Available online at www.sciencedirect.com
SCIENCE DIRECT
ELSEVIER
Preventive Veterinary Medicine 69 (2005) 245–263
PREVENTIVE VETERINARY MEDICINE
www.elsevier.com/locate/prevetmed

A model of lineage-1 and lineage-2 rinderpest virus transmission in pastoral areas of East Africa

J.C. Mariner [a,*], J. McDermott [b], J.A.P. Heesterbeek [c], A. Catley [d], P. Roeder [e]

[a] *RDP Livestock Services, P.O. Box 523, 3700 AM Zeist, The Netherlands*
[b] *ILRI, P.O. Box 30709, Nairobi, Kenya*
[c] *Faculty of Veterinary Medicine, Department of Farm Animal Health, University of Utrecht, Yalelaan 7, 3584 CL Utrecht, The Netherlands*
[d] *Community-Based Animal Health and Participatory Epidemiology (CAPE) Unit, Pan African Programme for the Control of Epizootics, African Union/Interafrican Bureau for Animal Resources, P.O. Box 30786, Nairobi, Kenya*
[e] *Animal Health Service, FAO, Viale delle Terme di Caracalla, 00100 Rome, Italy*

Received 12 May 2003; received in revised form 6 September 2004; accepted 8 February 2005

Abstract

The development of a stochastic, state-transition model of rinderpest transmission dynamics is described using parameter estimates obtained from both laboratory and participatory research. Using serological data, the basic reproduction numbers for lineage-1 rinderpest virus in southern Sudan and for lineage-2 rinderpest virus in Somali livestock were estimated as 4.4 and between 1.2 and 1.9, respectively. The model predictions for the inter-epidemic period in Sudan and Somalia (1.2 and 4.2 years, respectively) were in agreement with analysis of livestock-owner reports (1–2 years and 5 years, respectively).

Keywords: Rinderpest; State-transition model; Modelling; Basic reproduction number; Participatory epidemiology; Policy-making

* Corresponding author. Present address: Tufts University School of Veterinary Medicine, 200 Westboro Road, N. Grafton, MA 01536, USA. Tel.: +1 508 839 5302 (main)/+1 508 887 4742 (direct); fax: +1 508 839 7946.
E-mail address: jeffreymariner@yahoo.com (J.C. Mariner).

0167-5877/$ – see front matter © 2005 Published by Elsevier B.V.
doi:10.1016/j.prevetmed.2005.02.001

1. Introduction

Rinderpest is currently the subject of a major international eradication effort: the Global Rinderpest Eradication Programme (GREP). The GREP programme seeks the verifiable eradication of rinderpest by 2010 by emphasising targeted vaccination and surveillance. Using this approach, considerable progress has been made in eradicating rinderpest from Asia and most of Africa. However, three important regions of concern remain, each associated with a specific ''lineage'' of rinderpest virus (Barrett et al., 1998). Lineage-1, previously widespread in Africa, was confined to the southern Sudan in recent years and has generally been considered to be of moderate virulence. Lineage-2 was diagnosed in a series of wildlife outbreaks in southern Kenya in 1994 and apparently is maintained today as predominantly mild disease in the Somali pastoral areas of East Africa. Lineage-3 is an Asian lineage which until recently was present in the lowland areas of Pakistan. Pakistan is now in the process of verifying rinderpest eradication and there are no other known active foci of lineage-3 circulation. We consider only the southern Sudan (lineage-1) and Somalia (lineage-2) foci.

A number of features of rinderpest make it relatively easy to model. Rinderpest is a contagious disease of cattle, buffaloes and wildlife, caused by an RNA virus of the genus *morbillivirus* and transmitted by direct contact. There is no known carrier state and the virus is inactivated rapidly in the environment (Plowright, 1986). Infection either results in death or lifelong immunity. A thermostable vaccine (Mariner et al., 1990) is in wide-spread use and this vaccine generates lifelong immunity against all known strains (Plowright, 1984).

Eradication programs require timely decision-making based upon the most likely interpretation of all available information, often called best-bet scenarios (Waters-Bayer and Bayer, 1994). Taylor et al. (2002) have documented the inadequacy of quantitative serological and clinical surveillance techniques in the control of lineage-2 rinderpest in Tanzania. Conventional data on the extent and frequency of rinderpest outbreaks and the demographics and contact patterns of host populations are difficult to obtain in the isolated and insecure areas were rinderpest is remaining. Mariner and co-workers (Mariner and Paskin, 2000; Mariner and Roeder, 2003) have described methods for conducting timely participatory disease searches to document the current and historic incidence of rinderpest to serve as an intelligence base for decision-making. The Nilotic pastoral communities of southern Sudan (Schwabe and Makuet Koujok, 1981) (Catley et al., 2001) and the pastoral communities of Somali ecosystem (Catley and Mohammed, 1996; Catley et al., 2002) in the Horn of Africa are livestock specialists and excellent observers of disease incidence. Appropriate combinations of participatory disease intelligence and quantitative data form the most complete information base for strategic decision making in pastoral systems.

In this paper, a simple rinderpest model (assuming homogeneous contact experience in a large population) that was used in 2001 to study the endemic transmission of lineage-1 rinderpest virus in southern Sudan ecosystem and the mild rinderpest (suspected to be due to a lineage-2 virus) occurring in the greater Somali areas of Kenya, Ethiopia and Somalia is described. The model was based on the best-bet scenarios derived from both the participatory and quantitative data available at that time. The basic reproduction number (R_0) was estimated using serological data from the affected areas to assure that

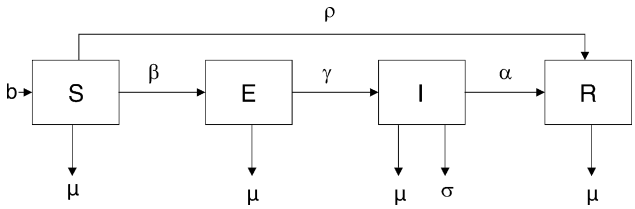

Fig. 1. Diagram of the rinderpest SEIR model structure. Non-specific mortality (μ) occurs in all four states. Births (b) all enter the susceptible (S) state. Only infectious (I) animals experience rinderpest mortality (σ). The rate at which animals move from the susceptible to exposed state (E) is governed by the effective contact rate (β). The rate at which exposed animals become infectious and infectious become resistant (R) is described by γ and α, respectively. Immunization is modelled as a transition from the susceptible state directly to the resistant state at the annual immunization rate (ρ).

transmission parameters reflect the viral strains and contact structure of the populations in question. Disease parameters collected from pastoralists in each of the two areas were used as model inputs and to qualitatively validate model predictions. The utility of this participatory modelling approach, its implications for rinderpest eradication strategies and the impact of resulting policy changes in these two remaining African foci are discussed.

