedure, it is likely that a proportion of selected sites will have no herds or animals within the specified radius (e.g. 10 km radius). In order to cope with this scenario 10 "spare" sampling sites per investigating team were generated.

In each previously selected and geo-referenced sampling site, 15 blood samples had to be collected from animals between 1 and 3 years of age. The purpose of this selection was to identify relatively recent circulation of RP virus and to avoid bleeding animals that could still possess maternal antibodies or had been vaccinated during previous RP

vaccination campaigns. In Somalia, the most recent RP vaccinations were carried out in 1998-99 in the Trans-Juba Regions of Southern Somalia (e.g. Lower Juba, Gedo and part of Middle Juba) (Terra Nuova, 1999).

In addition to the 15 blood samples per sampling site, 15 blood samples were collected using 5 samples from each of the age groups > 3-4 years old, > 4-5 years old, and > 5years old. A total of 60 sampling sites (10% of the total number of sampling sites) were included in this collection. The purpose of this stratification was to gather information on past exposure of the cattle population to RP. However, the results obtained from an animal older than 3 years will not provide any accurate estimate of the sero-prevalence in these age groups. The 60 sampling sites were randomly selected within the 600 sampling sites of the survey in the areas of high concentration of cattle. TABLE 3.4 provides the total number of blood samples that had to be collected by region.

TABLE 3.4: Total number of blood samples and sites (15 & 30 samples per site and total) by region.

Region	*Number of Sampling Sites*			*Total Number of Samples*
	15 Samples / Site	*30 Samples / Site*	*Total*	
Bakool	43	7	50	855
Bay	48	4	52	840
Galgadud	46	6	52	860
Gedo	52	6	58	960
Hiran	46	6	52	870
Lower Juba	64	6	70	1140
Lower Shabele	53	11	64	1125
Middle Juba	68	5	73	1170
Middle Shabele	67	7	74	1215
Mudug	53	2	55	855
TOTAL	**540**	**60**	**600**	**9900**

FIGURE 3.4 shows the overall structure of the survey.

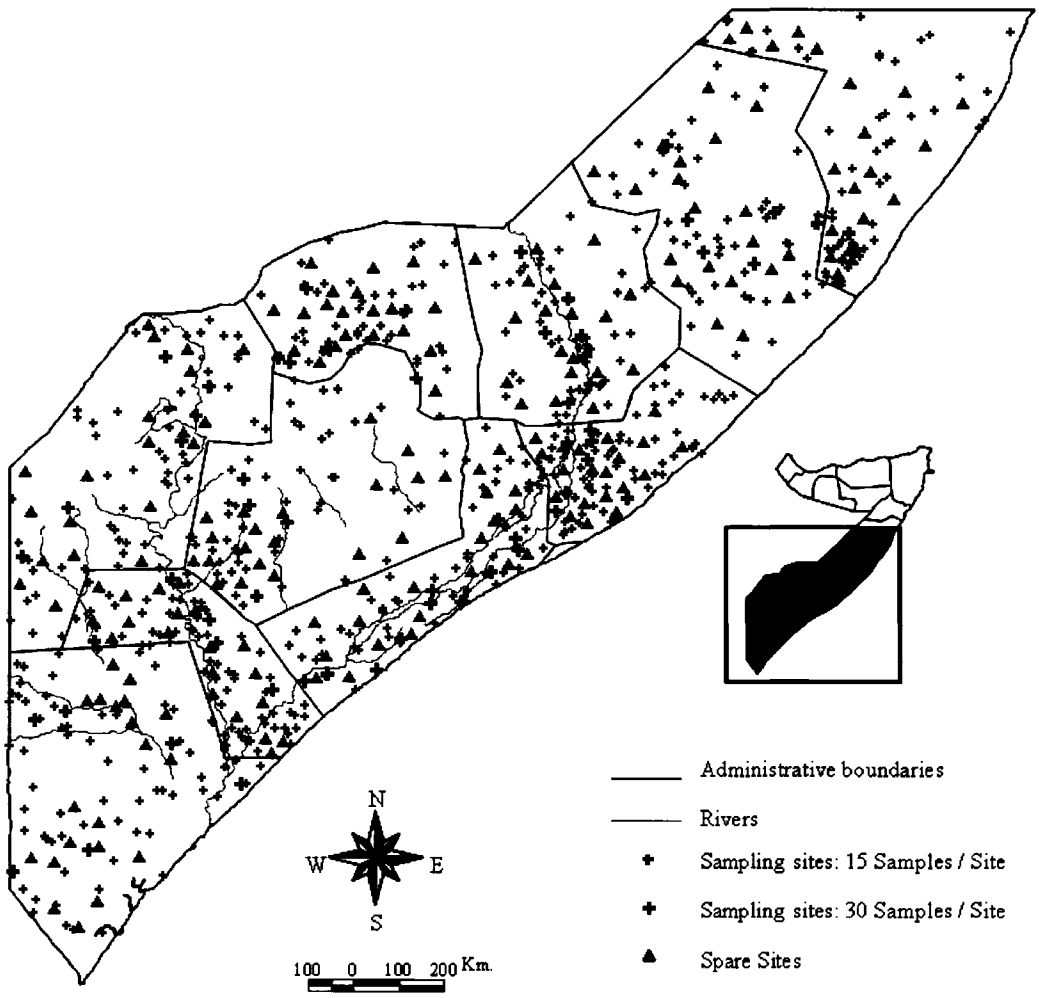

FIGURE 3.4: RP survey – Location of target sampling sites.

3.4. Set-up of the survey

Specifically designed questionnaires were prepared in order to gather information on: (i) livestock species owned (e.g. cattle, sheep, goat and camel); (ii) herd / flock size of each livestock species owned by the key informant; and (iii) location of the herd / flock during each season over the two years prior to the time of the survey.

The total number of serum samples had to be collected by 20 contracted private investigating teams, 2 per administrative region (TABLE 3.5). Each team was comprised of a team leader, an assistant, and a monitor, which operated under the

close supervision of permanent PACE staff. The task of the team leader and the assistant was to reach the selected sampling sites, bleed the required number of eligible animals, and complete specifically designed survey forms and questionnaires.

The monitor was tasked to assist the team leader in performing his duties, as well as dispatch all collected samples, forms, and questionnaires to the PACE Zonal Offices every 3 days.

TABLE 3.5: Number of sampling sites and serum samples by team.

Zone	*Team ID*	*Sampling Area*	*Total Num. of Sampling Sites*	*Num. of Sampling Sites (15 Samples)*	*Num. of Sampling Sites (30 Samples)*	*Total Num. of Samples*
Central Somalia	CA	Hiran (North)	26	22	4	450
	CB	Hiran (South)	26	24	2	420
	CC	Middle Shabele (East)	37	31	6	645
	CD	Middle Shabele (West)	37	36	1	570
	CE	Galgadud (North)	26	23	3	435
	CF	Galgadud (South)	26	23	3	435
	CG	Mudug (South)	28	26	2	450
	CH	Mudug (North)	27	27	-	405
Southern Somalia	SA	Bay (East)	26	25	1	405
	SB	Bay (West)	26	23	3	435
	SC	Bakool (East)	25	23	2	405
	SD	Bakool (West)	25	20	5	450
	SE	Lower Shabele (East)	32	28	4	540
	SF	Lower Shabele (West)	32	25	7	585
	SG	Middle Juba (North)	37	33	4	615
	SH	Middle Juba (South)	36	35	1	555
	SI	Lower Juba (North)	35	32	3	570
	SL	Lower Juba (South)	35	32	3	570
	SM	Gedo (North)	29	25	4	495
	SN	Gedo (South)	29	27	2	475
	TOTAL		**660**	**600**	**60**	**9900**

The needed number of pre-labeled cryovials was prepared prior to the survey for each team. The labeling included a progressive number starting from 1, as well as the identification code of each team. Since each serum sample had to be split into two aliquots, two series of cryovials bearing the same code were prepared for each team.

Sampling forms were designed to record information related to the sampling site and owner's name, as well as sex, age, and clinical signs (if any) of the bled animals.

3.5. Implementation of the survey

An initial training on *"Basic Applied Epidemiology Focused on Rinderpest Sero-Surveillance"* was carried out for team leaders and monitors at the PACE Zonal Offices.

The survey teams were provided with a GPS (Garmin III Plus®) where all coordinates identifying the sampling sites for each team were entered during the initial training.

Each sampling site, identified by a number, was plotted on a map that assisted the investigating teams to reach the neighborhood of the target site. Then, the teams used the GPS to navigate their way to the target sampling sites.

At each specific target site, the investigating teams identified the closest herd. The distance around the target site in which animals could be bled was set at 10 km radius.

Once the herd was identified the coordinates of the actual sampling site were recorded and a total of 15 eligible animals (i.e. cattle > 1 to 3 years old) were bled. In order to allow for the possibility of poor clotting or spoilage of samples, another two animals were bled at each site. Thus each team was expected to collect a total of 17 samples at each site.

If no herds or animals were found within the specified radius (10 km) the target sampling site was replaced by a "spare" location.

Serum samples were collected using plain vacutainers (10 ml) and sterile needles (20 G; one for each animal) and were allowed to clot for a maximum of 24 hours in the shade or a cool place.

Two aliquots of sera were transferred into the pre-labelled cryovials. Sera were decanted using sterile disposable Pasteur pipettes (3.5 ml).

Serum samples collected at each site were packed and labeled according to standards defined at the initial training course and stored in vaccine carriers until they were dispatched to the PACE Zonal Offices by the monitors. Serum samples had to be accompanied by original sampling forms and questionnaires.

Each sample collected was checked at the Zonal Offices for quality and quantity. Only samples collected according to standards and accompanied by properly filled sampling forms and questionnaires were retained and then dispatched to the KARI - Muguga Laboratory in Nairobi – KENYA.

All data recorded in the sampling forms and questionnaires were entered into specifically designed databases (Microsoft Access XP; Microsoft Corporation®).

The actual sampling sites were plotted on ArcView maps and compared to the target sites in order to monitor the accuracy of field operations.

The cold chain was guaranteed by vaccine carriers. Frozen ice packs were supplied by the monitors to the investigation teams every 3 days for replenishment.

3.6. Testing of the samples

All collected serum samples were tested for the presence of RP antibodies at the KARI - Muguga Laboratory in Nairobi – KENYA using an anti H Competitive ELISA. According to the protocol of the test, all samples having a percentage of inhibition (PI) > 50 were considered as a positive. The test is the one recommended by the OIE to screen large number of sera.

3.7. Evaluation of the survey

Field operations lasted for 37 days from the first day of training to the last day of samples collection in the field. The number of visited sites and collected samples is given in TABLE 3.6, while TABLE 3.7 provides the total number of questionnaires properly completed by Region.

TABLE 3.6: Total number of sites visited and samples collected against the target by region.

Region	*Number of Sampling Sites*				*Number of Samples per Site*				*Num. of Samples*	
	Cattle 1 to 3 Years	*Cattle all Age Groups*	*Total*	*% of the Target*	*Cattle 1 to 3 Years*		*Cattle all Age Groups*		*Total*	*% of the Target*
					Min.	*Max.*	*Min.*	*Max*		
Bakool	42	7	49	98.0	15	17	30	34	911	106.5
Bay	44	3	47	90.4	15	19	32	32	844	100.5
Galgadud	40	5	45	86.5	15	17	30	34	841	96.6
Gedo	51	6	57	98.3	15	17	32	34	1052	109.6
Hiran	44	6	50	96.2	10	17	30	34	925	106.3
L. Juba	56	8	64	91.5	15	17	30	34	1201	105.3
L. Shabele	50	11	61	95.3	15	17	32	34	1201	106.7
M. Juba	67	4	71	97.3	14	18	34	35	1256	107.3
M. Shabele	60	6	66	89.2	15	17	32	34	1186	97.6
Mudug	50	2	52	94.5	15	17	30	30	870	101.4
TOTAL	**504**	**58**	**562**	**93.5**	**10**	**19**	**30**	**35**	**10287**	**103.9**

TABLE 3.7: Number of properly filled questionnaires by region.

