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AN EPIDEMIOLOGICAL MODEL OF RINDERPEST. II. SIMULATIONS OF THE BEHAVIOUR OF RINDERPEST VIRUS IN POPULATIONS

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SUMMARY

Fixed parameters for different hypothetical strains of rinderpest virus (RV) and different susceptible populations are described together with details of their derivation. Simulations were then carried out in a computer model to determine the effects that varying these parameters would have on the behaviour of RV in the different populations. The results indicated that virulent strains of RV are more likely to behave in epidemic fashion whereas milder strains tend towards persistence and the establishment of endemicity. High herd immunity levels prevent virus transmission and low herd immunity levels encourage epidemic transmission. Intermediate levels of immunity assist the establishment of endemicity. The virus is able to persist in large populations for longer than in small populations. Different vaccination strategies were also investigated. In areas where vaccination is inefficient annual vaccination of all stock may be the best policy for inducing high levels of herd immunity. In endemic areas and in herds recovering from epidemics the prevalence of clinically affected animals may be very low. In these situations veterinary officers are more likely to find clinical cases by examining cattle for mouth lesions rather than by checking for diarrhoea or high mortalities.

INTRODUCTION

Compared with most other virus diseases rinderpest has an uncomplicated biology. There is only one serotype of the virus (RV), recovered animals are solidly immune to re-infection, there is no carrier state resulting in re-excretion of virus, no vertical transmission and no transmission by arthropod vectors. As a consequence it has proved possible to control and even eradicate the disease. The simple measures of slaughter and animal movement control which proved effective during the last century have now largely been replaced, except in virgin territories, by vaccination and movement control. The success of live vaccines led to an attempt through the JP15 programme to eradicate rinderpest from Africa. The programme was effective but terminated in 1976 before final elimination of the virus from two persistent foci. During the ensuing six years the virus emerged from these foci to infect nearly as many countries as before the start of JP15. This pandemic has stimulated repeated demands for the eradication of rinderpest from Africa.

Another morbillivirus, measles (MV), which has an equally simple epidemiology, has also been the target of possible eradication. However, despite concerted efforts MV persists in human populations. Efforts to understand this persistence and to improve control measures through computer simulation of MV in known populations have proved useful (Anderson and May, 1982; Bart, Orenstein, Hinman and Amler, 1983; Fine and Clarkson, 1983). Therefore simulations of rinderpest were attempted in the hope they would give insight into the behaviour

of RV in various populations. This might assist in the design of more efficient or economic control methods.

In particular information was sought on the following problems:

1. The minimum herd size necessary for persistence of the virus.
2. Clinical outbreaks frequently "burn out" before being visited for confirmation by veterinarians. In such situations is it still possible to detect the virus?
3. Strains of rinderpest virus vary considerably in virulence and transmissibility. It is generally accepted that epidemics are characterised by virulent strains whereas mild strains are typical in endemic areas. Do these different strains generate and proliferate by random mechanisms or are there pressures which encourage the predominance of particular strains?
4. If vaccination cannot be carried out with high efficiency what is the most effective schedule to give high levels of herd immunity?

This paper describes computer simulations of the behaviour of RV in different populations. There is a paucity of data on most of the variables used in the studies. This has meant that hypothetical models for different strains of rinderpest have had to be prepared using parameter values derived largely by assumption and extrapolation from the little data which is available. The derivation of the characteristics of the hypothetical strains and to some extent those of the host populations are described in detail since they form the basis of these simulations. Certain values may be considered contentious but it is felt that they are justified as they have been selected to cover the range which such variables e.g. incubation period, may be expected to cover in the field. They are only intended to illustrate possibilities rather than probabilities or to confirm previous field and experimental observations.

SIMULATIONS

These were carried out by starting the model (James and Rossiter, 1989) with a chosen herd situation and then initiating the disease outbreak through the introduction of five infected animals. This number was used to ensure that the virus "took" in the population rather than failing to spread from one or two infected introductions due to the model's stochastic approach. Simulations were carried out to test the effects of varying parameters described below and were usually run either until the virus died out in the population or persisted for at least one year.

RINDERPEST VIRUS STRAINS

Four strains of high, moderate, low and mild virulence were devised. The fixed values of each of the following variables are shown for each strain in Table I; contact incubation period, virus excretion period, incidence of mouth lesions, incidence of diarrhoea, incidence of mortality and contact rate. The distribution of these values between the strains relies heavily on the following sequence of assumptions which will be referred to as the Basic Assumption (BA); increased virulence = increased lesion formation = increased virus excretion = increased virus infectious dose = decreased incubation period. That is, highly virulent strains spread more readily and have shorter incubation periods than less virulent strains and *vice-versa*.