2. Materials and methods

2.1. Stochastic model structure

The model is a stochastic state-transition model developed in @Risk[1] software using a mass-action formulation as described by Jacquez (1996). The model contains four principal states: susceptible, exposed, infectious and resistant (SEIR). The structure of the model is presented in Fig. 1. The modelling terms are defined in Table 1.

Input parameters were incorporated as pert probability distributions rather than discrete values. Pert distributions can be visualized as truncated normal distributions that lack long tails of uncharacteristic extreme values. These distributions were designed to reflect both biological variation and the uncertainty in parameter estimates. The shape of the distribution was determined by entering minimum, maximum and most-likely values. During the sensitivity analysis, initial conditions for the exposed, infectious and recovered states were entered as fixed numbers. Population size was always entered as a fixed number. Later, in the model analysis of endemic disease, a pert distribution was utilized for the initial prevalence of exposure and immunity. The prevalences selected from the distributions in each iteration of the model were multiplied by the population size to give the number exposed and resistant. The initial number of susceptibles was calculated from the values specified for the other states and the population size.

Latin-hypercube sampling was used to select one value from each parameter distribution in each iteration of the model. (In Latin hypercube sampling, the probability distribution was divided into strata of equal probability and only one value was sampled

[1] Palisade Corporation, 31 Decker Road, Newfield, NY 14867, USA.

Table 1
Definition of modelling parameters for rinderpest in East Africa

Symbol	Definition	Information source
α	Rate of recovery (days^{-1})	Leiss and Plowright (1964), Plowright (1965), Wamwayi (personal communication)
β	Effective contact rate ($C \times p$) (days^{-1})	Majok et al. (1991), Terra Nuova (personal communication)
C	The physical contact rate (days^{-1})	Not estimated, contained in β
γ	Transition rate from exposed to infectious state (days^{-1})	Leiss and Plowright (1964), Plowright (1965)
μ	Non-specific mortality rate (days^{-1})	Majok et al. (1991), and data presented in this paper
ρ	Annual rate of immunization (successful vaccination) (days^{-1})	User defined
p	The probability that a physical contact results in infection	Not estimated, contained in β
σ	Rinderpest specific mortality rate (days^{-1})	Mariner and Roeder (2003), and data presented in this paper
ΔT	Time step (days)	User defined

randomly from within each stratum. The stratum selected for sampling in an iteration of the simulation was chosen randomly and sampled without replacement.)

The model was an open-population model where new susceptibles were born into the susceptible (S) state at each time step at rate b. Non-specific mortality occurred at the rate μ and resulted in losses from all four states. The non-specific mortality rate was set equal to the estimated birth rate. Rinderpest-specific mortality occurred at the rate σ from the infectious (I) state. The mass-action term incorporated the population size (N) at each time step as the sum of the four states (S + E + I + R).

The difference equations were:

$$\Delta \mathrm{S} = -C\,p\,\frac{\mathrm{SI}}{N}(\Delta T) + bN(\Delta T) - \mu \mathrm{S}(\Delta T) \tag{1}$$

$$\Delta \mathrm{E} = +C\,p\,\frac{\mathrm{SI}}{N}(\Delta T) - \gamma \mathrm{E}(\Delta T) - \mu \mathrm{E}(\Delta T) \tag{2}$$

$$\Delta \mathrm{I} = +\gamma \mathrm{E}(\Delta T) - \alpha \mathrm{I}(\Delta T) - \mu \mathrm{I}(\Delta T) - \sigma \mathrm{I}(\Delta T) \tag{3}$$

$$\Delta \mathrm{R} = +\alpha \mathrm{I}(\Delta T) - \mu \mathrm{R}(\Delta T) \tag{4}$$

The number of transitions from the susceptible to exposed state per time step was calculated using

$$\frac{\beta \times \mathrm{SI}}{N} \tag{5}$$

where the effective contact rate (β) was equal to the product of the contact rate (C) and the probability of transmission per contact (p). Apart from the probability distributions for

drawing the values of the major parameters, the model incorporated two stochastic elements. Because only whole animals can transit between states, a stochastic form of rounding was used where the fractional portion of all calculated transitions was compared against a random number to determine if the calculated number of transitions was rounded up or down. The second stochastic element was used to determine the number of transitions from the susceptible (S) to the exposed (E) class when the predicted number of transitions was small. Here, when the number of transitions calculated by Eq. (5) was ten animals or less the result was compared against a random number and either ten transitions or no transitions took place during the time step.

In simulations with this model, we added immunization to the system above. Immunization is handled as a transition directly from the susceptible to the resistant state. The transition utilized a sine function to mimic the seasonality of immunization due to the impact of the rainy seasons and the resulting pulsed pattern of vaccination. The annual rate of immunization can be entered for each year, thus allowing different strategies to be modelled.

The values estimated for model input parameters are summarized in Table 2. These values were used in all simulations unless otherwise noted. Latent and infectious periods as measured in laboratory experiments for lineage-1 virus and derived from laboratory-based expert opinion for lineage-2 virus (Leiss and Plowright, 1964; Robson et al., 1959; Plowright, 1965) were used to estimate γ and α, respectively. Information on the mortality associated with lineage-2 rinderpest in Somalia was based on livestock owner information collected as part of a participatory investigation in Somalia (Mariner and Roeder, 2003).

In Sudan, population structure, life-expectancy and rinderpest case mortality were estimated from proportional piling exercises (Mariner and Paskin, 2000; Catley et al., 2001; Catley et al., 2002). Briefly, 15 different groups of respondents who had identified rinderpest as a problem in response to an open-ended question were asked to identify the cattle age classes they recognized and the age limits for each category. A row of circles was drawn on the ground with one circle corresponding to the age classes identified. Drawings on index cards were placed next to each circle that represented the age categories. Respondents were then given one hundred counters (beans in this case) and asked to divide them into piles representing the relative number of animals in each age category in their herd. Thereafter, a second row of circles was drawn on the ground beneath the first row and the respondents were asked to move counters from each age class pile to second row to represent the relative number of animals by age-class that died in a major rinderpest outbreak. Once this was completed, the counters were returned to their original age-class

Table 2
Model parameter estimates for rinderpest in East Africa

Parameter (days^{-1})	Lineage-1			Lineage-2		
	Min	Mode	Max	Min	Mode	Max
β	1.1	1.3	1.4	0.15	0.17	0.19
γ	0.14	0.20	0.22	0.13	0.15	0.18
α	0.14	0.17	0.20	0.083	0.100	0.125
σ	0.08	0.11	0.14	0.005	0.010	0.015
b and μ (10^{-3})	0.49	0.55	0.62	0.49	0.55	0.62

piles and the respondents were asked to move counters to the second row to represent the relative number of animals that died in a minor rinderpest outbreak. After each step in the process, the scores were tabulated, data was recorded and respondents were asked to clarify the reason for their choices in probing questions.