Zone	Region	Num. of Questionnaires	Region	Num. of Questionnaires
Central Somalia	Galgadud	61	Middle Shabele	73
	Hiran	72	Mudug	72
Southern Somalia	Bakool	55	Lower Juba	64
	Bay	63	Lower Shabele	61
	Gedo	59	Middle Juba	71
TOTAL 651				

FIGURE 3.5 provides the actual sampling site of the survey.

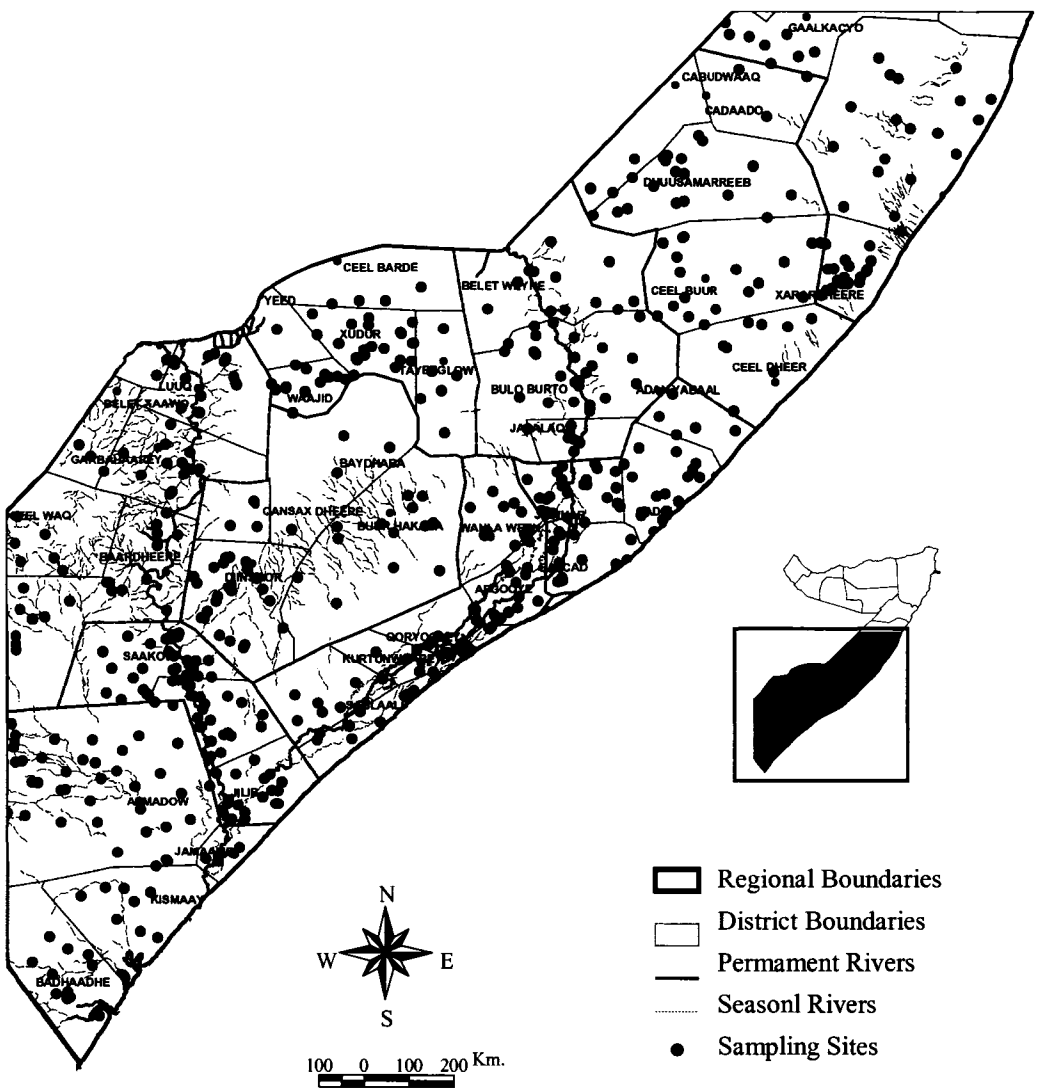

FIGURE 3.5: RP survey – Location of actual sampling sites.

3.8. Conclusions

This study represents the first structured large scale cross-sectional sero-survey carried out in Somalia over the past two decades.

The use of random map coordinates does not need any inventory of animal populations, since the sampling frame is represented by all possible coordinates that can be generated inside a specific area of interest, overcoming one of the major problems that random sampling often poses.

Ninety three percent of the target primary sampling units were reached and 104% of the target number of samples was properly collected. The failure to reach a few sampling sites (6.5%) was mainly due to open conflict or the prevailing insecurity in the area. However, the success rate indicates that the methodology is applicable and that it can be successfully implemented by local professionals if properly trained. The field implementation was in fact carried out by contracted Somali veterinary professionals who operated under the close supervision of the project staff.

Worldwide it has been estimated that there are between 50 and 100 million nomadic or transhumant pastoral people (Omar, 1992). Pastoralists manage over 120 million cattle or cattle equivalent units of livestock (McDowell, 1980), and they own most of the livestock in many countries of Africa including Algeria, Morocco, Niger, Senegal, Mali, Mauritania, Libya, Tunisia, Nigeria, Sudan and Somalia. The proposed approach may be utilized as a model to generate reliable animal data in other nomadic pastoral areas of Africa in order to enable decision makers to better address priority diseases and potentially, in the long term, to access more lucrative markets (e.g. developed countries).

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CHAPTER 4:
EXPLORATORY SPATIAL ANALYSIS OF RINDERPEST SERO-PREVALENCE IN CENTRAL AND SOUTHERN SOMALIA

4.1. Introduction

RP is an acute or subacute contagious viral disease of cattle and other *Artiodactyla* characterized by necrosis and erosions in the gastrointestinal tract that result in severe diarrhea and dehydration. Morbidity and mortality rates may exceed 90%, but inapparent infections may also occur. The decimation of cattle populations by RP has influenced events in human history (Gamgee, 1866; Ofcansky, 1981).

After its first introduction into Africa through Egypt from a ship at Alexandria in 1841, RP has been considered responsible for the decimation of cattle and wildlife up to the extent that this may have altered the ecology of large areas of southern, central and east Africa (Stevenson-Hamilton, 1911; Taylor & Watson, 1967).

The first coordinated effort to eradicate RP from the continent started in 1962 under the Joint Program 15 (JP15) followed in 1987 by the Pan-African Rinderpest Campaign (PARC) and then by the current Pan-African Control of Epizootics (PACE). In 1994, FAO established a new priority program, *Emergency Prevention System for Transboundary Animal and Plant Pests* (EMPRES), of which the initial trust was RP with the *Global Rinderpest Eradication Programme* (GREP) in

conjunction with the OIE, and a target date for the worldwide eradication by the year 2010.

Until 1994, when a mild form of RP was detected and diagnosed in the Tsavo East National Park and subsequently in the Nairobi National Park (1994-96), the main endemically infected area in East Africa was believed to be southern Sudan. It is believed that the source of infection from southern Sudan regularly invaded adjacent areas of Uganda, Kenya and occasionally Ethiopia. All virus isolates recovered from southern Sudan and these neighboring areas since 1983 were of the African type 1 lineage.

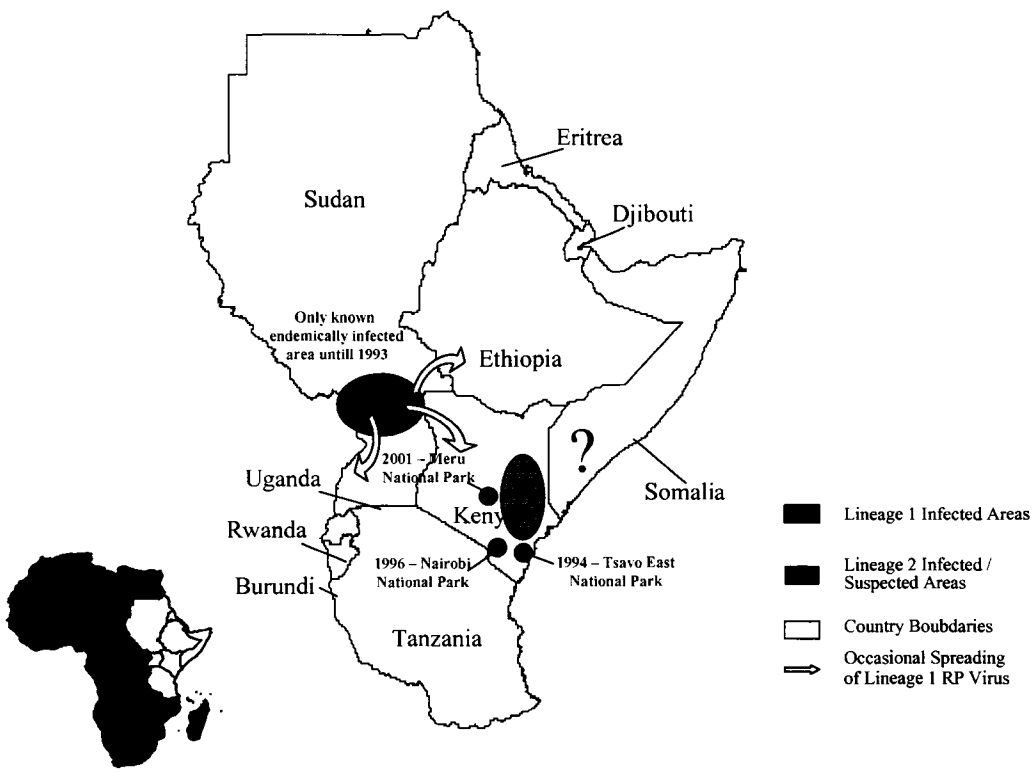

FIGURE 4.1: Rinderpest infected / suspected areas in East Africa after the RP African Type 2 Lineage outbreaks in 1994 – 96 and 2001 in Kenya.

Initially the Tsavo RP outbreak was thought to have originated from southern Sudan but the molecular evidence clearly showed that the Tsavo virus and the isolates from

Nairobi National Park were completely different genetically and fell into the African type 2 lineage. Isolates of this lineage had been recovered from West Africa as late as 1983 but not since 1962 in East Africa. Thus a second main focus of RP in East Africa was revealed after having remained undetected throughout the period of the JP15 campaign and eight years of PARC. The exact location of this focus was uncertain but surveillance had concentrated on north eastern Kenya and southern Somalia (Barrett *et al.*; 1998) (FIGURE 4.1). In October – November 2001 an outbreak of mild RP was again detected and confirmed in buffalo in the Meru National Park, but no "evidence" of RP virus circulation was found in domestic animals (MoARD, 2001) (FIGURE 4.1).

At present it is believed that the only suspected focus of RP circulation in the world is situated in the so called Somali Eco-System, which encompasses Somalia and the Somali inhabited areas of Kenya and Ethiopia. However, only limited and scanty information on the real extension of virus circulation in Somalia have been generated mainly due to the widespread insecurity in the country since the collapse of the government in 1991. Moreover, this particular strain induces very mild or undetectable clinical reaction in cattle, while it appears to induce more severe signs in wildlife. Kock (2000) considers that, in cattle populations, a strain of this lineage causes only mild disease which is often missed by herders and is thus considered of no economic importance to them.

The behavior of this virus increases considerably the difficulties of detecting clinical cases of the disease especially when almost undetectable signs must be noticed in the very mobile nomadic cattle herds of the Somali inhabited areas of the Horn of Africa.

A large scale cross-sectional sero-survey was conducted in 10 administrative regions of central and southern Somalia in order to identify the last remaining foci of RP circulation in the country. The aim of the study was to enhance the understanding of the epidemiology of the disease in the area and geographically differentiate the remaining foci of infection for subsequent elimination.

4.2. Materials and Methods

4.2.1. Survey design and implementation

The detailed description of the design, implementation and evaluation of the study is provided in Chapter 3.