TABLE I

The parameters for four model strains of rinderpest virus

	High virulence	Moderate virulence	Low virulence	Mild
Incubation period (days)	4	6	8	10
Virus excretion period (days)	6	5	4	3
Contact rate	2·0	1·0	0·5	0·25
Mouth lesions (% prevalence)	100	100	80	15
Diarrhoea (% prevalence)	100	50	25	5
Mortality (% prevalence)	90	25	7	2

Contact incubation period (CIP)

Leiss and Plowright (1964) determined a mean CIP of 8·6 days for 11 cattle infected with RV strain RGK/1. Since they had shown that virus excretion starts before the onset of pyrexia in some animals and that the mean titre of virus excretion tended to parallel viraemia their CIP was calculated from the time of first exposure to a viraemic donor until onset of pyrexia in the "in-contact" animal. However, this initial virus excretion was at a very low level and only occurred in a small proportion of animals so it was unlikely that transmission usually occurred during that time. Therefore it is suggested that the CIP be revised to be the time after first exposure to animals excreting more than 2·0 $\log_{10}$ $tcid_{50}$ per nasal swab. A recalculated CIP for the data of Leiss and Plowright (1964) would therefore be 5·6 days. Similarly, the figure of seven to eight days determined by Rweyemamu, Reid and Okuna (1974) for strain RGK/1 can be reduced to five to six days. Strain RGK/1 is taken as intermediate between high and moderate virulence strains for the purposes of the model.

Using this method of determination a CIP of eight days can be calculated for data obtained using strains of low virulence isolated from cattle and buffalo, *Syncerus caffer,* in northern Tanzania (Plowright, 1963) and of nine days for the strain RET/1 isolated from an eland in Tanganyika (Robson, Arnold, Plowright and Scott, 1959). Thus although there are some exceptions e.g. CIP of 10·3 days for four cattle infected with the virulent Malakal strain of RV (Plowright, pers. comm.), these values tend to show that CIP increases with decreasing virulence and support the range of CIP used in the simulations.

Virus excretion period (VEP)

Rinderpest spreads by close contagion. Some studies (Leiss and Plowright, 1964; Plowright, 1963) have shown efficient transmission, whereas other studies of experimental contact infection have been unable to produce the disease with such reliability (Taylor, Plowright, Pillinger, Rampton and Staple, 1965; Plowright, 1968; Rweyemamu *et al.,* 1974). It is assumed that RV transmission is not 100% efficient and must be associated with peak periods of virus excretion. Therefore, the period of nasal excretion of infectivity of more than 2·0 $\log_{10}$ $tcid_{50}$ per swab from the data of Leiss and Plowright (1964) for RGK/1 is used, i.e. five to six days. Although there are isolated reports that some mild strains spread rapidly the BA is adhered to and VEP decreases with virulence.

Mouth lesions (ML)

These are the commonest clinical signs of rinderpest (Scott, 1967; Plowright, 1968) and are considered to occur at 100% frequency in the two most virulent model strains. The 80% incidence for ML in the low virulence strain is based on the 15 of 22 and 21 of 21 *Bos taurus* × *Bos indicus* cattle showing these lesions in studies by Plowright (1963) and Robson *et al.* (1959), respectively. The incidence of 15% for the mild strain is derived from the detection of ML in only three of 17 zebu cattle infected with the RET/1 strain (Robson *et al.*, 1959).

Diarrhoea

The high virulence model strain causes 100% incidence of diarrhoea. The incidences for low virulence and mild models are derived from the data of Robson *et al.* (1959) and Plowright (1963). The value of 50% for the moderate virulence model strain is an interpolation.

Mortality

Mortality in rinderpest rarely approaches 100% unless complicated by intercurrent infections (Scott, 1967). Accordingly the highly virulent model strain is assumed to cause 90% mortality. Again, values for low virulent and mild model strains are derived from the data of Robson *et al.* (1959) and Plowright (1963) and interpolated to provide the value for moderate virulence.

Contact rate (C_R)

There are no data which can be used to estimate the value of C_R. Therefore the range of values used has been determined by trial and error. Results obtained using this range allow the model to simulate outbreaks that appear similar to those reported from the field. It adheres to the BA and increases with virulence.

POPULATIONS

The simulated populations were, as stated earlier (James and Rossiter, 1989), assumed to be homogeneous. The following parameters were required; herd size, herd age composition, overall herd mortality, neonatal calf mortality, herd calving rate, seasonality of calving, overall herd immunity, persistence of maternal immunity and vaccination schedules. Certain values were used most frequently and are detailed below. Where simulations used other values these are stated in the results.

Herd size

Herd sizes of 1,000 or 10,000 were frequently used. The former was considered representative of village congregations of cattle, the latter representative of units which gather near water in dry seasons. In practice such units are not homogeneous but were treated as such for the sake of these preliminary findings. Units of 100,000 or more were infrequently used since the assumptions of homogeneity and perfect mixing would be much less realistic for such large populations.

Mortality

Overall herd mortality was set at 10% p.a. and neonatal mortality at 20%. These are typical of values encountered in pastoral herds in East Africa and in reality vary markedly with husbandry and weather.