Mortality proportions were greatest in younger age groups and the overall mortality proportion was 57.3% for severe outbreaks and 32.3% for milder outbreaks (95% C.I. 46.5–68.1 and 24.8–39.7%, respectively). In the Sudan scenario, the pert distribution for the daily rinderpest-mortality rate was set to reproduce case-mortality proportions in this range. In the Somali scenario, a case mortality proportion of 10% was used as the target based on the results of herder interviews (Mariner and Roeder, 2003).

The birth and non-specific-mortality rates were estimated by a combination of two methods. First, the average age of the cattle (5.7 years) sampled by Majok et al. (1991) in their random sample of Bahr el Ghazal Province was used as to estimate a birth rate of 17.5% per year. Secondly, a weighted average of the age-class data provided by herders as part of our study was calculated as an additional estimate of life expectancy using the average ages of 0.5, 1.5, 3.0 and 9.0 years for the four age classes identified by the herders. The average life expectancy calculated from the data provided by herders was 4.9 years (birth rate of 20.4%). The estimates of birth rate from the serological data (17.5%) and the proportional piling exercises (20.4%) are in agreement. Noting that herders reported that 17.2% of their herds were less than 1 year old and that considerable mortality must occur in the first month of life, a composite estimate of 20% was used as the annual birth rate in the model for both the Sudan and Somali scenarios.

The basic reproduction number R_0 is the number of secondary infections resulting from one primary case in a totally susceptible population (Anderson and May, 1991; Diekmann and Heesterbeek, 2000). The basic reproduction number is a feature of both the infectious agent and the host population in which transmission is occurring, but without a control measure being active. The contact rate, C, and the probability that a contact results in transmission, p, are in practice very difficult to measure. The product of C and p is the effective contact rate (β) and simple techniques exist to estimate this composite rate. The formula we used for R_0 for the model described in Eqs. (1)–(4) was

$$R_0 = \beta \frac{\gamma}{(\gamma + \mu)(\mu + \alpha + \sigma)} \tag{6}$$

where $\gamma/(\gamma + \mu)$ was the probability that a newly infected individual survived the latent period and where $1/(\mu + \alpha + \sigma)$ was the average duration of the infectious period, during which β new cases are made per unit of time. The first two ingredients can be estimated separately and R_0 can be estimated from serological data as described below in Section 2.2. It is important to stress that the values of R_0 estimated from serological data are not a direct input in the stochastic model, they are used to estimate the distribution of values used for β, which are directly incorporated in the model.

Quantitative outputs from the model are also probability distributions and include R_0, the duration of viral persistence, annual average of daily point prevalence, daily incidence proportion, case mortality proportion, immunity proportion and annual numbers of cases, morbidity, mortality and vaccination. The distribution of R_0 output by the model is calculated from α, β, γ, μ and σ as described by Eq. (6). This output distribution is

compared against the range of R_0 values estimated from the serological data and as an internal validity check.

The model produces graphic data including epidemic curves that provide information on the periodicity of epidemics as well as their shape and pattern.

2.2. Deterministic estimation of R_0 and the herd immunity threshold

The basic reproduction number is an estimate of the transmissibility of an agent in a totally susceptible population. When immune animals are present and contact is homogeneous, transmissibility is described by the effective reproduction number (R_e). If the proportion susceptible (non-immune) is S/N then:

$$R_e = (S/N)R_0. \tag{7}$$

In the populations where the force of infection ($\beta S/N$) is constant and vaccination coverage is low or non-existent, the value of R_0 can be estimated deterministically from the proportion of the population that is susceptible to infection (Anderson, 1992). Each case of infection is essentially just replacing itself and the effective reproduction number (R_e) is equal to 1. In this special case:

$$R_0 = \frac{N}{S}. \tag{8}$$

At prevalence of immunity levels above this threshold, R_e will be <1 and on average, infectious cases will not replace themselves. If the prevalence of immunity is maintained above this level by vaccination for a sufficient period of time related to the transmission cycle of the disease, the disease will die out. Thus, the antibody prevalence in stable endemic settings is also an indicator of the threshold value required for the interruption of transmission.

The deterministic critical herd immunity threshold (h) required to interrupt disease transmission can be back calculated from R_0 using the simple relationship (Anderson and May, 1991)

$$h = 1 - \frac{1}{R_0}. \tag{9}$$

To estimate R_0 for lineage-1 rinderpest virus in southern Sudan, the results of a randomised survey of 4074 sera from Bahr el Ghazal Province (Majok et al., 1991) provided the principal estimate of the proportion susceptible in a representative pastoral population. Vaccination coverage was $<15\%$. Two small, unpublished data sets derived from unvaccinated populations in Ayod, Jonglei Province, (100 sera) and Naita, Eastern Equatoria Province, (80 sera) provided by Operation Lifeline Sudan Livestock Programme were analyzed separately, but not used as model input. All Sudan samples were assayed using serum neutralization test (SNT).

For lineage-2 rinderpest virus in Somali livestock, R_0 was estimated using the results of a survey of 1229 serum from unvaccinated cattle in Lower Juba (Terra Nuova, personal communication) tested by the rinderpest competitive enzyme linked immunosorbent assay (C-ELISA) (Anderson et al., 1991). The range of R_0 values for lineage-2 was determined

using three different C-ELISA percent inhibition (PI) cut-off levels, 35, 40 and 50% to assess the impact of variation in test sensitivity on the range of estimates of R_0.

2.3. Experimentation with the stochastic model

A sensitivity analysis was completed on the initial conditions for the E, I and R states and the total population size. Model simulations consisted of 100 iterations and were run over a period of 2 years. Sensitivity analysis was conducted separately for both lineage-1 and lineage-2 parameter values.

The sensitivity analysis for the E and I states was completed with fixed equal numbers of exposed and infectious (1, 2 and 5 to 40 in steps of 5) and the number recovered set to zero in a total population 10,000. In the analysis for the R state, the percentage recovered was increased in increments of 5% from 0 to 100%. For this analysis, the initial numbers exposed and infectious were both set to 10 in order to reduce the number of minor outbreaks, which were not of interest. (A minor outbreak is defined as an introduction of infection that dies out before generalization of infection can take place due to stochastic effects. A major outbreak is generalized infection usually in the form of a classic epidemic.)

The sensitivity analysis on the initial conditions for population size was completed in increments of 10,000 to determine the critical community size (CCS). The initial condition for the recovered state was set to 75 and 30% in the lineage-1 and lineage-2 models, respectively. These values had resulted in the longest outbreaks in the sensitivity analysis for the initial conditions in the R state. For this analysis, the initial conditions for both exposed and infectious were set to 0.1% of the total population to maintain the same ratio of exposed and infectious to the total population as in the sensitivity analysis for the R state.

The effect of the initial prevalence of immunity on the persistence of an introduction of lineage-1 infection was investigated in more detail using a uniform distribution for the initial number resistant that ranged from 0 to 200,000. The numbers initially exposed and infectious were set directly to ten to mimic the introduction of an infected herd.