Briefly, the study consisted of a cross-sectional sero-survey. The sample size was calculated for a two-stage cluster sampling, and the randomization of the primary sampling units was obtained by randomly generating map coordinates. The between cluster variance (V_c) in the study area was calculated according to Thrusfield (1998) utilizing data collected from previous studies (Terra Nuova, 1999 & 2001) and then used for the computation of the final sample size for each administrative region. The sample size was calculated for: (i) 50% expected prevalence, (ii) 95% confidence interval, and (iii) 5% desired absolute precision. A total of 9,000 serum samples had to be collected in duplicate in 600 randomly selected sampling sites.

In each sampling site, at least 15 eligible animals (e.g. cattle 1 to 3 years old) were to be bled. The purpose of this selection was to identify relatively recent circulation of RP virus and to avoid bleeding animals that could still possess maternal antibodies or had been vaccinated during previous RP vaccination campaigns. In Somalia, the most recent RP vaccinations were carried out in 1998-99 in the Trans-Juba Regions (e.g.

Lower Juba, Gedo and part of Middle Juba) (Terra Nuova, 1999). The total number of samples was proportionally allocated according to the cattle population density of each region of interest. Ten administrative regions were included in the study (e.g. Bakool, Bay, Galgadud, Gedo, Hiran, Lower Juba, Lower Shabele, Middle Juba, Middle Shabele and Mudug south of Galkayo). The study area is shown in FIGURE 4.2.

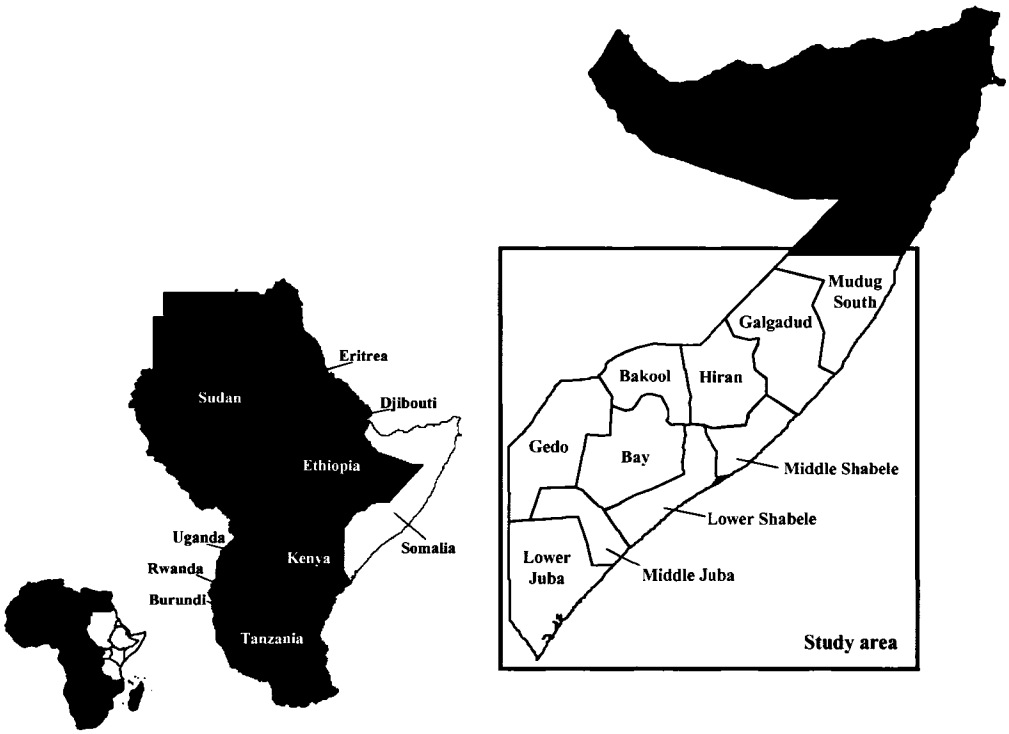

FIGURE 4.2: Study area

The area of intervention was selected according to the cattle and wildlife density of the country as well as the "evidence" of RP viral activity in the past two decades. A further proportional allocation was carried out within region according to the areas of "high" and "low" cattle density.

In addition to the 15 blood samples per sampling site, 15 blood samples were collected using 5 samples from each of the following age groups: (i) 3-4 years old, (ii)

4-5 years old, and (iii) > 5years old. A total of 60 sampling sites (10% of the total number of sampling sites) were included in this collection. Age stratification was used in order to gather information on past exposure of the cattle population to RP. However, the results obtained from the animal older than 3 years will not provide any accurate estimate of the sero-prevalence in these age groups.

Ten spare coordinates for each sampling team were additionally generated to allow for the possibility of not reaching a specific sampling site or not finding animals in it. The total number of serum samples was collected by 20 (two per administrative region) contracted private investigating teams. Each team, composed of a team leader, an assistant and a monitor, operated under the close supervision of permanent PACE staff. The task of the team leader and the assistant was to reach the selected sampling sites, bleed the required number of eligible animals and complete specifically designed questionnaires. The monitor had to assist the team leader in performing his duties as well as to dispatch all collected samples, forms and questionnaires to the PACE Zonal offices every 3 days.

When a specific target site was reached, the investigating team had to look for herd(s) closest to the target bleeding point. The distance around the target site in which animals could be bled was set at 10 km radius. If no animals could be found or the site was not reachable, then a spare site had to be used instead. When the herd was identified, the coordinates of the actual sampling site were recorded and a total of 16 to 17 eligible animals were bled in order to allow for poor clotting or spoilage of samples.

All collected sera were tested for the presence of RP Antibodies at the KARI - Muguga Laboratory in Nairobi – KENYA using a RP Competitive Enzyme Linked

Immunosorbent Assay (C-ELISA) directed against the H protein of the virus (Anderson *et al.*, 1991). According to the protocol of the test, all samples having a percentage of inhibition (PI) > 50 were considered as a positive. The test is the one recommended by the OIE to screen large number of sera.

4.2.2. Data analysis

The survey data were analyzed using the "`svymean`" function of STATA® 8.0 SE. The observed between cluster variance was taken into account in the analysis of the data for the calculation of the standard error used for the construction of the confidence intervals.

The observed RP sero-prevalence was displayed using a Choropleth Map, and a Box Map (Hinge: 3.0) was created to investigate the presence of outliers (Anselin 1994 & 1999).

In order to test the spatial autocorrelation of the observed sero-prevalence, several spatial weight matrices were created. The 4th order rook contiguity weight matrix that included all lower order contiguities was utilized for the analysis.

The significance of the "global spatial autocorrelation" of the data was tested using a Moran's I statistic (Moran, 1948) and visualized in the form of a Moran Scatter Plot (Anselin, 1995 & 1996). The scatter plot displays the standardized variables so that the units in the graph correspond to standard deviations. The significance of the univariate Moran's I was tested against a reference distribution generated from 999 random permutations (Anselin *et al.* 2002).

The reference distribution was calculated for spatially random layouts with the same data (values) as observed. The randomization used an algorithm to generate spatially random simulated data sets as outlined by Anselin (1986). The pseudo-significance level (p) was computed as the ratio of the number of statistics for the randomly generated data sets that were equal to or exceeded the observed statistic + 1, over the number of permutations used + 1.

A Local Indicator of Spatial Autocorrelation (LISA) was used to detect "local spatial dependency" of the data (Anselin, 1995). This yields a measure of spatial autocorrelation for each individual "location" that was computed using the same spatial weight matrix. The types of spatial association were displayed in form of a LISA Cluster Map and the significance levels in the form of a LISA Significance Map. Once again the significance levels were computed by comparing the local Moran to reference distributions obtained from 999 random permutations. Mapping and spatial analysis were carried out using ArcGIS® 8.3 and GeoDa® 0.9.5-i respectively.

4.3. Results

The detailed evaluation of the sero-survey is provided in Chapter 3. Briefly, field operations lasted for 37 days from the first day of training to the last day of samples collection in the field. A total of 9,216 serum samples (102.4% of the target) were properly collected from cattle aged 1 to 3 years in 562 sampling sites (93.6% of the target). In addition, 1,071 sera (119.0% of the target) were collected from cattle older than 3 years in 58 sampling sites (96.6% of the target). A certain number of sampling sites could not be reached due to the prevailing insecurity of the area or inaccessibility of the location and they could not be replaced. However, 2.4% and 19.0% of sera

were collected in excess to the target from cattle aged 1 to 3 years or older than 3 years respectively. Although not all target sampling sites could be reached, an "excess" of sera to the target was collected. This was due to the fact that all samples properly collected in each site in excess to the target (e.g. 15) were retained and tested.

TABLE 4.1 provides the observed sero-prevalence and 95% confidence intervals for young (1 to 3 years) and old (> 3 years) animals.

TABLE 4.1: Observed RP sero-prevalence and 95% confidence interval for young (1 to 3 years) and old (> 3 years) animals in the study area.

Region	*Cattle 1 to 3 Years*		*Cattle > 3 Years*	
	Prev. (%)	*95% CI (%)*	*Prev. (%)*	*95% CI (%)*
Bakool	0.6	(0.2; 1.4)	0.0	(0.0; 5.9)
Bay	0.7	(0.3; 1.6)	3.3	(0.0; 9.0)
Galgadud	1.3	(0.6; 2.4)	0.0	(0.0; 9.9)
Gedo	17.8	(15.3; 20.7)	15.1	(6.9; 23.2)
Hiran	4.3	(2.9; 6.0)	7.5	(2.7; 12.3)
Lower Juba	16.9	(14.6; 19.6)	18.6	(11.4; 25.8)
Lower Shabele	2.6	(1.7; 3.8)	2.9	(0.3; 5.6)
Middle Juba	16.0	(13.8; 18.4)	16.3	(0.0; 33.1)
Middle Shabele	0.6	(0.3; 1.3)	1.0	(0.0; 2.9)
Mudug	0.4	(0.1; 1.1)	0.0	(0.0; 3.7)
TOTAL	**6.8**	**(5.8; 7.8)**	**6.8**	**(4.7; 8.9)**

Since the sample size for animals older than 3 years was not calculated to provide any accurate estimate of the sero-prevalence in these age groups, only the data obtained from young (1 to 3 years) animals were used in the spatial analysis.

The geographical distribution of the observed sero-prevalence is shown in FIGURE 4.3. FIGURE 4.4 shows the Box Map (Hinge: 3.0) of the observed sero-prevalence values. The Box Map was created to highlight outlier values that might have an effect on further analysis.

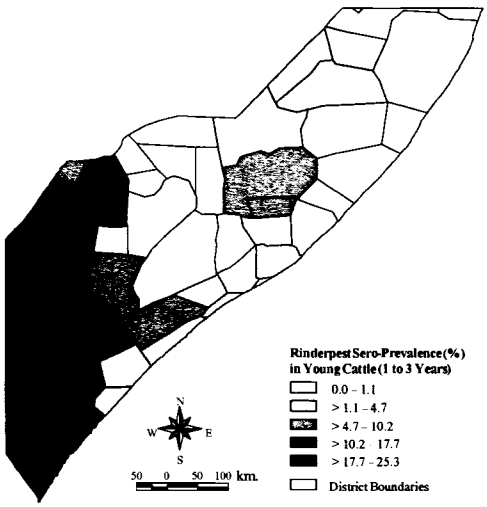

FIGURE 4.3: Geographical distribution of the observed RP sero-prevalence in the study area.

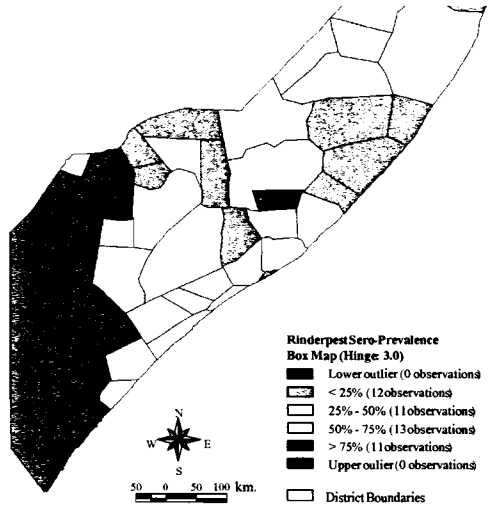

FIGURE 4.4: Box map (Hinge: 3.0) of the observed RP sero-prevalence in the study area.