Calving rate

This was set at 1·8% per month which is consistent with reported rates in East African pastoral systems. Although there is some variation in the seasonal calving rate in pastoral herds year-round calving is a common husbandry objective in order to insure a constant supply of milk. Young calves are always a feature of pastoral herds and the model used a constant monthly calving rate throughout the year.

Immunity levels

These were set at a desired level e.g. 70% at the start of a simulation. The level then falls as immune animals die and susceptible replacements enter the herd. Immunity levels could be raised by introducing vaccination at a chosen time and level of efficiency. Vaccination or field infection did not cause development of immunity in calves 0–180 days old if these had immune dams. This maternal immunity then waned between 180 to 270 days and vaccination in such animals was assumed to be effective in 50% of cases. Therefore virulent challenge of partial-immunes caused a less virulent syndrome than in fully-immunes so the RV model strain used in these animals was always less virulent than that used in fully susceptible animals.

RESULTS

These are presented mainly as a series of epidemic curves depicting the prevalence of infected animals in the herd.

Contact rate

The effect of varying C_R for a strain of RV with all other parameters constant is shown in Fig. 1 using a herd of 10,000 cattle with an initial herd immunity of 60%. With increasing transmissibility of virus strains the epidemic curve reached its peak earlier and higher than less transmissible strains, which spread less rapidly and persisted at lower prevalences for longer periods of time. Due to a lack of susceptibles highly transmissible strains tended to die out more rapidly than did less transmissible strains. Changes in the C_R produced greater degrees of change in epidemic curves than any other parameter, indicating that the model is especially sensitive to it and that it may be the most important single factor in determining the spread of the virus.

Virus excretion period

Increased VEP caused higher and earlier peaks of the epidemic curve whereas shorter periods caused lower and later peaks with a tendency to persist. The effect of increasing the VEP was similar, as would be expected, to increasing the C_R.

Contact incubation period

The model was relatively insensitive to changes in this parameter. The peak prevalences of infected animals were only slightly decreased and delayed by doubling the CIP.

Herd size

The onset of the peak of the epidemic curve was delayed by increasing herd size, but its height was not altered by changes in herd size (Fig. 2). A plot of the

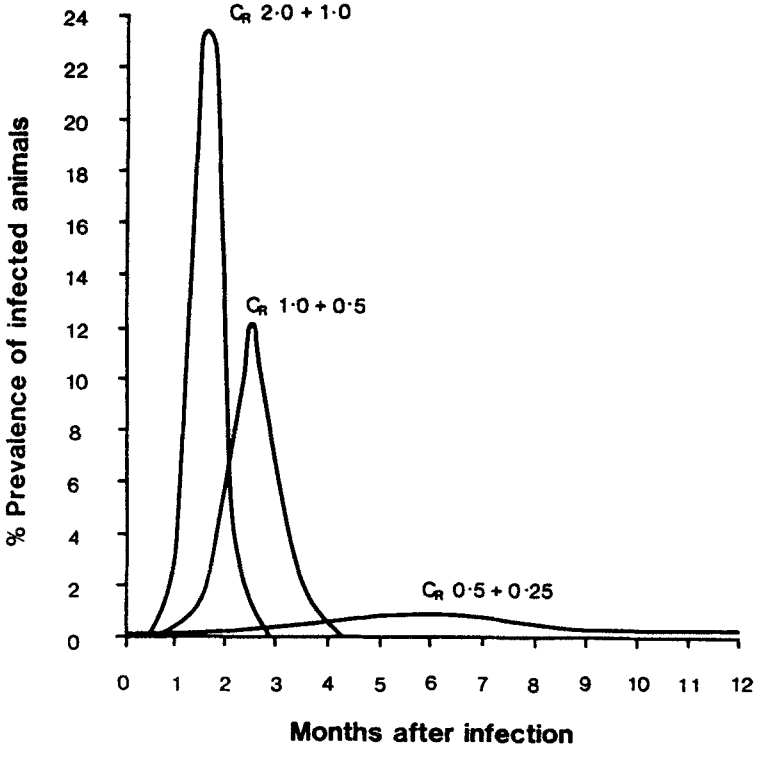

FIG. 1. The prevalence of infected animals in outbreaks of rinderpest caused by strains with different contact rates.

A herd of 10,000 animals with 60% initial herd immunity was infected with virus which had a contact incubation period of 6 days, virus excretion period of 5 days and disease mortality of 25%. The pairs of contact rates represent those for a given strain in fully susceptible and partially susceptible animals respectively.