In the second stage of the analysis, initial conditions were selected that produced endemic patterns of infection in the sensitivity analysis. A population size of 200,000 (above the CCS) was used in these and all other simulations unless otherwise noted. The simulations were run over a 12-year time period in order to assure that the epidemic curves were consistent and sustained. The inter-epidemic period was determined by counting the elapsed time in days between the first and last epidemic peak and dividing by the number of inter-epidemic troughs in each of the 100 iterations of the model. The initial prevalence of exposure and immunity were set as pert distributions based on the range of conditions observed at the end of the simulations in the sensitivity analysis that most closely approximated endemic infection. The minimum, most-likely and maximum prevalence of infectious conditions for lineage-1 were 0.00035, 0.00085 and 0.0014, respectively. In the case of lineage-2, prevalence of infectious was set to minimum, most-likely and maximum of 0.008, 0.0011 and 0.0014. The minimum, most-likely and maximum prevalence of immunity initial conditions for lineage-1 were 0.70, 0.75 and 0.80, and for lineage-2 0.25, 0.30 and 0.35, respectively.

For lineage-2, the length of outbreaks was re-examined in small communities of 10,000 and 20,000 head using the initial conditions described in the last paragraph with the objective of studying the risk of persistence of the disease in isolated areas in more detail.

The impact of increasing levels of seasonal mass immunization on the inter-epidemic period where lineage-1 rinderpest is endemic was assessed by increasing annual immunization coverage from 0 to 60% in 10% increments and running the model over a sufficiently long time period for endemic patterns to stabilize (12 years). In the initial simulations, the input parameters were set (locked in @Risk) to the most-likely values stated in Table 2 to facilitate direct comparison of the five vaccination scenarios. Thereafter, sampling of the full parameter distributions stated in Table 2 was allowed leading to variation in the length of the inter-epidemic period and the ability to study more complex interactions between vaccination and the natural periodicity of the epidemic curve.

2.4. Comparing model outputs to field data

In southern Sudan and Somalia, participatory epidemiologic techniques were used to collect data on the incidence of rinderpest outbreaks and the inter-epidemic period between major waves of disease. Data on outbreaks was collected using semi-structured interviews where pastoralists were asked to identify and describe major disease problems using open-ended questions. Rinderpest was never introduced as a topic by the interviewer. If the participants identified and could give a clear description of rinderpest, they were asked the dates, locations and descriptions of disease outbreaks. Only detailed and clear reports that were internally consistent were used as data. Ultimately, the information trail in the Somali ecosystem led to active outbreaks that were confirmed by the agar gel immuno-diffusion test and reported to the OIE. The inter-epidemic period was determined from tabulated reports using time series analysis. A detailed description of the methodology and data validation process has been published by Mariner and Paskin (2000) and is the method of active disease surveillance recommended by GREP (Mariner et al., 2003a, 2003b). The methodology and results of the Somalia study have been reported by Mariner and Roeder (2003).

The patterns of endemic disease described by the livestock owners and the estimates of inter-epidemic period from these studies were used to qualitatively validate the model. In the case of lineage-2, the estimates of R_0 derived in this paper were compared against subsequent independent sero-surveys conducted in the Somalia ecosystem. This was not possible in the Sudan as the disease is apparently no longer circulating (Office International des Epizooties, 2003).

3. Results

3.1. Deterministic estimates of R_0

The estimate of R_0 was 4.4 for lineage-1 (Table 3) rinderpest virus and between 1.2 and 1.9 for lineage-2 (depending on the threshold value chosen for the C-ELISA).

Table 3
Values of R_0 for rinderpest calculated from serological data for Sudan (lineage-1) and Somalia (lineage-2)

Location	ELISA cut-off (%)	Year of survey	Sample size	Positive (%)	R_0	h[a] (%)	Source
Sudan							
Bahr el Ghazal	–	1980	4074	77.4	4.4	77	Majok et al. (1991)
Ayod	–	1991	100	71.0	3.5	71	UN-OLS[b]
Naita	–	1997	80	81.2	5.3	81	UN-OLS
Somalia							
Lower Juba	50	1998	1231	10.0	1.2	14[c]	Terra Nuova
	40		1231	29.8	1.4	30	
	35		1231	46.3	1.9	46	

[a] Deterministic herd immunity threshold.
[b] United Nations – Operation Lifeline Sudan.
[c] Based on the predicted prevalence calculated from the test prevalence using a sensitivity estimate of 70%.

3.2. Stochastic model output

The lineage-1 sensitivity analysis on the initial conditions for the exposed and infectious states found that when ≤ 2 exposed and ≤ 2 infectious animals were introduced both major and minor outbreaks resulted. When initial conditions of 5 exposed and 5 infectious animals were used, only major outbreaks resulted. Regardless of the initial number exposed and infectious, major outbreaks were uniform in shape, height and duration. When E and I were both set to 10, means for the peak numbers of infectious cases and duration of the outbreak were 1926 cases and 84 days, respectively. The range of outbreak durations was 71–100 days.

In the lineage-1 sensitivity analysis for the recovered state, increasing the percentage immune reduced the peak number infectious in major outbreaks but increased the duration of the outbreaks. At 40% initial immunity, occasional minor outbreaks again began to be observed. At 50% initially immune, the mean of the peak number infectious in major outbreaks was 470 cases and they had lost their classic peaked appearance. The duration of major outbreaks was from 100 to 192 days. A 75% initial prevalence of immunity resulted in the longest major outbreaks observed (mean and maximum 215 and 396 days, respectively). Only 45% of the iterations at this level of initial of immunity resulted in major outbreaks and the maximum peak number of infectious cases was 113. At 90% initial prevalence of immunity, most introductions failed and only occasional major outbreaks occurred. In the major outbreaks that did occur, the maximum peak number of infectious cases and duration was 24 cases and 95 days, respectively.

In the lineage-1 sensitivity analysis for total population size, increasing the population size resulted in progressively longer outbreaks. At a population size of 30,000, infection was first observed to persist beyond the two-year termination of the model (7% of iterations). The shape of the epidemic curve was complex, suggesting important stochastic effects. At a population size of 50,000, the epidemic curve assumed an oscillating wave shape with two epidemic peaks over the 2-year duration of the iterations. Infection persisted >2 years in 26% of the iterations. Stochastic extinction of virus transmission (fade-outs) were still observed in population sizes of 200,000 or more, but occurred in

<5% of the iterations indicating that the CCS for lineage-1 is on the order of 200,000 head of cattle.

In the case of lineage-2, the sensitivity analysis on the initial number exposed and infectious found that even up to 20 infectious and 20 exposed animals introduced into a fully susceptible population still lead to both minor and major outbreaks. Major outbreaks peaked at 450 infectious cases and lasted up to 400 days, much longer than with lineage-1.