No outlier values were detected. The global spatial dependency of the data was tested by the mean of a Moran's I statistic (FIGURE 4.5). The significance of the Moran's I was tested against a reference distribution obtained from 999 random permutations (FIGURE 4.6).

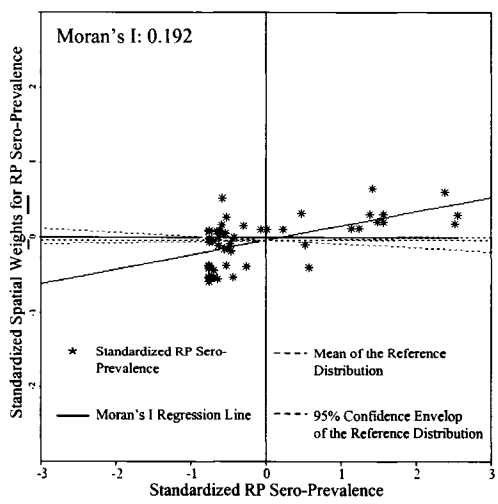

FIGURE 4.5: Moran's I scatter plot for the standardized values of the observed RP sero-prevalence.

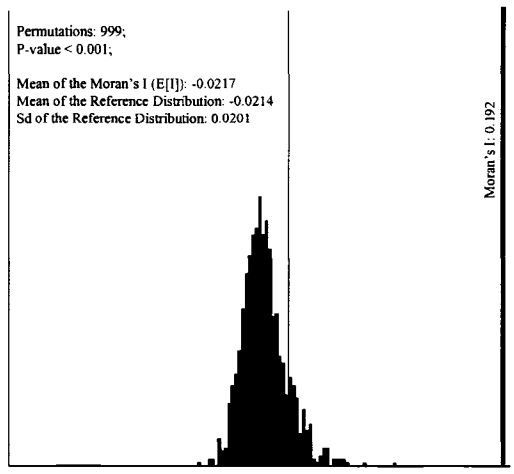

FIGURE 4.6: Reference distribution (999 random permutations) utilized to test the significance of the Moran's I.

The Moran's I indicates a significant global spatial dependency ($p < 0.001$, FIGURE 4.6) of the observed sero-prevalence in the study area. The local spatial dependency was tested using a LISA statistic. FIGURE 4.7 shows the LISA Cluster Map of the local spatial associations for the observed sero-prevalence. The significance levels of the individual spatial associations are given in FIGURE 4.8. Their significance was tested against reference distributions obtained from 999 random permutations.

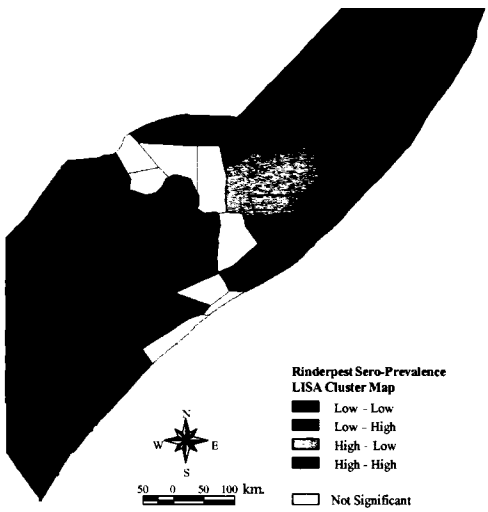

FIGURE 4.7: LISA cluster map of the local spatial dependency of the observed sero-prevalence in the study area.

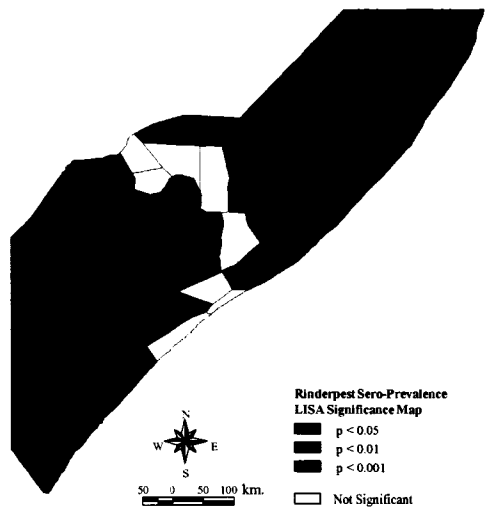

FIGURE 4.8: LISA significance map of the local spatial dependency of the observed sero-prevalence in the study area.

4.4. Discussion and Conclusion

This work represents the first structured large scale cross-sectional study carried out in the cattle rearing areas of Somalia over the past two decades. The prevailing insecurity conditions that have characterized Somalia since 1991 were one of the main reasons that prevented the implementation of epidemiological investigations in the country. However, the absence of veterinary services since the government collapse and the lack of qualified veterinary professionals are also important factors that negatively affected the implementation of a structured survey. Moreover, the

husbandry system of Somalia is characterized by the high mobility of the Somali herds / flocks which are constantly moved by the Somali nomadic pastoralists in search of water and better pasture. Under this scenario the application of a randomized survey in order to collect statistically valid data has presented a challenge for decades.

This study has shown that conventional investigation methodologies can be adapted and successfully applied in nomadic husbandry conditions and in areas of prevailing insecurity. The great involvement of well trained Somali veterinary professionals guaranteed the access to even very insecure areas. Only 6.5% of the target sampling sites could not be reached. However, sera in excess of the target quote were collected in the visited sites using a structured methodology.

Different RP sero-prevalences have been observed in the investigated areas. The highest values were detected in three administrative regions (e.g. Lower Juba, Middle Juba & Gedo) that border Kenya to the west (FIGURE 4.3). Intermediate sero-prevalence was identified in Hiran and partially Lower Shabele, while the remaining regions were almost negative (TABLE 4.1). It is interesting to note that in the latter regions almost no antibodies could be detected even in the older age groups (> 3 years) indicating that RP had not occurred for several years in these areas. However, since the sample size for animals older than 3 years was not calculated to provide any accurate estimate of the sero-prevalence, the information derived from this age group should be interpreted with caution.

The study has highlighted areas of potential RP circulation and maintenance. Past evidence of viral activity was detected in these same areas (Flanagan & Mariner,

1993; Mariner and Roeder, 2003; Terra Nuova, 1999; Terra Nuova, 2001). However, the highest observed prevalence did not exceed values of about 17%.

RP is a highly contagious viral disease of cattle and other *Artiodactyla*, whose morbidity and mortality rates may exceed 90%. Infected animals mount a vigorous response against the virus. Viral antigens are produced in large amounts throughout the lymphoid tissues and affected epithelia (Nakamura, 1957; Rossiter & Jessett, 1982; Scott, 1967; Scott & Brown, 1961) and stimulate an effective antibody response which begins two to five days after the onset of clinical disease in virulent infections, and some six to ten days after infection with mild or avirulent strains (Johnson, 1962; Nakamura, 1940; Plowright & Ferris, 1959; Scott & Brown, 1958; Walker *et al.*, 1946). The earlier response consists predominantly of IgM antibodies (Anderson & Rowe, 1982; Okuna & Rweyemamu, 1974) followed by IgG, which usually persist for life (Plowright 1984; Plowright & Ferris, 1962; Rweyemamu *et al.*, 1974). Generally high titers (10^2-10^3 $\log_{10}$ VN_{50}) of neutralizing antibodies remain at easily detectable levels (in excess of 10^1 $\log_{10}$ VN_{50}) for the rest of the animal's life (Plowright, 1984; Plowright & Ferris, 1962; Plowright & Taylor, 1967).

The "low" observed prevalence could be attributed to the high mortality rate of the disease, which after an epidemic would leave only few "survivors" that bear detectable neutralizing antibodies. However, even when moderate to highly virulent strains are involved in epidemics, sero-prevalence levels up to 77.4% could be detected (Majok *et al.*, 1991). In Somalia, no major clinically detectable outbreaks have been reported for the past decade, and RP is not reported as a disease of concern by Somali pastoralists (results not shown).

The 1994-96 (Barrett *et al.*, 1998; Kock *et al.*, 1999) and 2001 (MoARD, 2001) outbreaks in Kenya were clinically evident in wildlife, but the cattle population seemed not to be reacting at clinically detectable levels. The ease with which the disease is transmitted depends upon the strain of the virus (Scott, 1955). Transmission is believed to be by droplet, either in the breath of an infected animal or in its secretions and excretions, but close contact is required lest the virus be destroyed in passage.

Since this RP strain induces very mild or unapparent clinical signs, the amount of virus shed by infected animals could be markedly reduced, lowering in return the morbidity rate of the disease. Under these circumstances, this strain might circulate unnoticed in the Somali cattle herd and induce only low sero-prevalence after which an outbreak might start to fade.

The spatial analysis has highlighted a significant global spatial dependency of the observed sero-prevalence (FIGURES 4.5 & 4.6). The local high-to-high and high-to-low spatial dependences detected by the LISA test indicate the existence of two potential clusters of infection in the country (e.g. Lower Juba, Middle Juba, Gedo and part of Lower Shabele: Cluster 1 & Hiran: Cluster 2) (FIGURE 4.7). On the other hand, the significant Low-to-High and Low-to-Low local spatial autocorrelation might indicate that the remaining investigated areas of the country were free from infection. This is further supported by the absence of neutralizing antibodies in the old animals in the same areas. However, since RP is predominantly transmitted by close contact between infectious and susceptible animals, in the complete absence of physical barriers, movement restrictions, quarantine or vaccination interventions in

Somalia, it would be expected that the disease would have spread to some or all cattle areas of the country.

Nevertheless, cattle density and mobility of the Somali herds are not homogeneous across the investigated areas. The influence of these two parameters among others on the observed RP sero-prevalence distribution should be further investigated. The identification of the main risk factors for RP maintenance and spread will considerably enhance the understanding of the epidemiology of the disease in the area and will allow more focused eradication interventions.

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CHAPTER 5:
DETERMINATION OF RISK FACTORS FOR RINDERPEST OCCURRENCE IN CENTRAL AND SOUTHERN SOMALIA

5.1. Introduction

RP is currently the subject of a major international eradication effort coordinated by FAO in conjunction with the OIE with a target date for the worldwide eradication by the year 2010 (Anon., 1997). At present it is believed that the Somali pastoral areas of East Africa (which encompasses Somalia and the Somali inhabited areas of Kenya and Ethiopia) maintain the last foci of RP in the world (Mariner *et al.* 2005).

The data obtained from a large scale cross-sectional sero-survey conducted in ten administrative regions of central and southern Somalia indicate the existence of two potential clusters of infection in the country (Chapter 4). However, in the absence of physical barriers, movement restrictions, quarantine or vaccination interventions, it would be expected that the disease would have spread to some or all cattle areas of the country.

In fact, RP virus has a short direct cycle of infection and is spread by close contact (Cooper, 1932; Idnani, 1944; Taylor *et al.*, 1965). As originally noted by Lowe (1942), the spread of RP is like a bushfire; it continually moves on the new uninfected animals leaving behind dead or immune animals. The disease stops when it runs out

of susceptibles. Therefore RP is maintained by large, heterogeneous populations with a sufficient supply of susceptible animals.

The high mobility of the nomadic Somali herds, which constantly move in search of water and better pasture, represents an ideal environment for the maintenance of RP. Nevertheless, cattle density and herd mobility are not homogeneous across the country. Approximately 60% of the Somali population is nomadic pastoralists. Nomadic pastoralism uses herd diversification (in terms of livestock species) and herd dispersion as a risk-reduction strategy in case of local forage shortage, raising, disease outbreaks, etc. and it is characterized by the large sizes and high mobility of the herds. Beside the nomadic pastoralism, agro-pastoral and settled mixed farming systems exist in the country. They represent approximately 25% of the Somali population and, contrary to nomadic pastoralism, agro-pastoral and mixed farming systems are characterized by low mobility and small herd size.