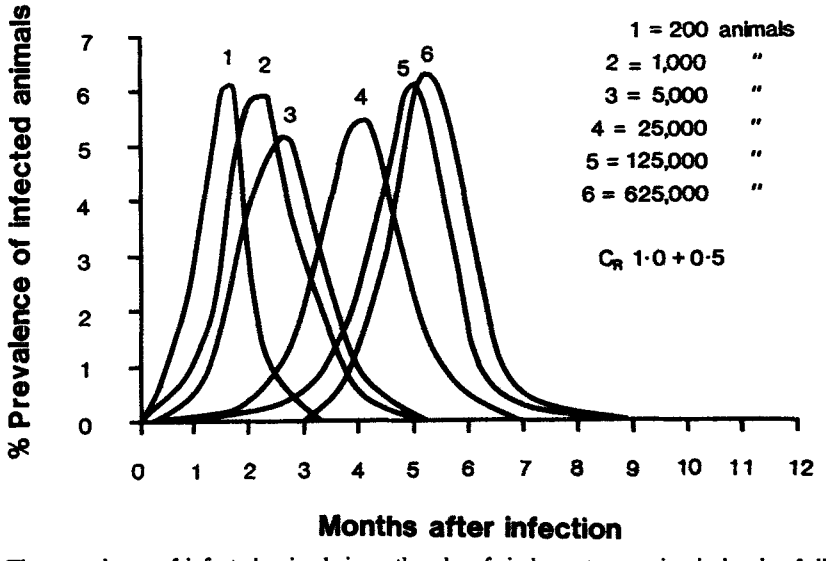

FIG. 2. The prevalence of infected animals in outbreaks of rinderpest occurring in herds of different sizes.

All herds had an initial herd immunity of 60%. The virus used was the model for moderate virulence (Table I).

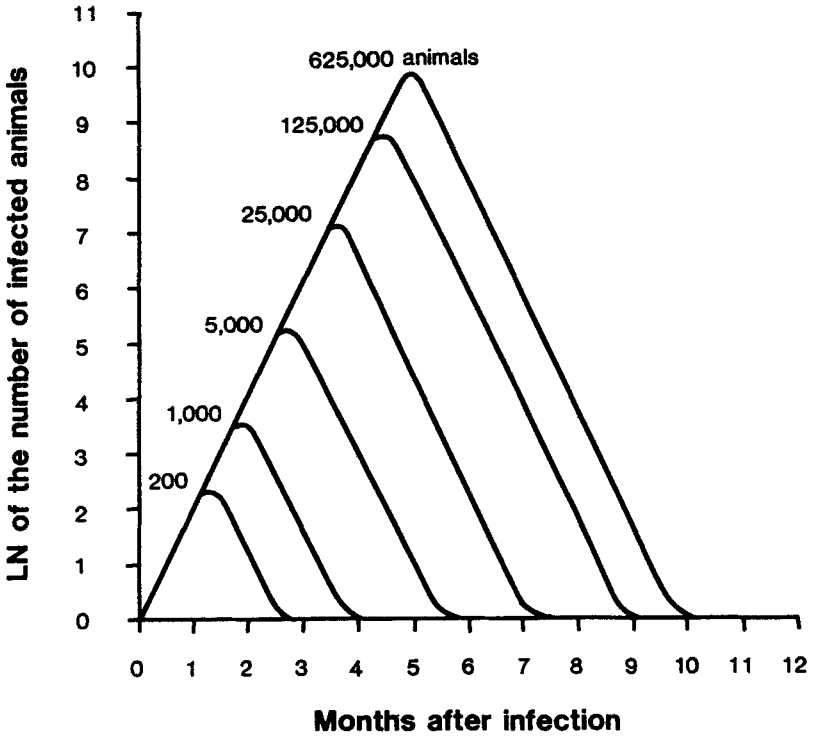

FIG. 3. The number of animals affected in outbreaks of rinderpest in herds of different sizes. Same data as Fig. 2.

logarithm of prevalence of infected animals against time (Fig. 3) showed that the virus multiplies at the same rate in any size of herd until restricted by lack of susceptibles. Increasing herd size increased the length of the epidemic curve.

Initial Herd Immunity

The model was almost as sensitive to changes in initial herd immunity (IHI) as to changes in C_R. Low levels of IHI allowed the development of earlier, higher peak prevalences of infected animals than did higher levels of immunity (Fig. 4). At certain levels of immunity the virus was able to persist in the population for at least 12 months at very low prevalences.

The influence of these various parameters in determining epidemic or endemic behaviour of RV are summarised in Table II.

Herd immunity levels following herd infection

Changes in herd immunity levels after infection of the herd are shown in Fig. 5. Faster, larger outbreaks caused swifter and more elevated rises in the proportion of immune animals in the herd than did slower, smaller outbreaks. It was this swift, high build up of herd immunity which caused epidemic rinderpest to die out due to a lack of susceptibles. In situations where the virus persisted for