Increasing the prevalence of immune animals in the lineage-2 model in increments of 5% reduced the height of epidemic peaks and increased the percentage of minor outbreaks. The maximum duration of outbreaks increased, but there was no trend towards an increase in the mean duration of major outbreaks. When the initial prevalence of immunes was 25%, infection persisted beyond the two-year termination of the simulation in 6% of the iterations (in a population of 10,000 head). At this level of initial immunity, the epidemic curves were highly stochastic and it was no longer possible to distinguish between major and minor outbreaks. When initial immunity was set to 40%, infection persisted beyond 2 years in one of the iterations. In the simulation with a 45% initial prevalence of immunes, only two outbreaks lasted beyond 300 days and the longest was 550 days.

The lineage-2 sensitivity analysis on the initial conditions for population size indicated that the disease was able to persist beyond 2-years in populations as small as 20,000 head of cattle (15% of the iterations). As the population size was increased, curves became less stochastic and the percentage of populations infected beyond the two termination of the model continued to increase. At population size of 200,000, the disease faded-out in 6% of the iterations, a finding similar to that of lineage-1.

The output from the stochastic model are presented in Table 4. The table includes the mean and range of the predicted case mortality proportion, R_0 and the inter-epidemic period as well as final-year average prevalences of immunity and infection. The mean inter-epidemic periods were estimated as 1.2 and 4.2 years for lineage-1 and lineage-2, respectively.

In the simulations with lineage-2 virus in a population of 10,000 head, infection persisted beyond 1 year in 26% of the iterations. With a total population of 20,000 head, outbreaks persisted for >2 years in 18% of iterations.

The effects of initial prevalence of immunity on viral persistence following an introduction of lineage-1 virus under the conditions of southern Sudan are illustrated in Fig. 2. At prevalence of immunity levels below 70%, the effect of increasing prevalence of immunity was to extend the duration of the epidemic but reduce the height of the epidemic curve and the overall mortality. At approximately 70% initial prevalence of immunity, a

Table 4
Model outputs for rinderpest in East Africa

Parameter	Lineage-1			Lineage-2		
	Min	Mean	Max	Min	Mean	Max
Prevalence of immunity (%)	75	78	81	23	33	41
Prevalence of infectious (%)	0.14	0.17	0.19	0.08	0.16	0.34
R_0	4.0	4.6	5.2	1.3	1.5	1.8
Case mortality proportion	0.34	0.40	0.45	0.06	0.09	0.13
Inter-epidemic period (years)	1.1	1.2	1.4	4.0	4.2	4.6

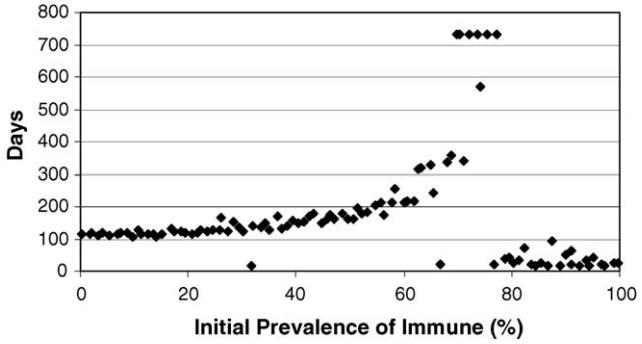

Fig. 2. The effect of initial prevalence of immunity on the length of outbreaks. The model was run over 2 years after the introduction of 10 exposed and 10 infectious animals. The initial prevalence of immune animals was varied between 0 and 200,000 by sampling a uniform distribution. The length of the outbreak is plotted against the percent initially immune. Note that at low prevalences of immunity outbreaks last about 150 days. As the initial prevalence of immunity was increased to about 50%, the length of the outbreak increased to the range of 200–400 days. Between about 70 and 77% immunity, the introduction of infected animals resulted in persistent infection that was still on going at the end of 730 days in 6 iterations. When prevalence of immunity surpassed 78%, mainly minor outbreaks occurred.

threshold was reached where infection was still present at 730 days and the shape of the epidemic curves was consistent with oscillating waves of disease. Above 77% initial prevalence of immunity, although secondary cases could occur (minor outbreaks), no successful introductions took place.

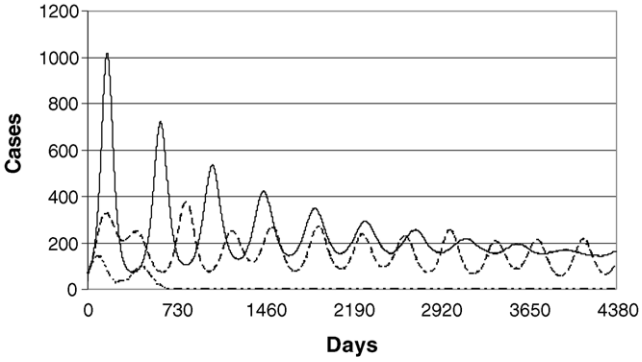

Fig. 3. Effect of immunization rate on the epidemic curve. In this graph, the effect of 0 (solid line), 30 (dashed line) and 60% (dashed and dotted line) successful immunization rates on the epidemic curve setting are presented using the expected values for the input distributions in the Sudanese setting for input parameters. Note that the onset of moderate levels of immunization tends to suppress or delay the first epidemic peak. By the fourth round of vaccination, the inter-epidemic period is forced into a regular annual cycle by the vaccination campaign. In the example of 60% immunization, the disease had fade-out by the end of year 2. Note that this is a stochastic effect and that in iterations with 60% immunization disease persisted from 1 to 7 years before fading-out. If the parameter input distributions are sampled, more complex patterns including bimodal peaks with a 3-year inter-epidemic pattern are possible.

The effect of sub-optimal vaccination strategies on the inter-epidemic period in simulations using the expected values of the parameter distributions for the Sudanese situation is presented in Fig. 3. Low to moderate levels of vaccination suppress the first epidemic peak and eventually force an annual cycle of epidemics. This effect was evident with annual vaccination coverage as low as 10%. As coverage was increased above 30%, fade-outs began to occur in the second year. Prevalence of infection was suppressed by as much as 90% in the sub-optimal vaccination scenarios relative to the no vaccination scenario. When the model was allowed to sample the input parameter distributions, more complex patterns resulted including bimodal peaks with apparent inter-epidemic periods of 3 years.

The final seroprevalence and R_0 estimates output by the model are internally consistent with the data input to the model for the endemic areas of Sudan and Somalia. A subsequent randomized rinderpest sero-survey (2465 sera collected from 135 sites) in the Lower and Middle Juba Regions of Somalia completed in 2002 found a test prevalence of 16.1% using the C-ELISA with a 50% cut-off point (Tempia et al., 2003). Cluster prevalences did not exceed 60%. The test prevalence is equivalent to a predicted prevalence of 23.0% and an R_0 estimate of 1.3.