In order to identify potential risk factors for the occurrence of the disease, the sero-prevalence of RP observed in the study area was modeled using logistic regression models. Geographic Information Systems (GIS) were used to model the mobility of the herds as well as to derive other spatial variables that were then included in the regression analysis.

5.2. Materials and Methods

5.2.1. Survey design and implementation

The detailed description of the design, implementation and evaluation of the study is provided in Chapter 3.

In summary, the study consisted of a cross-sectional sero-survey. The sample size (for: (i) 50% expected prevalence, (ii) 95% confidence interval, and (iii) 5% desired absolute precision) was calculated for a two-stage cluster sampling design, and the primary sampling units were obtained by randomly generating map coordinates.

The total number of samples (n = 9,000) was assigned to 600 randomly selected locations which were proportionally allocated according to the cattle population density of each region of interest. Ten administrative regions were included in the study (e.g. Bakool, Bay, Galgadud, Gedo, Hiran, Lower Juba, Lower Shabele, Middle Juba, Middle Shabele and Mudug south of Galkayo).

In each sampling location the coordinates of the bleeding site were recorded, a total of 16 to 17 eligible animals (1-3 years old) were bled in order to allow for poor clotting or spoilage of samples, and specifically designed questionnaires were administered to the livestock owners of the sampled herds. The information collected from the livestock owners included: (i) livestock species owned (e.g. cattle, sheep, goat and camel); (ii) herd / flock size of each livestock species owned by the key informant; and (iii) location of the herd / flock during each season over the two years prior to the time of the survey. The age and sex of each sampled animal were also recorded.

All the collected sera were tested for the presence of RP Antibodies at the KARI - Muguga Laboratory in Nairobi – KENYA using a RP Competitive Enzyme Linked Immunosorbent Assay (C-ELISA) directed against the H protein of the virus (Anderson *et al.*, 1991). As per test's protocol and the OIE recommendations, all samples having a percentage of inhibition (PI) > 50 were considered as a positive.

5.2.2. Selection of the explanatory variables

The selection of the independent variables was dictated by the way of transmission of the disease. RP is a highly contagious disease which is mainly transmitted by close contact between infected and susceptible animals (Idnani, 1944). Animal movements, population density, cattle herd size and proximity of the herds to livestock trade routes are factors that are likely to increase the chances of contact between infected and susceptible individuals increasing in return the probability of disease transmission.

The age of the animal was also considered a factor that could explain the observed sero-prevalence. In fact a higher prevalence of the disease may be expected in older animals that have been exposed to the risk of contracting the disease for a longer period of time than the younger ones.

Sheep and goats are known to be susceptible to RP, and their ability to transmit the disease to cattle is well documented (Scott & Brown, 1961; Bidjeh *et al.*, 1997). In the nomadic system, small ruminants are commonly kept with cattle. Therefore, sheep and goat herd size was also considered as a potential risk factor for the occurrence of RP in the study area. The explanatory variables included in the study are provided in TABLE 5.1.

TABLE 5.1: Explanatory variables considered in the study.

Code	*Variable Description*	*Variable Unit*
Hr	Home range of the herd calculated with the minimum convex polygon method.	km^2
M_Dens	Mean cattle population density of the herd locations over the two years prior to the time of the survey.	Head of cattle / km^2
M_Dist	Mean distance of the herd locations from cattle trade routes over the two years prior to the time of the survey.	km
Age	Age of the animal.	Years
HS_C	Cattle herd size.	Num. of Animals
FS_S	Size of the sheep flock which belongs to the owner of the cattle included in the survey.	Num. of Animals
FS_G	Size of the goat flock which belongs to the owner of the cattle included in the survey.	Num. of Animals

5.2.3. Data analysis

The serological prevalence of each herd was modeled using logistic regression since the response variable is a proportion and the error function is assumed to follow the binomial law (McCullagh & Nelder, 1989). The link function used was the logit function defined as:

$$\text{logit}(p) = \ln\left[\frac{p}{1-p}\right] \qquad [5.1]$$

Since the survey was based on a two-stage cluster sampling design, a "model-based" statistical analysis, which considers that the data are generated from a simple random sampling procedure, was not considered suitable. Instead, a "design-based" analysis, which considers stratification, clustering and statistical weights, was used (Korn & Graubard, 1990; Roberts *et al.*, 1987; Skinner *et al.*, 1989; Thomas & Rao, 1987). Contrary to the conventional χ^2-test which is used in the "model-based" approach, a deviance analysis which utilizes an F-test was instead used to assess the contribution of the different explanatory variables in the model (TABLE 5.1) (Collett, 1991). According to the survey design, the strata in the model were considered to be the *"Administrative Regions"* and the primary sampling unit the *"Sampling Locations"*.

The home-range (Hr) of each herd included in the study was estimated by first obtaining the geographic coordinates of all locations where the herd had been reported to be situated during the different seasons (e.g. Jiilaal: Dec. – Mar.; Gu': Apr. – Jun.; Xagaa: Jul. – Sep. & Dayr: Oct. – Nov.) over the two years prior to the time of the survey.

The geographic coordinates of each reported location were obtained from available databases for Somalia, Kenya and Ethiopia (Africa Data Sampler, 2000; DoRSRR, 2002; UNDOS, 2003). If a particular reported location was not included in the available databases then the location was visited and the geographic coordinates were recorded.

The home range for each herd was then calculated using the Minimum Convex Polygon (MCP) method (White & Garrott, 1990), and a buffer of 5 km around the MCP was created in order to account for the daily mobility of the herds. The area of the home-range polygon was then extracted and used as an explanatory variable in the model.

This variable was created to reflect the mobility of each herd included in the study. FIGURE 5.3b provides an example of the method used to calculate the individual herd home-range.

The mean cattle population density of the herd locations (M_Dens) over the two years prior to the survey was calculated by generating a cattle density map. The cattle population density for each administrative region was derived by cattle growth projections based on the livestock census for Somalia for 1989 (Anon., 1989).

A neighborhood analysis (moving average) was performed on the available data in order to make the cattle density map more realistic (Michel *et al.*, 2002) (FIGURE 5.1). The cattle density in each herd location was then extracted, and the values were averaged and used as an explanatory variable in the model.

This variable was created to reflect the influence of cattle density in maintaining RP in the study area. FIGURE 5.3c provides an example of the method used to calculate the mean cattle density value for the herd locations.

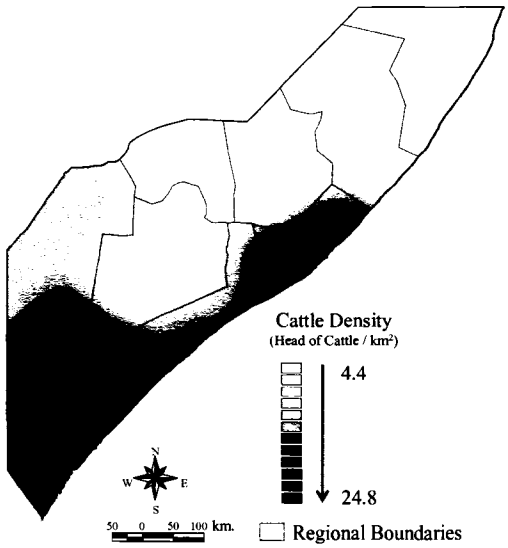

FIGURE 5.1: Map of cattle density.

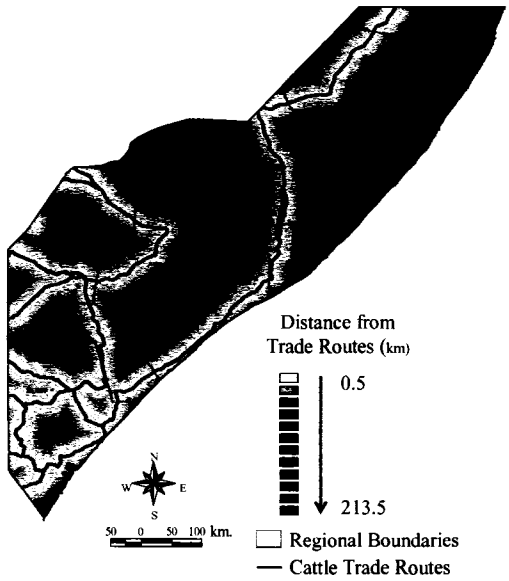

FIGURE 5.2: Map of linear distance from cattle trade routes.

The mean distance of the herd locations from cattle trade routes (M_Dist) over the two years prior to the survey was calculated by first generating a linear distance map (FIGURE 5.2).

The linear distance from each herd location to the closest cattle trade routes was then extracted and the values at each location were averaged and used as an explanatory variable in the model. This variable was created to reflect the influence of cattle trade routes in spreading RP in the study area. FIGURE 5.3d provides an example of the method used to calculate the mean distance of the herd locations from the cattle trade routes.

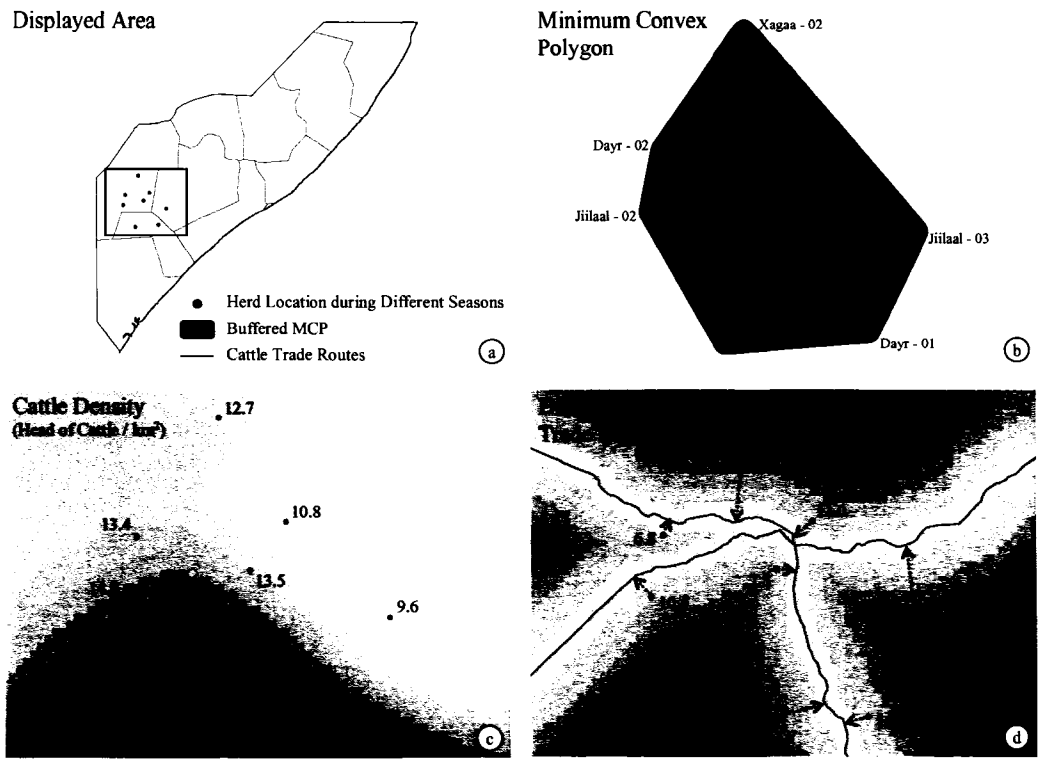

FIGURE 5.3: a) Area displayed in the example; b) Example for the calculation of the individual herd home-range with the MCP method; c) Example of calculation of the mean cattle density (e.g. [12.7 + 13.4 + 13.5 + 10.8 + 14.8 + 9.6 + 20.3 + 10.8] / 8 = 13.2); and d) Example of the calculation of the mean linear distance from herd locations to the closest cattle trade routes (e.g. [43.5 + 6.8 + 11.0 + 10.5 + 7.5 + 32.5 + 47.5 + 10.5] / 8 = 21.2).