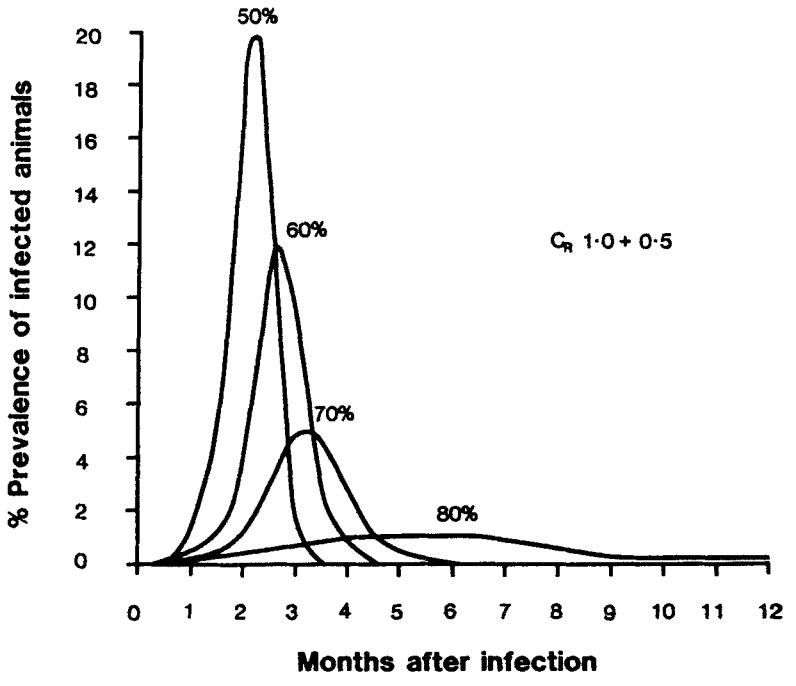

FIG. 4. The prevalence of infected animals in outbreaks of rinderpest occurring in herds with different levels of initial herd immunity.

A herd of 10,000 animals was infected with the moderate virulence model of rinderpest virus (Table I).

long periods at low prevalence the proportion of immune animals remained stable.

Persistence of virus in populations

The early results indicated that it was possible for strains of RV with varying C_R to persist in populations depending upon the IHI level. The probability of

TABLE II

The effect of altering various parameters on the probability of rinderpest virus persisting in a population

Parameter	Alteration in parameter	
	Decrease	Increase
Contact rate	INCREASED PERSISTENCE[1]	DECREASED PERSISTENCE
Virus excretion period	INCREASED PERSISTENCE	DECREASED PERSISTENCE
Initial herd immunity[2]	Increased persistence[1]	Decreased persistence
Contact incubation period	Decreased persistence	Increased persistence
Herd size	DECREASED PERSISTENCE	INCREASED PERSISTENCE

[1] Upper case = marked effect; lower case = slight or possible effect.

[2] Very low and very high levels decrease probability of persistence, intermediate levels encourage persistence.

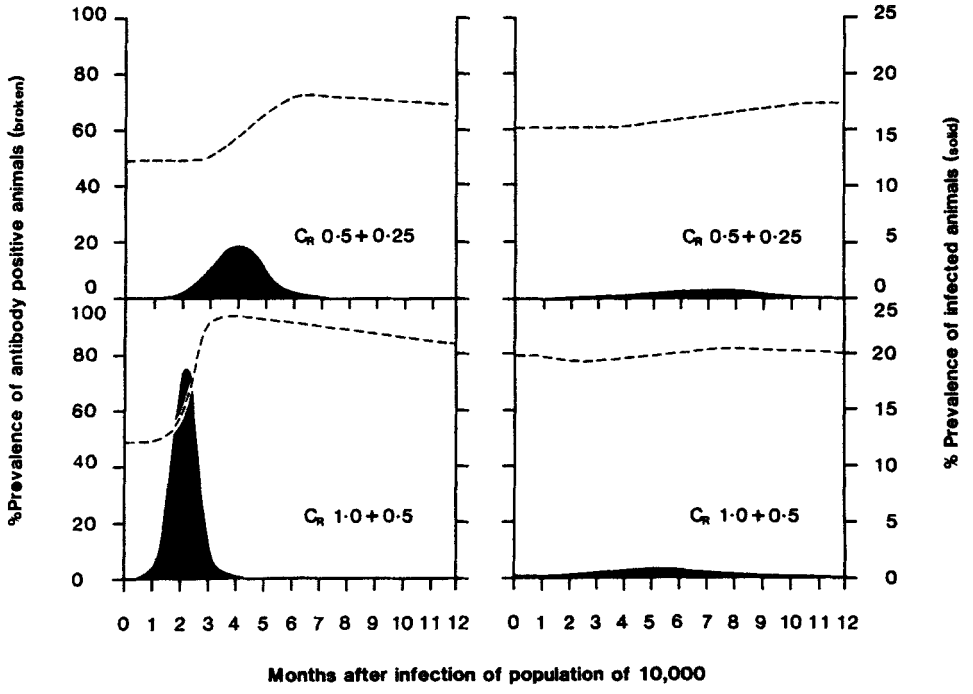

FIG. 5. The prevalence of infected animals and antibody positive animals in outbreaks of rinderpest caused by virus with different contact rates in herds with different initial immunity.