4. Discussion

The sensitivity analysis demonstrated characteristic behaviour for stochastic models of infectious disease. When the number of infected animals introduced was small both major and minor outbreaks occurred. As the infectious dose increased, only major outbreaks resulted. As the initial level of immunity was increased the probability of minor outbreaks increased and the stochastic behaviour of the epidemic curve increased (due to the smaller population of susceptibles present). As the total population size was increased the probability of stochastic fade-out declined until a community size large enough to support indefinite transmission was achieved.

The estimates of R_0 for the lineage-1 rinderpest virus in the range of 4–5 agree with the field experience in eradication campaigns. Rinderpest has been provisionally eradicated over vast areas, but immunity prevalence levels of 80% have rarely been achieved. Measured levels of population immunity are more typically in the range of 60–70% (IAEA, 1992; IAEA, 1993).

It should be noted that the Sudanese serologic data was the result of serum neutralisation testing whereas the Somali data was obtained with the C-ELISA (Office International des Epizooties, 2002). The sensitivity of the SNT is >99.5%. Concerns regarding the sensitivity of the C-ELISA for lineage-2 virus at the 50% PI cut-off have led to reconsideration of the cut-off point. For this reason, the authors chose to calculate R_0 using two cut-off points, 35 and 40% on the recommendation of the IAEA, and to model the uncertainty in the estimate. More recently, estimates of the sensitivity of the C-ELISA using the 50% cut-off point have been made and are 70% (C.I. $\pm$ 15%) (Drs. John Crowther and Roland Geiger, personal communication). Estimations of R_0 in the range of 1 to 2 are relatively insensitive to imprecision of serological estimates. The range of R_0 values (1.2–1.9) used in the model reflect this imprecision.

As lineage-2 rinderpest virus has persisted as a more or less occult problem over the past few decades, much less is known about the control of this virus (Mariner and Roeder, 2003). The values of R_0 found in this study (1.2–1.9) indicate that a 50% prevalence of immunity would be sufficient to control rinderpest due to lineage-2 in cattle in a one-host system. However, lineage-2 rinderpest in the Somali ecosystem is circulating in a multi-host system involving buffalo, kudu and wart hog at a minimum. One-host analyses of R_0 do not provide an accurate estimate of the effort required to eradicate a disease from a multi-host system when vaccination is applied to only one host. Roberts and Heesterbeek (2003) have described a method based on generational matrices to estimate an eradication threshold in a multi-host system when the intervention is applied to one host group. The authors recommend that when and where vaccination is applied in the Somali ecosystem, efforts should continue to seek the highest levels of immunity possible.

The homogeneous model estimated a CCS for rinderpest on the order of 200,000 head of cattle for both lineages. This is not surprising given that the estimates for measles are on the order of 250,000 to 400,000 people (Keeling and Grenfell, 1997). Mollison (1995) notes that the CCS is primarily determined by the relation between the generation time of the infection and the replacement rate of hosts in homogeneous populations. This is consistent with our finding that the CCS for the two lineages were similar. The determination of CCS in stochastic models are dependent on the degree of stochasticity incorporated in the model. Recognizing the limitations of the homogeneous model and the available information on stochasticity in the system under study, the authors suggest that this estimate should be treated with caution. The important point is that the concept of CCS does apply to rinderpest and should guide the targeting of vaccination in the case of lineage-1. Populations of $<$100,000 head of cattle are unlikely to act as principal reservoirs of infection.

The finding that populations of 10,000 to 20,000 head of cattle could support lineage-2 replication for periods of more than 1 and 2 years, respectively, suggests that relatively small communities could serve as medium-term reservoirs of infection over periods that span the duration of most vaccination campaigns. This is primarily due to the $\sim$5 year inter-epidemic period associated with lineage-2. This suggests that even small vaccination lacunae in infected or high risk areas could cause eradication failure. This may in part explain the enigmatic persistence of lineage-2 rinderpest in the Somali ecosystem despite periodic mass vaccination campaigns. This highlights the importance of epidemiologically targeting interventions through the identification of all infected and high risk communities.

This is the first rinderpest model to incorporate actual empirical calculations of R_0 data and to use these values to estimate β, the effective contact rate. Previous rinderpest models had used expert opinion on the characteristics of epidemics in combination with the results of laboratory data on latency and virus shedding to construct models (James and Rossiter, 1989; Rossiter and James, 1989; Tille et al., 1991). In these previous models, values of β were introduced that gave the desired shape to the epidemic curve and duration of the epidemic. However, application of Eq. (6) to the parameter assumptions of these models indicates that values of R_0 in the range of 2–5 for highly pathogenic virus and 1.2–2 for mild virus were used. Thus, the empirical estimates of R_0 and the findings of the present model are consistent with the expert opinion implicit in previous models.

The predominant opinion expressed by herders in endemic areas of southern Sudan was that some form of rinderpest outbreak occurred every 1–2 years. It has long been argued by experts that an inter-epidemic period approaching two years is required for enough susceptibles to build up in a population to support a new epidemic. The result from southern Sudan was considerably different from that found in Somalia where time series analysis of livestock owner reports indicated an inter-epidemic period of 5 years (Mariner and Roeder, 2003).

Using the southern Sudan parameter estimates, the model indicates an inter-epidemic period of 1.2 years and this compares favourably with the herders estimate of one to two years and conventional expert opinion. Furthermore, using parameter values based on the Somali endemic system, the model predicted an inter-epidemic period of 4.2 years. The difference in the estimates of inter-epidemic period for southern Sudan and Somalia are largely due to the differences between R_0 values between the two lineages in their respective populations. The results indicate that the model is qualitatively valid in both endemic settings.

It is important to note that both modelling predictions of the inter-epidemic period were made without vaccination and that field data was collected in the absence of appreciable vaccination. The community size of 200,000 was chosen in the simulations so as to be at or above the CCS for stochastic extinctions of virus transmission and within the range of community sizes in Sudan and Somalia ($\sim$10,000–700,000 head of cattle). Fade-out and the effect of vaccination on epidemic periodicity are important phenomena that will be described in detail in a future publication.

The model has important implications for surveillance programs. The OIE pathway for the verification of the eradication of rinderpest currently requires that random serological and clinical surveillance be carried using a sample size sufficient to detect clinical cases of rinderpest with 95% confidence if they were present at a 1% prevalence or more (Office International des Epizooties, 1999). For clinical surveillance, this requires examining all animals in 300 herds per epidemiological population, assuming the examination method is a 100% sensitive. As indicated in Table 4, the model predicts that the average prevalence of infectious cases during periods of dynamic endemic persistence does not exceed 0.0019 (0.19%) and 0.0034 (0.34%) for lineage-1 and lineage-2, respectively. Thus, the prevalence of clinically detectable cases will be on the order of 0.1% in an affected population. Only in the most catastrophic epidemics would the prevalence of clinical cases approach 1% for an extremely brief period. Thus, the current OIE requirement is unlikely to detect clinical disease if it were present.