All explanatory variables were tested for significance and were entered in the model according to their significance in the univariable logistic analysis. When the preliminary main effects model (containing only the significant variables at $\alpha = 0.05$) was obtained, the linearity of each independent variable to the logit was tested. Since all independent variables were continuous, the fractional polynomial method was used to assess the assumption of linearity. According to the suggestions by Royston and Altman (1994) the power transformations were restricted to the following set:

$\wp = \{-2,-1,-0.5,0,0.5,1,2,3\}$ where $\wp = 0$ denotes the log of the variable

The multivariable fractional polynomial method was implemented in a "model-based" analysis set-up (Royston & Ambler, 1998a, 1998b & 1999; Sauerbrei & Royston,

1999). The suggested transformations of the independent variables were then tested for significance using a "design-based" approach (Hosmer & Lemeshow, 2000).

After the main effects model (containing only the significant variables at the correct power at $\alpha = 0.05$) was obtained, all first order interactions were tested for significance. Each first order interaction was calculated as the product of each pair of independent variables included in the main effects model. The goodness-of-fit of the preliminary final model (containing only the significant variables at the correct power and the significant first order interaction at $\alpha = 0.05$) was then assessed using the Hosmer-Lemeshow (Hosmer & Lemeshow, 1980) and Brown (Brown, 1982) goodness-of-fit tests.

After the final model was obtained, the spatial autocorrelation of the observed and predicted prevalence and the residuals of the model were tested. For the spatial autocorrelation analysis an inverse distance spatial weight matrix was created. The geographic location used for the construction of the matrix was considered to be the centroid of the home-range polygon of each individual herd.

The "global spatial autocorrelation" of the data was tested using a Moran's I statistic (Anselin, 1996; Moran, 1948). The significance of the univariate Moran's I was calculated against a reference distribution generated from 999 random permutations (Anselin *et al.* 2002).

The reference distribution was obtained from spatially random layouts with the same data (values) as observed. The randomization used an algorithm to generate spatially random simulated data sets outlined in Anselin (1986). The pseudo-significance level (p) was computed as the ratio of the number of statistics for the randomly generated

data sets that were equal to or exceeded the observed statistic + 1, over the number of permutations used + 1.

The logistic regression analysis was carried out in STATA® 8.0 SE, while the MCPs were obtained using the Hawthorne's Analysis Tool extension of ArcGIS 8.3. The cattle density and distance maps were created using the Spatial Analyst extension of ArcGIS 8.3. The analysis of the spatial autocorrelation of the residuals of the model was carried out in GeoDa 0.9.5-i.

5.3. Results

The summary statistics of the explanatory variables considered in the study are provided in TABLE 5.2.

TABLE 5.2: Summary statistic of all independent variables considered in the study.

Observations	9216
Strata	10
Primary Sampling Units	562

Variable	*Mean*	*Std. Dev.*	*Min.*	*Max.*	*Unit*
Hr	2300.9	2990.2	79.2	29732.8	km^2
M_Dens	13.4	6.8	4	24	Head of cattle / km^2
M_Dist	43.8	46.3	0.5	190.5	Km
Age	2.0	0.7	1	3	Years
HS_C	46.7	42.4	11	364	Num. of Animals
FS_S	32.5	32.0	0	253	Num. of Animals
FS_G	48.8	52.4	0	407	Num. of Animals

Only Hr, M_Dist and M_Dens were significant at $\alpha = 0.05$. HS_C was not significant at the set value of α ($p = 0.058$) but it was retained in the model because it was considered an important prediction variable. The preliminary main effect model is given in TABLE 5.3.

TABLE 5.3: Preliminary main effect model.

Variables	*Coef.*	*Std. Err.*	*t*	*P > |t|*	*95% Conf. Interv.*
Const.	-3.32178	0.26241	-12.66	< 0.001	-3.83723; -2.80633
Hr	0.00009	0.00002	4.41	< 0.001	-0.00005; 0.00014
M_Dist	-0.00987	0.00249	-3.96	< 0.001	-0.01477; -0.00497
M_Dens	0.05123	0.01464	3.52	< 0.001	0.02260; 0.07987
HS_C	0.00235	0.00124	1.90	0.058	-0.00007; 0.00479

After the preliminary main effect model was obtained, the linearity of the prediction variable to the logit was tested using the multivariable fractional polynomial method. The fractional polynomial fitting algorithm converged after 3 cycles. The suggested fractional polynomial transformations are provided in TABLE 5.4.

TABLE 5.4: Suggested fractional polynomial transformation (where power = 0 is log[variable]).

New Variables	*Suggested Fractional Polynomial Transformation*	*Power*
MDens1	$\left(\frac{M\_Dens}{10}\right)^{-0.05} - 0.8636$	-0.05
MDist1	$\frac{M\_Dist+0.0009}{100} - 0.4387$	1
MDist2	$\left(\frac{M\_Dist+0.0009}{100}\right) * \ln\left(\frac{M\_Dist+0.0009}{100}\right) + 0.3615$	1
Hr1	$\ln\left(\frac{Hr}{10000}\right) + 1.469$	0
Hr2	$\frac{Hr}{10000} - 0.2301$	1
HSC1	$\left(\frac{Hs\_C}{100}\right)^{-1} - 2.14$	-1

The main effect model (TABLE 5.5) was obtained by testing the significance of all variables after the fractional polynomial transformation.

TABLE 5.5: Main effect model.

Variables	*Coef.*	*Std. Err.*	*t*	*P > |t|*	*95% Conf. Interv.*
Const.	-1.85759	0.13619	-13.64	< 0.001	-2.12511; -1.59006
Hr1	0.47108	0.06873	6.85	< 0.001	0.33608; 0.60609
MDens1	-2.18949	0.44148	-4.96	< 0.001	-3.05670; -1.32229
HSC1	-0.19122	0.04980	-3.84	< 0.001	-0.28904; -0.09339
MDist2	-3.87876	1.40945	-2.75	0.006	-6.64731; -1.11021

The preliminary final model (TABLE 5.7 & 5.8) was obtained by testing the significance of all first order interaction (TABLE 5.6).

TABLE 5.6: First order interactions

Variables	*First Order Interactions*
MDens1_MDist2	MDens1*MDist2
MDens1_Hr1	MDens1*Hr1
MDens1_HSC1	MDens1*HSC1
MDist2_Hr1	MDist2*Hr1
MDist2_HSC1	MDist2*HSC1
Hr1_HSC1	Hr1*HSC1

TABLE 5.7: Final model (model coefficients).

Variables	*Coef.*	*Std. Err.*	*t*	*P > |t|*	*95% Conf. Interv.*
Const.	-1.89551	0.12595	-15.05	< 0.001	-2.14291; -1.64811
Hr1	0.57370	0.08492	6.76	< 0.001	0.40689; 0.74052
MDens1	-2.77244	0.69628	-3.98	< 0.001	-4.14013; -1.40475
HSC1	-0.19018	0.04757	-4.00	< 0.001	-0.28363; -0.09673
MDist2	-4.374189	1.37618	-3.18	0.002	-7.07739; -1.67098
MDens1_HSC1	0.27470	0.07183	3.82	< 0.001	0.13392; 0.41549
MDist2_Hr1	-0.99906	0.39183	-2.54	0.011	-1.76705; -0.23108
MDens1_MDist2	3.56041	1.57794	2.26	0.024	0.467705; 6.65312

TABLE 5.8: Final model (odd ratio).

Variables	*Odd Ratio*	*95% Conf. Interv.*
Hr1	1.7748	1.5021; 2.0970
MDens1	0.0625	0.0239; 0.0861
HSC1	0.8262	0.7530; 0.9077
MDist2	0.0125	0.0008; 0.1880
MDens1_HSC1	1.3333	1.0007; 1.7766
MDist2_Hr1	0.3682	0.1708; 0.7936
MDens1_MDist2	35.1777	1.5963; 775.203

Both the Hosmer-Lemeshow (*Num. of groups: 10;* χ^2*: 8.13; df: 8; p: 0.42*) and the Brown (χ^2*: 1.254; df: 1; p: 0.263*) goodness-of-fit tests indicate that the model has a good fit.

The estimated probability and its odd ratio for the 81 combinations of minimum, mean and maximum values of the four variables included in the model (TABLE 5.2) is given in TABLE 5.9. The odd ratio is calculated for each estimated probability to the odd of the probability value derived from the combination of the mean values of the four variables (e.g. 0.1071).

TABLE 5.9: Estimated probability and odd ratio (calculated for each estimated probability to the odd of the probability value derived from the combination of the mean values of the four variables: 0.1071) for the 81 combinations of minimum, mean and maximum values of the four variables included in the model.

[ ] Odd Ratio		Hr								
		Min.			Mean			Max.		
		M_Dens			M_Dens			M_Dens		
HS_C	M_Dist	Min.	Mean	Max.	Min.	Mean	Max.	Min.	Mean	Max.
Min.	Min.	0.004374 [0.04102]	0.004287 [0.04020]	0.004247 [0.03983]	0.009734 [0.09178]	0.009541 [0.08994]	0.009453 [0.08911]	0.017800 [0.16921]	0.017449 [0.16582]	0.017290 [0.16428]
	Mean	0.004925 [0.04621]	0.005191 [0.04872]	0.005319 [0.04993]	0.033071 [0.31935]	0.034809 [0.33674]	0.035639 [0.34507]	0.129298 [1.38654]	0.135385 [1.46203]	0.138270 [1.49819]
	Max	0.002805 [0.02627]	0.002091 [0.01957]	0.001826 [0.01708]	0.000092 [0.00086]	0.000069 [0.00064]	0.000060 [0.00056]	0.000007 [0.00006]	0.000005 [0.00005]	0.000004 [0.00004]
Mean	Min.	0.011477 [0.10840]	0.012631 [0.11944]	0.013201 [0.12490]	0.025320 [0.24255]	0.027827 [0.26726]	0.029062 [0.27947]	0.045702 [0.44716]	0.050124 [0.49270]	0.052295 [0.51523]
	Mean	0.012909 [0.12211]	0.015269 [0.14478]	0.016495 [0.15659]	0.082892 [0.84393]	0.096788 [1.00000]	0.103867 [1.08222]	0.281831 [3.66414]	0.317528 [4.34418]	0.334770 [4.69877]
	Max	0.007379 [0.06941]	0.006188 [0.05814]	0.005705 [0.05358]	0.000244 [0.00228]	0.000204 [0.00191]	0.000188 [0.00176]	0.000018 [0.00017]	0.000015 [0.00014]	0.000014 [0.00013]
Max.	Min.	0.014849 [0.14073]	0.016850 [0.16003]	0.017860 [0.16979]	0.032625 [0.31489]	0.036932 [0.35806]	0.039097 [0.37990]	0.058535 [0.58052]	0.066030 [0.66011]	0.069777 [0.70038]
	Mean	0.016695 [0.15853]	0.020351 [0.19397]	0.022290 [0.21287]	0.105018 [1.09562]	0.125545 [1.34051]	0.136112 [1.47113]	0.337515 [4.75694]	0.383988 [5.82021]	0.406204 [6.38731]
	Max	0.009559 [0.09011]	0.008273 [0.07789]	0.007740 [0.07283]	0.000317 [0.00296]	0.000274 [0.00256]	0.000256 [0.00239]	0.000024 [0.00022]	0.000020 [0.00019]	0.000019 [0.00018]

The residuals of the final model were then tested for spatial autocorrelation using a Moran's I statistic. No spatial autocorrelation was detected (TABLE 5.10).

TABLE 5.10: Spatial autocorrelation test for the observed and predicted prevalence and residuals of the model.

Variables	*Moran's I*	*Pseudo Significance Level (p)*
Observed Prevalence	0.339	< 0.001
Predicted Prevalence	0.610	< 0.001
Model's Residuals	0.012	0.178

5.4. Discussion and Conclusion

The logistic regression analysis has identified and quantified the magnitude (TABLE 5.8) of four main risk factors for RP occurrence in central and southern Somalia, namely: (i) herd mobility (Hr1); (ii) cattle density (MDens1); (iii) cattle herd size (HSC1); and (iv) distance from cattle trade routes (MDist2). These findings are not surprising since RP is mainly transmitted by close contact between susceptible and infected animals. The transmission is believed to be by droplet, either in the breath of an infected animal or in its secretions and excretions (Idnani, 1944), but close contact is required unless the virus is destroyed in passage. All the identified risk factors contribute to the increase of the contact rate between individuals in the population.