A herd of 10,000 animals was used. The virus parameters used were those for the moderate virulence model (Table I) with varied contact rates.

such persistence occurring was investigated further by determining the proportion of outbreaks that persisted for at least 12 months in 10,000 cattle with a known IHI using RV with a known C_R. As a preliminary to this the ability of a strain to establish itself or "take" in a herd was also examined. Establishment for two months (approximately four cycles of infection) was considered indicative of "take".

The effect of IHI on the ability of RV with different C_R to "take" is shown in Fig. 6. Strains with low transmissibility were prevented from "taking" by relatively low levels of IHI whereas only very high levels of IHI prevented "take" by highly transmissible strains. The probability of a strain persisting for 12 months, which was taken as an arbitrary measure of endemicity, was then determined by 10 repeated simulations of virus which had "taken" in the herd. This probability was determined for strains with different C_R at differing IHI levels and the results are shown in Fig. 7. All strains of RV persisted in the population depending upon the IHI, increasingly transmissible strains only persisting at increasingly higher IHI. The peak probability for persistence of the low and moderate strains was 1·0 indicating that with these strains it was relatively easy to choose an IHI in which the replacement rate of susceptibles always balanced the rate of spread of RV in the herd. The peak probability of 0·9 for the highly transmissible strain was also very high and might also have reached 1·0 if a narrower range of IHI had been examined.

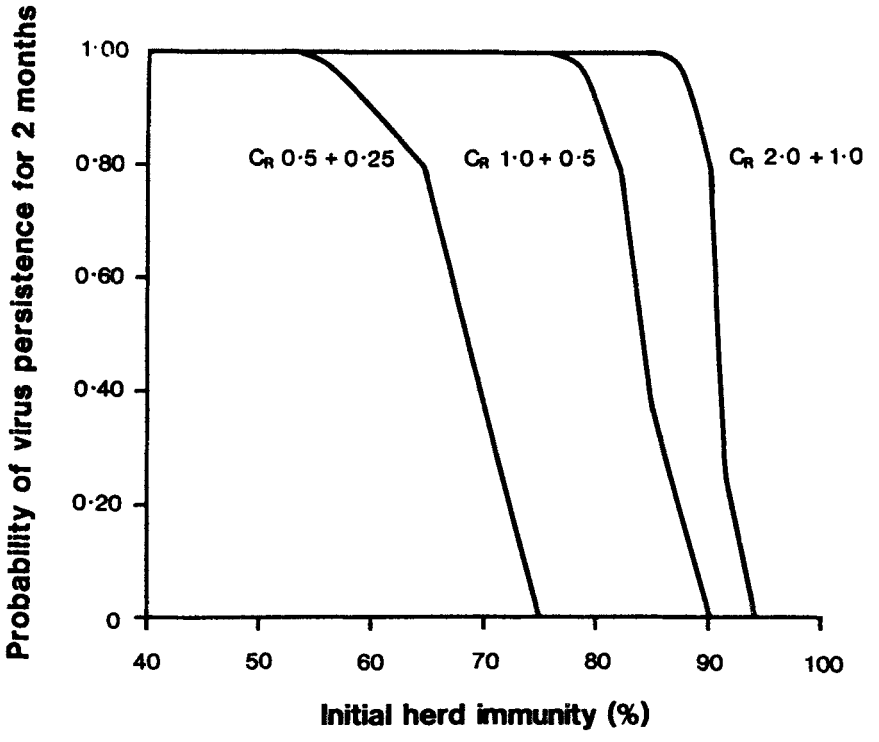

FIG. 6. The probability of rinderpest virus persisting for at least 2 months in herds with different levels of initial herd immunity.

A herd of 10,000 animals was used. The virus parameters were those for the moderate virulence model with varying contact rates. The pairs of contact rates represent those for a given strain in fully susceptible and partially susceptible animals respectively.

More important was the range of IHI over which a strain had a high probability of persisting. This was significantly greater for the less transmissible strains. In the examples used (Fig. 6) the range of IHI which would give a probability of persistence greater than 0·5 decreased from 9·1% to 3·8% to 1·7% when CR was increased from 0·5 to 1·0 to 2·0. In repeated simulations using RV with a CR of 0·5 the immunity level in herds persistently infected at low prevalences rose after infection from 55% to 60% to 65% to 75%. In one of 10 simulations RV with a C_R of 0·5 persisted for 12 months in a herd of 1,000 cattle with 60% IHI.