In order to have 95% confidence of detecting disease at more realistic prevalence levels (0.1%) using random sampling methods, the sample size would need to be at least 3000 herds per population (Cannon and Roe, 1982). This would be a cumbersome and costly undertaking. The use of participatory epidemiological approaches have been shown to be a sensitive and efficient alternative to random sampling methods in clinical surveillance (Mariner and Roeder, 2003). This is due to the fact the participatory methodologies make use of all available intelligence including traditional knowledge and well developed traditional pastoral communication networks (Mariner et al., 2003a).

The impact of initial prevalence of immunity (Fig. 2) on the persistence of lineage-1 rinderpest following an introduction is an illustration of the value of the model. Low and

intermediate prevalences of immunity relative to the transmissibility of an agent protect some individual but not the population. These initial conditions are equivalent to low levels of vaccination prior to an introduction. Partial vaccination coverage sets conditions for insidious introductions that are difficult to detect and that can lead to low level endemism.

The modelling of low immunization rates in infected populations demonstrated that vaccination can suppress the prevalence of disease as much as 10-fold and double the inter-epidemic period. This supports the view that sub-optimal, institutionalised vaccination can mask the presence of disease and decrease the efficacy of surveillance programs.

In an eradication program, regardless of whether the population is infected or free of infection, poor vaccination is worse than no vaccination. The results illustrate the importance of the cessation of vaccination in the OIE pathway as a prerequisite to the verification of viral eradication (Office International des Epizooties, 1999). They indicate that vaccination should only be carried out when and where high levels of coverage can be achieved. If good coverage cannot be achieved, it is better not to vaccinate.

The model assumed a homogeneous contact structure implying that all individuals had an equivalent contact experience. However, human and animal populations in the pastoral areas of Africa are structured along ethnic lines. The various tribes and even clans within tribes herd their cattle as identifiable sub-populations. Thus, national or regional populations can be viewed as collections of sub-populations with specific group and inter-group contact rates. The serological data utilized to estimate transmissibility in this study is primarily sub-population or community level data. The global prevalence of immunity needed to eradicate rinderpest from a country is probably not as great as that required to interrupt transmission within a defined sub-population. This suggests regional or national immunity levels of less than the community level critical threshold of 77.4% predicted by the homogeneous model can be sufficient to eradicate rinderpest. This was in fact what was observed during the Pan African Rinderpest Campaign (IAEA, 1992, 1993). By the same token, the level of immunity required to interrupt transmission at the individual herd level may be higher than that estimated from data at the sub-population level.

Studies with heterogeneous contact models have shown that optimal vaccination strategies should target high density, high-risk populations (May and Anderson, 1984). In terms of rinderpest eradication, this implies focusing vaccination on large sub-populations with high contact rates. In the case of lineage-1, vaccination in key populations must attain or exceed the herd immunity thresholds (h) defined by the deterministic calculations. The approach of participatory epidemiology is particularly well suited to the studying of contact structure and risk as a dimension of traditional community structure. The effect of population structure on rinderpest transmission in a heterogeneous model is explored in a subsequent paper.

Surveillance and eradication guidelines are based upon a number of epidemiologic assumptions that should be tested for internal coherence and agreement with available epidemiological information. Open population models allow experimentation with both epidemic and endemic patterns of disease transmission. Stochastic models permit investigation of CCS. The use of participatory epidemiology to collect expert opinion from livestock owners who have first-hand experience with rinderpest addresses the common criticism that models are distanced from the practical reality of the field. This simple, easily-understood model has served as an effective communication tool that has helped

decision-makers to better understand the dynamics of rinderpest. The model directly contributed to the decision to revise the eradication strategy in southern Sudan in 2001 and the strategy described in the last paragraph was adopted. Institutionalized mass vaccination was ceased and resources were focused on large, high-risk communities identified by participatory techniques. An intensive 12-month program of community-based vaccination (Catley et al., 2004; Mariner et al., 1994) achieved >80% vaccination within the targeted areas and vaccination was immediately halted. Rinderpest has not been detected in southern Sudan since that time and the Government of Sudan officially reclassified the region from an infected to a surveillance zone in their declaration to the Office International des Epizooties (OIE) in 2003. Intensive, participatory surveillance is in place and it is anticipated that Sudan will declare Provisional Freedom from Rinderpest in the near future (Office International des Epizooties, 2003). This declaration invokes an OIE verification process that includes the randomized sero-surveillance described above.

In the Somali eco-system, community-based delivery systems are not fully in place and there is not yet a consensus on geographical extent of the endemic system due to the clinically mild form of disease in cattle. The suspicion that small communities are potentially capable of harbouring infection for a prolonged period of time has lead to a strategy where vaccination is being with-held while a community-based delivery and surveillance system is being built that can reliably achieve epidemiologically targeted vaccination to cattle at maximum coverage levels despite chronic insecurity (Catley et al., 2004).

Acknowledgements

The authors would like to acknowledge the support of the Community Animal Health and Participatory Epidemiology Unit of the African Union/Programme for the Pan African Control of Epizootics for their support of the fieldwork and initial modelling work. The CAPE Unit is funded by the Department for International Development (United Kingdom). The authors would also like to thank the Animal Health Division of FAO for their support of further modelling and the dissemination of results.

References

Anderson, J., McKay, J.A., Butcher, R.N., 1991. The use of monoclonal antibodies in competitive ELISA for the detection of antibodies to rinderpest and peste des petits ruminants viruses. The Sero-Monitoring of Rinderpest Throughout Africa. Phase One. Proceedings of the Final Research Coordination Meeting of the IAEA Rinderpest Control Projects. International Atomic Energy Agency, Vienna, Austria.

Anderson, R.M., 1992. The concept of herd immunity and the design of community-based immunization programmes. Vaccine 10, 928–935.

Anderson, R.M., May, R.M., 1991. Infectious Disease of Humans. Oxford University Press, New York.

Barrett, T., Forsyth, M.A., Inui, K., Wamwayi, H.M., Kock, R., Wambua, J., Mwanzia, J., Rossiter, P.B., 1998. Rediscovery of the second African lineage of rinderpest virus: its epidemiological significance. Vet. Rec. 142, 669–671.

Cannon, R.M., Roe, R.T., 1982. Livestock Disease Surveys: A Field Manual for Veterinarians. Australian Government Publishing Service, Canberra.

Catley, A., Irungu, P., Simiyu, K., Dadye, J., Mwakio, W., Kiragu, J., Nyamwaro, S.O., 2002. Participatory investigations of bovine trypanosomiasis in Tana River District Kenya. Med. Vet. Entomol. 16, 1–12.