Additional ways of contagion like airborn transmissions have been considered. Experimentation has proven that airborne transmission is a theoretical possibility over several hundred meters (Hyslop, 1979), but circumstantial evidence does not support that this type of transmission can play a role in the spread of the disease under natural conditions.

Furthermore, the fragile nature of the virus ensures that most infectivity survives for only a few hours outside the host, though some may persist under favorable

conditions for up to two to four days (Shilston, 1917; Todd & White, 1914). Carcass decomposition inactivates the virus within one to three days (Curasson, 1932).

The ease with which the disease is transmitted depends upon the strain of the virus (Scott, 1955). Virulent strains induce very severe clinical manifestation in the host, which, during the earliest stages of clinical disease, excrete infectious virus in their ocular, nasal, oral, vaginal secretions and faeces (Curasson, 1932; Hall, 1933; Hornby, 1926; Scott, 1964). Those secretions represent the primary ways of contagion between infected and susceptible animals.

However, the clinical form observed in Somalia is extremely mild (African Lineage 2 Type RP virus). Infected animals can hardly be detected, and in most cases the infection passes unnoticed in the herd. Under these circumstances, whereby very little or no clinical reaction (discharges) is induced, a high contact rate between individuals may be necessary to allow effective virus transmission. Sedentary husbandry systems, low cattle density and small herd size may reduce considerably the chances of transmission of the virus. This may explain the absence of RP antibodies in certain zones of the study areas even in the absence of physical barriers, movement restrictions and quarantine or vaccination interventions in the country.

However, proximity to cattle trade routes can considerably increase the risk of contracting the disease. Trade herds are normally large, move quickly, may pass through infected areas and are grazed alongside the trade routes often utilizing the same pasture of the nomadic herds. The animals that are moved for export can therefore play an important role in spreading the disease over long distances in very short time.

Sheep and goats are known to be susceptible to RP and their ability to transmit the disease to cattle is well documented (Scott & Brown, 1961; Bidjeh *et al.*, 1997). In Somalia herd diversification (in terms of livestock species) is a common feature, since it allows pastoralists to better exploit the natural resources available in different environments. It is not rare that sheep and goats are herded by the same family alongside with cattle. However, the size of the herds / flocks and their relative composition in terms of livestock species may vary considerably from area to area. Sheep and goats herd size herded with cattle were considered a potential risk factor in the study, but neither of the two variables were significant in the model.

Very little is known about the susceptibility of small ruminants to the African Type Lineage 2 RP virus, and more investigations should be conducted to rule out the role of sheep and goats in spreading this particular virus.

Three out of nine first order interactions were significant in the model, namely: (i) Cattle Density * Cattle Herd Size; (ii) Distance from Cattle Trade Routes * Herd Mobility (home-range); and (iii) Cattle Density * Distance from Cattle Trade Routes.

The transmission of the virus is likely to occur very efficiently within a herd. Big herds can maintain the disease more efficiently than small herds and induce a large number of infectious animals that in return may transmit to other in-contact herds. The transmission between herds may be facilitated in highly populated areas. Similarly, it can be argued that infection contracted through contact with trade cattle can be spread across the resident population more efficiently in areas where the herd mobility is elevated and / or in highly populated areas.

No spatial autocorrelation for the residuals of the model was detected. This means that the explanatory variables accounted for the spatial dependence identified for the observed and predicted prevalence.

The combined use of GIS and statistical analysis techniques enabled modeling of the complexity of the interactions of the nomadic Somali herds and determining the risk factors for the occurrence of the disease in the country. Taking these factors into consideration will make it possible to design more focused surveillance and eradication intervention strategies. However, particular attention should be paid to the role that trade animals might play in spreading the disease from infected to naïve areas of the country.

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CHAPTER 6:
MAPPING THE RISK OF RINDERPEST OCCURRENCE IN SOMALIA: A PROPOSED ZONATION APPROACH FOR MOBILE HUSBANDRY SYSTEMS

6.1. Introduction

The results from a large scale cross-sectional sero-survey highlighted the existence of two potential clusters of RP infection in central and southern Somalia (Chapter 4). These areas with the neighboring Somali pastoral areas of Kenya and Ethiopia are believed to maintain the last foci of RP in the world.

The risk factors for RP occurrence were identified by logistic regression analysis of the survey data (Chapter 5). These results suggest the existence of RP infected and free areas in the country. Under these circumstances it would be possible to establish zoning using the survey outcome following the OIE guidelines, which allow the declaration of provisional freedom for RP or freedom from RP disease on zonal basis.

According to the OIE standards, the RP disease free zones must be separated from the rest of the country and from neighboring infected countries by a surveillance zone and zoo-sanitary measures which effectively prevent the entry of infection must be in place. However, it is required that the extent of the zones and their limits should be clearly delineated by natural, artificial or legal boundaries that are ***effective in***

preventing the introduction of the disease (OIE, 2005). The husbandry system practiced by the Somali pastoralist is mainly nomadic and herds / flocks are constantly moved in search of water and better pasture. Under this scenario where geographical barriers are difficult to be identified and animal movement restrictions are seldom applicable, the delimitation of the zones' geographical boundaries remains a challenge. The aim of this paper is to propose a zonation approach based on the risk factors identified throughout this study.

6.2. Materials and Methods

Four main risk factors for RP occurrence in the study area were identified through logistic regression analysis. The identified risk factors were: (i) mobility of cattle herd (Hr1), (ii) cattle herd size (HSC1), (iii) cattle population density (MDens1), and (iv) proximity to trade route (MDist2) (Chapter 5). After "cross-validation", the logistic regression model (TABLE 6.1), which includes the four main effect variables and three first order interactions (Chapter 5), was used to predict the spatial risk of RP occurrence in the study area.

TABLE 6.1: Final model (model coefficients).

Variables	*Coef.*	*Std. Err.*	*t*	*P > |t|*	*95% Conf. Interv.*
Const.	-1.89551	0.12595	-15.05	< 0.001	-2.14291; -1.64811
Hr1	0.57370	0.08492	6.76	< 0.001	0.40689; 0.74052
MDens1	-2.77244	0.69628	-3.98	< 0.001	-4.14013; -1.40475
HSC1	-0.19018	0.04757	-4.00	< 0.001	-0.28363; -0.09673
MDist2	-4.374189	1.37618	-3.18	0.002	-7.07739; -1.67098
MDens1_HSC1	0.27470	0.07183	3.82	< 0.001	0.13392; 0.41549
MDist2_Hr1	-0.99906	0.39183	-2.54	0.011	-1.76705; -0.23108
MDens1_MDist2	3.56041	1.57794	2.26	0.024	0.467705; 6.65312

The model (TABLE 6.1) was cross-validated via Bootstrap method (Efron, 1979 & 1982; Efron & Stein, 1981; Efron & Tibshirani, 1993), whereby the model parameters were reassessed via re-sampling of the available data. In order to be able to use the identified risk factors in a GIS environment, a distribution map for each of the four main effect variables was created. The distribution maps for cattle herd mobility (home-range) and cattle herd size were generated using Kriging techniques (Carrat & Valleron, 1992; Davis, 1986; Davis & McCullagh, 1975; Olivier & Webster 1990; Pfeiffer, 1994 & 2000; Pfeiffer *et al.*, 1997; Stein, 1999; Webster *et al.*, 1994).

The home-range and herd size values (z) for each herd included in the study were used to generate the krigged surface utilizing as location the coordinates (x, y) of the centroid of the home-range polygon estimated for each individual herd (Chapter 5). The cattle density map was derived from the available data (Anon., 1989). A neighborhood analysis (moving average) was performed on the data to make the map more realistic (Michel *et al.*, 2002) (Chapter 5). The distance from cattle trade routes was obtained by generating a linear distance map (Chapter 5). When distribution maps for four risk factors were generated the fractional polynomial transformations identified by the logistic regression analysis (Chapter 5) were applied to the maps. Furthermore, three distribution maps for the first order interactions identified as significant in the logistic regression model (Chapter 5) were generated. Finally the estimated *logit* [$g(x)$] map was created using the reassessed logistic regression model and the risk (probability: p) map for RP occurrence was generated by the exponentiation of the estimated *logit* (Lemeshow *et al.*, 1988) as follows:

$$p = \frac{e^{g(x)}}{1 + e^{g(x)}} \qquad [6.1]$$

The risk map was then reclassified to identify low, medium and high risk areas for RP occurrence. The Bootstrap was carried out in STATA 8.0 SE, while the Kriging analysis in S-Plus. The results of the Kriging analysis were then exported in ArcGIS 8.3. The remaining analysis was implemented using the Spatial Analyst Extension of ArcGIS 8.3.

6.3. Results

The reassessed model parameters are given in TABLE 6.2. The model parameters were re-estimated by re-sampling 1,000 times the available dataset. At each cycle 90% of the primary sampling unit was randomly re-sampled and the model was refitted. The best model coefficients were then derived from the distribution of the model coefficients' population obtained from the Bootstrap.

TABLE 6.2: Logistic regression model reassessed via Bootstrap method (1,000 resampling cycles of 90% of the primary sampling units).

Variables	*Coef.*	*Std. Err.*	*95% Conf. Interv.*
Const.	-1.8974	0.1385	-2.1695; -1.6249
Hr1	0.5745	0.0977	0.4052; 0.8004
MDens1	-2.7298	0.7285	-4.2977; -1.4112
HSC1	-0.1933	0.0514	-0.2983; -0.0966
MDist2	-4.5643	1.4864	-7.7059; -1.8841
MDens1_HSC1	0.2680	0.1160	0.0221; 0.4851
MDist2_Hr1	-0.9764	0.3446	-1.6318; -0.2817
MDens1_MDist2	3.3745	1.5562	0.4587; 6.3869

The krigged surfaces for cattle home-range and herd size were created by first fitting a second order polynomial transurface to the data (Lessard *et al.*, 1990). Then, the covariance structure of the data was modeled via a covariance function as follows:

- Home-range's covariance function: *Exponential* ($\theta = 0.24$; $\alpha = 0.35$)
- Herd size covariance function: *Exponential* ($\theta = 0.47$; $\alpha = 0.60$)

Where: θ is "*range*" and α is "*nugget*"

The best covariance function for each dataset was selected using the Akaike's Information Criteria (AIC) (Cressie, 1993), which was utilized to compare the fit of several covariance functions (e.g. Gauss, Exponential, Spherical and Matern). The final surface was generated by kriging the residuals of the transurface using the selected covariance function. The krigged maps for cattle home-range and herd size are given in FIGURE 6.1 & 6.2 respectively.

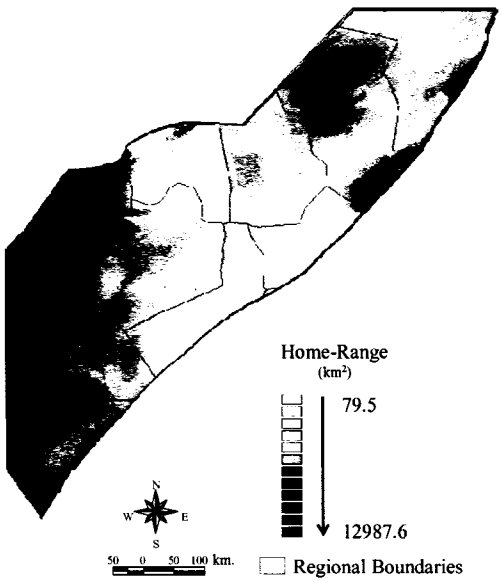

FIGURE 6.1: Cattle home-range distribution map obtained via Kriging method.