Seasonality of calving

All of the results given above were obtained in simulations using a constant rate of calving throughout the year. It was thought that seasonality of calving, especially one short calving season per annum, would concentrate the supply of new susceptibles into a short period thereby making it more difficult for the virus to persist through the remaining part of the year. A three month calving season at a calving rate of 7·2% per month was used. Commencement of the calving season

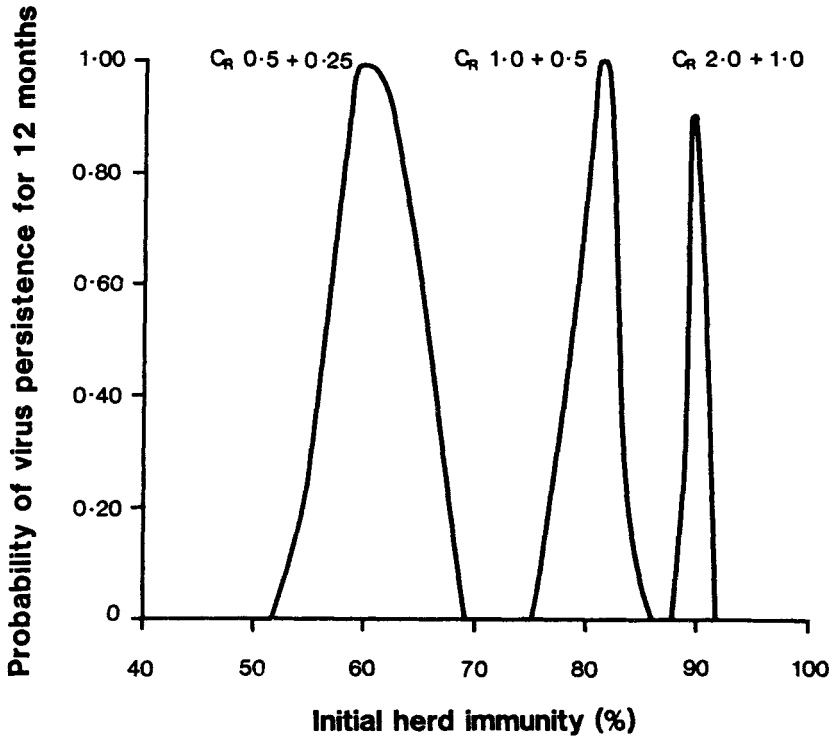

FIG. 7. The probability of rinderpest virus persisting for at least 12 months in herds with different levels of initial herd immunity.
Same herd and virus parameters as for Fig. 6.

in January, April, July and October was simulated with initial herd infection at the start of the year in each case. A mild strain, $C_R = 0{\cdot}5$, was used under conditions known to give a high probability of persistence: 10,000 animals with 60% IHI. The simulation was run twice for each of the four different calving seasons and persistence occurred in all cases. Results from the model showed that this was probably due to the relatively low IHI giving rise to two populations of calves, one born to non-immune dams and therefore susceptible at birth and others which received colostral immunity and became susceptible six to nine months later. The supply of susceptibles was therefore spread over at least six months which was sufficient to support virus persistence.

Vaccination

The model was also used to investigate the development of herd immunity following three different vaccination strategies;

1. vaccination of all stock which is a procedure frequently used for emergency cover and as the first step in eradication programmes employing vaccination;
2. vaccination of all stock once during each of the first two years of life which is the most commonly practised sequel to blanket vaccination and ensures

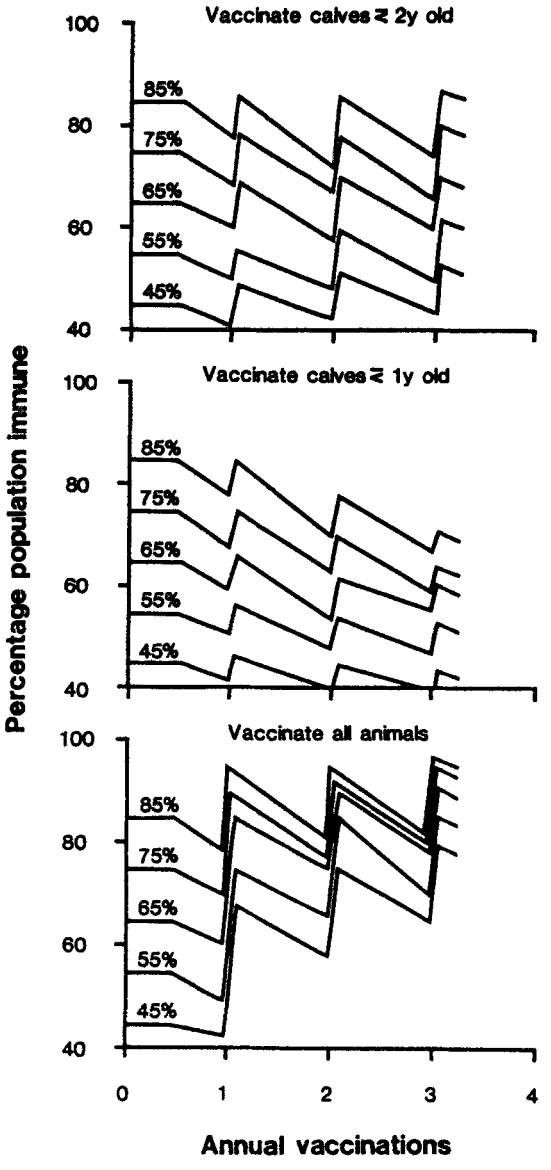

FIG. 8. The prevalence of antibody positive animals in herds vaccinated with rinderpest virus by 3 different techniques and at different levels of efficiency.