Catley, A., Leyland, T., Mariner, J.C., Akabwai, D.M.O., Admassu, B., Asfaw, W., Bekele, G., Hassan, H.Sh., 2004. Para-veterinary professionals and the development of quality, self-sustaining community-based services. OIE Rev. Sci. Tech. 23, 225–252.

Catley, A., Okoth, S., Osman, J., Fison, T., Njiru, Z., Mwangi, J., Jones, B.A., Leyland, T.J., 2001. Participatory diagnosis of a chronic wasting disease in cattle in southern Sudan. Prevent. Vet. Med. 51, 161–181.

Catley, A.P., Mohammed, A.A., 1996. The use of livestock-disease scoring by a primary animal health programme in Somaliland. Prevent. Vet. Med. 23, 175–186.

Diekmann, O., Heesterbeek, J.A.P., 2000. Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation. John Wiley & Sons, Chichester.

IAEA, 1992. The Sero-Monitoring of Rinderpest Throughout Africa: Phase II. FAO/IAEA Division of Nuclear Techniques in Food and Agriculture, International Atomic Energy Agency, Vienna.

IAEA, 1993. The Sero-Monitoring of Rinderpest Throughout Africa: Phase II. FAO/IAEA Division of Nuclear Techniques in Food and Agriculture, International Atomic Energy Agency, Vienna.

Jacquez, J.A., 1996. Compartmental Analysis in Biology and Medicine. BioMedware, Ann Arbor.

James, A.D., Rossiter, P.B., 1989. An epidemiological model of rinderpest I. Description of the model. Trop. Anim. Health Prod. 21, 59–68.

Keeling, M.J., Grenfell, B.T., 1997. Disease extinction and community size: modeling the persistence of measles. Science 275, 65–67.

Leiss, B., Plowright, W., 1964. Studies on the pathogenesis of rinderpest in experimental cattle. I. Correlation of clinical signs, viraemia and virus excretion by various routes. J. Hygeine 62, 81–100.

Majok, A.A., Zessin, K.H., Baumann, M.P.O., Farver, T.B., 1991. Analyses of baseline survey data on rinderpest in Bahr el Ghazal Province, with proposal of an improved vaccination strategy against rinderpest for southern Sudan. Trop. Anim. Health Prod. 23, 186–196.

Mariner, J.C., Akabwai, D.M.O., Toyang, J., Zoyem, N., Ngangnou, A., 1994. Community-based vaccination with thermostable Vero cell-adapted rinderpest vaccine (Thermovax). The Kenyan Veterinarian 18, 507–509.

Mariner, J.C., House, J.A., Sollod, A.E., Stem, C., van den Ende, M., Mebus, C.A., 1990. Comparison of the effect of various chemical stabilizers and lyophilization cycles on the thermostability of a Vero cell-adapted rinderpest vaccine. Vet. Microbiol. 21, 195–209.

Mariner, J.C., Jeggo, M.H., van't Klooster, G.G., Geiger, R., Roeder, P.L., 2003a. Rinderpest surveillance performance monitoring using quantifiable indicators. OIE Rev. Sci. Tech. 22, 837–847.

Mariner, J.C., Hussein, M., Roeder, P.L., Catley, A., 2003b. The use of participatory disease searching as a form of active disease searching in Pakistan for rinderpest and more. In: Proceedings of the 10th International Symposium on Veterinary Epidemiology and Economics.

Mariner, J.C., Paskin, R., 2000. Manual on Participatory Epidemiology. Food and Agriculture Organisation, Rome, 81.

Mariner, J.C., Roeder, P.L., 2003. Use of participatory epidemiology in studies of the persistence of lineage-2 rinderpest virus in East Africa. Vet. Rec. 152, 641–647.

May, R.M., Anderson, R.M., 1984. Spatial heterogeneity and the design of immunization programmes. Math Biosci. 72, 83–111.

Mollison, D., 1995. Epidemic Models: Their Strucuture and Relation to Data. In: The Strucuture of Epidemic Models 17-34. D. CUP.

Office International des Epizooties, 1999. Recommended Standards for Epidemiological Surveillance Systems for Rinderpest. In: International Animal Health Code 395-400. Office International des Epizooties, Paris.

Office International des Epizooties, 2002. Rinderpest. In: Manual of Standards: Diagnostic Tests and Vaccines 2000 (Office International des Epizooties, Ed.), Office International des Epizooties, Paris, France, Chapter 2.1.4.

Office International des Epizooties, 2003. Rinderpest in Sudan: Progress along the OIE pathway (extension of the surveillance zone). Disease Information 16, 1.

Plowright, W., 1965. III. - Rinderpest. Vet. Rec. 77, 1431–1438.

Plowright, W., 1984. The duration of immunity in cattle following inoculation of rinderpest cell culture vaccine. J. Hyg. 92, 285–296.

Plowright, W., 1986. Rinderpest in the world today. State Vet. J. 40, 44–70.

Roberts, M.G., Heesterbeek, J.A.P., 2003. A new method for estimating the effort required to control infectious disease. Proc. R. Soc. Series B. Biol. Sci. 270, 1359–1364.

Robson, J., Arnold, R.M., Plowright, W., Scott, G.R., 1959. The isolation from an eland of a strain of rinderpest virus attenuated for cattle. Bull. Epizootic Dis. Africa 7, 97–102.

Rossiter, P.B., James, A.D., 1989. An epidemiological model of rinderpest II. Simulations of the behavior of rinderpest virus in populations. Trop. Anim. Health Prod. 21, 69–84.

Schwabe, C.W., Makuet Koujok, I., 1981. Practices and beliefs of the traditional Dinka healer in relation to provision of modern medical and veterinary services for Southern Sudan. Hum. Org. 40, 231–238.

Taylor, W.P., Roeder, P.L., Rweyemamu, M.M., Melewas, J.N., Majuva, P., Kimaro, R.T., Mollel, J.N., Mtei, B.J., Wambura, P., Anderson, J., Rossiter, P.B., Kock, R., Melengeya, T., Van den Ende, R., 2002. The control of rinderpest in Tanzania between 1997 and 1998. Trop. Anim. Health Prod. 34, 471–487.

Tempia, S., Njeumi, F., Cagnolati, V., Salman, M.D., Wamwayi, H.M., Abdi, A.M., 2003. Serological investigation of rinderpest in Lower and Middle Juba Regions of southern Somalia. In: Proceedings of the 10th International Symposium on Veterinary Epidemiology and Economics.

Terra Nuova, personal communication.

Tille, A., Lefevre, C.L., Pastoret, P.P., Thiry, E., 1991. A mathmatical model of rinderpest infection in cattle populations. Epidemiol. Infect. 107, 441–452.

Wamwayi, H., personal communication.

Waters-Bayer, A., Bayer, W., 1994. Planning with pastoralists: PRA and more. A review of methods focused on Africa. Working Paper. Deutsche Gesellschaft fur Technische Zusammemarbeit (GTZ) Division 422, Eschborn, p. 153.