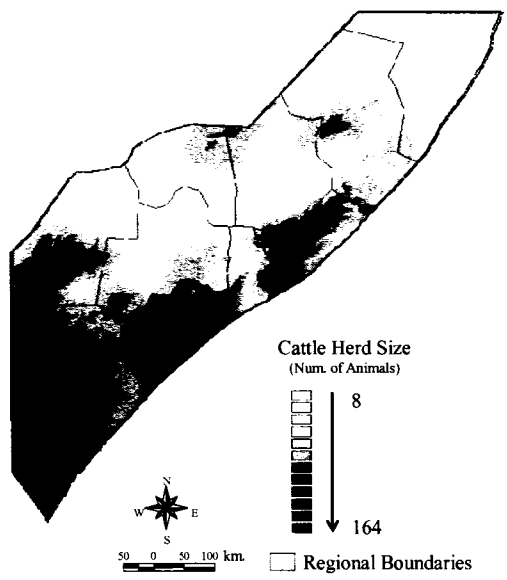

FIGURE 6.2: Cattle herd size distribution map obtained via Kriging method.

A distribution map for each of the variables (e.g. fractional polynomial transformation of the identified risk factors and significant first order interactions) included in the logistic regression model was then created (FIGURE 6.3a to 6.3g). The map for the estimated *logit* (FIGURE 6.3h) was generated using the distribution maps as variables in the logistic regression model reassessed via Bootstrap method (TABLE 6.2).

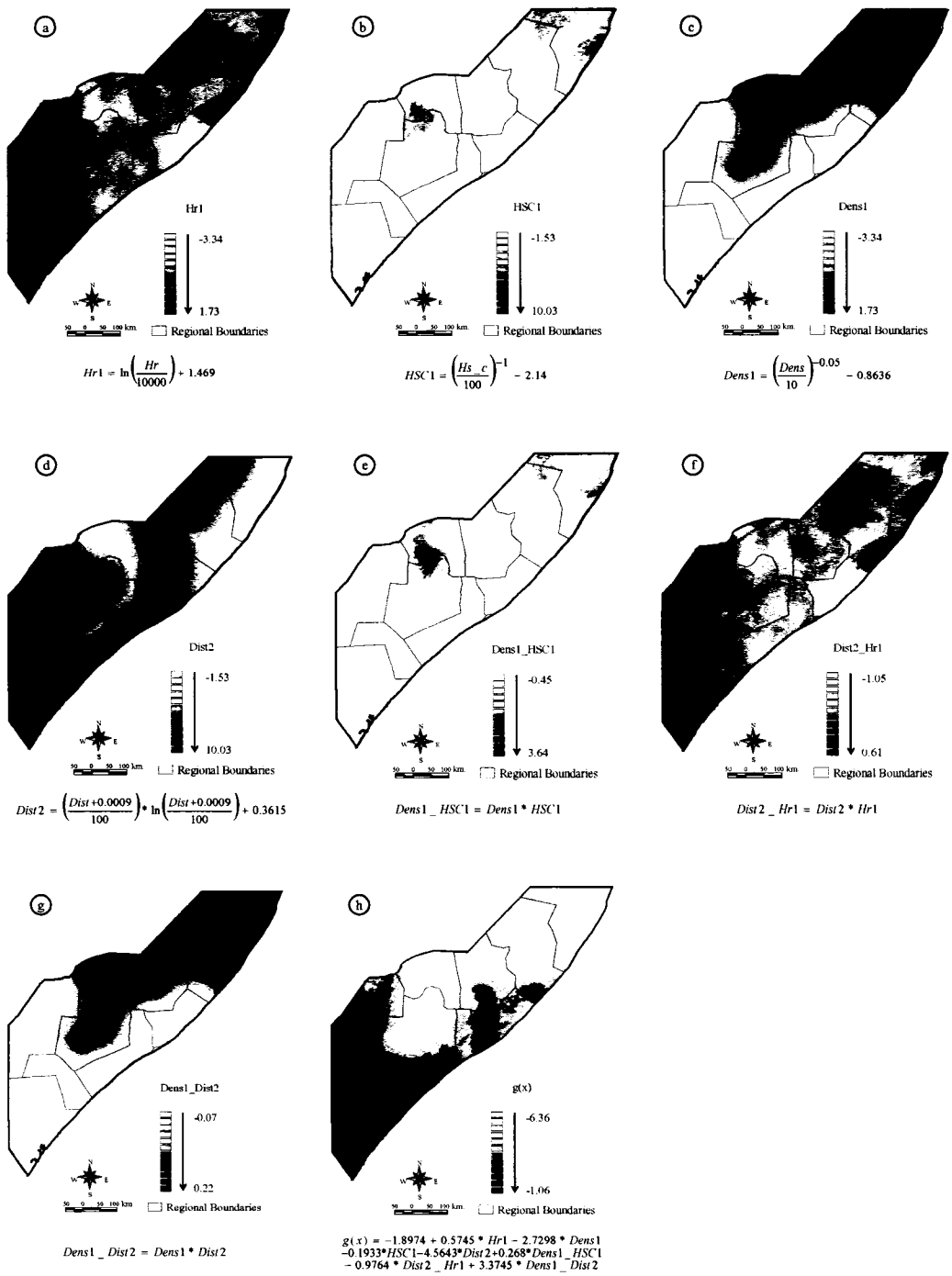

FIGURE 6.3: Distribution maps for (a) Fractional polynomial transformation of cattle home-range (*Hr*): *Hr1*; (b) Fractional polynomial transformation of cattle herd size (*Hs_C*): *HSC1*; (c) Fractional polynomial transformation of cattle density (*Dens*): *Dens1*; (d) Fractional polynomial transformation of distance from cattle trade routes (*Dist*): *Dist2*; (e) Interaction between *Dens1* and *HSC1*: *Dens1_HSC1*; (f) Interaction between *Dist2* and *Hr1*: *Dist2_Hr1*; (g) Interaction between *Dens1* and *Dist2*: *Dens1_Dist2*; and (h) Estimated *logit*: *g(x)*.

The risk (probability: *p(x)*) map (FIGURE 6.4) for RP occurrence was generated by the exponentiation of the estimated *logit* map. The risk map was then reclassified to reflect 3 categories of risk (FIGURE 6.5) as follows:

- Low Risk: $p(x) < 0.005$
- Medium Risk: $0.005 \leq p(x) \leq 0.03$
- High Risk: $p(x) > 0.03$

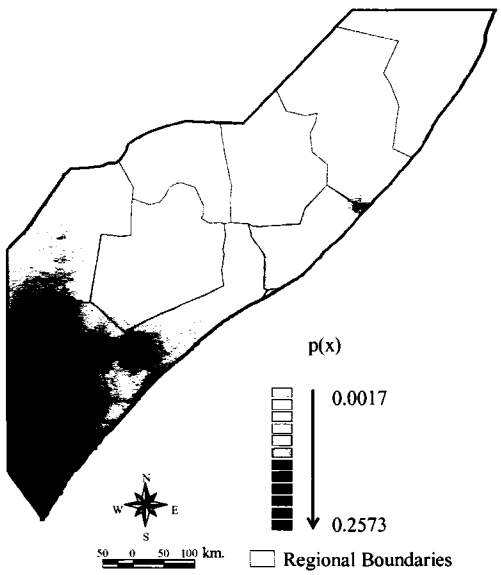

FIGURE 6.4: Risk [*p(x)*] map for RP occurrence.

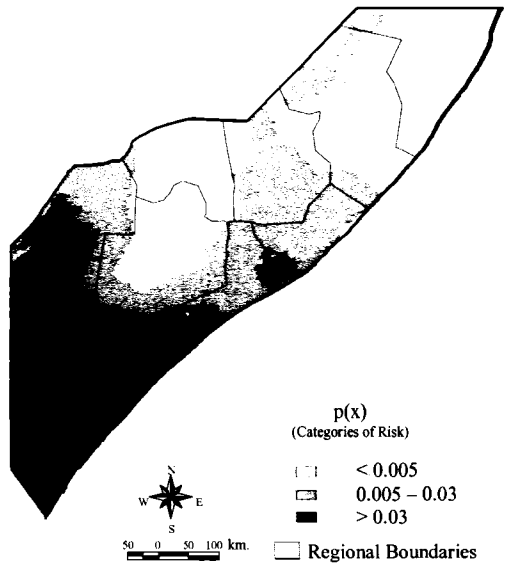

FIGURE 6.5: Reclassified risk [*p(x)*] map for RP occurrence.

6.4. Discussion and Conclusion

The delimitation of infected and free zones remains a challenge in the nomadic systems due to the high mobility of herds and flocks, which are constantly moved in search of water and better pastures. In addition, the restriction of animal movements is not a viable option in nomadic contexts since limiting the access of the nomadic herds to certain areas may hinder their chances of survival.

Therefore, within this context, scientifically sound alternatives for zonation, which cope with the nomadic nature of the husbandry system need to be explored. In this paper a zonation approach based on the identification and quantification of risk factors for RP occurrence has been proposed.

The use of spatialization of the available data and their integration into a GIS, coupled with conventional statistical models, offered the possibility of assessing the spatial risk of RP occurrence in the study areas as a whole. This allowed the identification of areas of high, medium and low risk for RP occurrence and provided a way to delineate "geographical boundaries" that may be utilized for RP zonation. Such zonation approach may be particularly useful in nomadic pastoral systems.

The identified low risk areas may be declared as RP free zones, while medium and high risk zones may be regarded as surveillance and infected zones respectively. The rationale for this is that the areas where the combination of risk factors for RP occurrence does not allow the establishment of infection can be quite safely considered free of RP.

However, intense surveillance is needed in the medium risk areas in order to detect any viral activity moving from the infected to the free zones. Furthermore, strict control and surveillance measures must be established along the existing cattle trade routes. In fact, the proximity of the nomadic herds to cattle trade routes has been identified as an important risk factor for RP occurrence. Furthermore, cattle trade herds may rapidly move the infection across zones and national borders.

The prediction model was reassessed via Bootstrap method. However, further validation (using data from further surveys) of the risk map will increase the

confidence in the accuracy and the "effectiveness" of the geographical boundaries identified for zonation.

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CONCLUSIONS

This study represents the first structured large scale cross-sectional sero-survey carried out in Somalia over the past two decades. The use of random map coordinates that has been adopted for the survey do not require any inventory of animal populations, since the sampling frame is represented by all possible coordinates that can be generated inside a specific area of interest, thereby overcoming one of the najor problems that random sampling often poses. Moreover, the success rate ndicates that the methodology is applicable and that it can be successfully mplemented by local professionals if properly trained.

Given what is alluded to above, the proposed investigation approach represents a key ool to generate reliable information from nomadic pastoral areas, which may enable lecision makers to better address priority diseases and potentially, in the long term, to access more lucrative markets in developed countries by complying with internationally acceptable surveillance standards. This will in turn foster fair trade and contribute to poverty alleviation.

Furthermore, the integration of conventional statistical methods, spatial analysis and GIS has proven to be particularly useful in capturing the complexity of the interactions that characterize the livestock population of the mobile livestock keeping systems of Somalia.

In fact, the spatialization of the available data and the use of conventional statistical methods and spatial analysis combined with the utilization of GIS have allowed: (i)

the identification of potential foci of RP maintenance in the study areas; (ii) the identification and quantification of RP risk factors; and (iii) the prediction of the spatial risk for RP occurrence. The latter may assist in the zonation of the country according to the OIE guidelines for RP.

In conclusion, the utilized methodology has proven to be able to produce reliable information from mobile livestock keeping systems. Furthermore, the study has generated important baseline data that will provide better directions towards the final RP eradication effort in the Somali Ecosystem.

In this regard, the final eradication of RP needs good cooperation and harmonization of activities between the countries that are considered to maintain the last foci of RP in the world (e.g. Ethiopia, Kenya and Somalia). In order to guarantee such harmonization of interventions, the Somali Ecosystem Rinderpest Eradication and Coordination Unit (SERECU) has recently been established within the AU/IBAR headquarter in Nairobi (Anon., 2005a). Currently, the proposed methodology has been recommended by SERECU to simultaneously investigate the status of RP in the three countries in order to generate relevant information for the entire Somali Eco-System (Anon., 2005b). The data obtained from the study will lead to the formulation of a synergistic RP eradication strategy that will aim at eradicating the remaining foci of the disease from the Horn of Africa and therefore from the world.

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