A herd of 10,000 animals with an initial herd immunity of 0% was used. The percentages shown above each graph are the level of vaccination efficiency.

that animals vaccinated whilst still passively immune are immunised the following year;

3. vaccination of all stock only once in their first year of life which was thought to represent the most inefficient form of calfhood vaccination likely to occur in practice.

Each strategy was examined using different levels of vaccination efficiency. Vaccination efficiency is defined as the percentage of animals in a given population effectively immunised during a campaign. Herd immunities under the different strategies and levels of vaccination efficiency were followed for four years after an initial vaccination of all stock at the start of the first year (Fig. 8). Vaccination of animals only in their first year was obviously inefficient; the herd immunity constantly fell at all levels of vaccination efficiency. Vaccination of all calves under two years old was more efficient but only maintained herd immunity levels around the level of vaccination efficiency. Vaccination of all stock, however, led to herd immunity levels rising every year particularly for low vaccination efficiencies. After four years vaccination of all stock at 55% efficiency had produced a herd immunity of 86% whereas the comparable levels for vaccination of calves twice and once only were 62% and 54% respectively.

Prevalence of clinical signs

The prevalence of infected animals in populations where RV was able to persist for 12 months or more ranged between 0·5 and 2%. Allowing for a proportion of these animals being in the incubation period then as few as 0·25% to 1% of a population with endemic rinderpest would have clinical disease. Therefore extrapolating from the models of clinical signs for low virulence and mild disease which predominate in endemic areas mortality from rinderpest in such areas could be as low as 0·005% to 0·07% of the population with only 0·013% to 0·25% showing typical diarrhoea. Mouth lesions will be more prevalent at 0·033% to 0·8% of the population.

Where the prevalence of infected animals is higher, say 2% to 5%, the proportion of animals with typical diarrhoea or dying from the disease will still be very low but mouth lesions will be more common. Such situations might occur

TABLE III

The number of animals that must be sampled in order to give a 0·95 probability of detecting rinderpest at various prevalences in populations of different sizes

Population size (number of animals)	Disease prevalence 0·5%	1·0%	2·0%	5·0%
80	80[1] (100)[2]	76 (95)	62 (78)	42 (53)
400	311 (78)	211 (53)	124 (31)	55 (14)
2,000	517 (26)	288 (14)	143 (7)	58 (3)
10,000	581 (6)	294 (3)	148 (1·5)	59 (0·6)

[1] number of animals.
[2] percentage of population.

at the end of a localised epidemic e.g. in the final month of the outbreak caused by virus with CR 1·0 to 0·5 in Fig. 1. Under such circumstances the prevalence of ML would be approximately two to three times higher than diarrhoea and four to 10 times higher than mortality (Table I). The sample sizes that would be required to detect infected animals at such low prevalence rates of clinical disease are shown in Table III.

DISCUSSION

Field observation suggests that in those areas where rinderpest is endemic the size of the population which supports it is usually large, in the order of 0·1 to 1·0 million cattle as in Karamoja district of Uganda or parts of Southern Sudan. The model, however, indicated that it may be possible for RV to persist in much smaller populations. Persistence was consistently demonstrated in a herd of 10,000 and even on one isolated occasion in a herd of 1,000. The heterogeneity of real cattle populations as opposed to the homogeneity assumed in the model will tend to decrease the chance of RV persisting in such small herds. However, the results show that under certain circumstances it may be possible for the virus to persist for months or years at clinically unrecognisable levels in smaller populations than those conventionally thought capable of maintaining RV. Such a situation might account for the unrecognised persistence of RV in the isolated Sonjo Valley in northern Tanzania from early 1961 to late 1965 (Taylor and Watson, 1967).

The low prevalence rates of infected animals in endemic situations indicate that, if veterinarians are to detect clinical cases for laboratory confirmation, then they must examine the relatively large numbers of cattle suggested by Table III. Similarly, careful clinical examination for mouth lesions and pyrexia should detect more infected animals than cursory visual examination for diarrhoea or enquiries about mortality. Such an approach has already proved valuable in the field (P. B. Rossiter, unpub.).

The study of the probability of RV persisting under different conditions provided a possible new explanation for the predominance of mild disease in endemic areas. The mechanism generally thought to be responsible for this is the selection of increasingly resistant cattle or other hosts (Scott, 1967). Undoubtedly host variation is involved but the selective advantage conferred on mild strains by their greater probability of persistence under herd immunity pressure (Fig. 7) could play a part. Mild strains are more likely than virulent strains to balance their rate of spread through a herd to the supply of new susceptibles. This tendency for mild strains to persist longer in infected herds will increase the probability of the mild strain infecting a new herd. Therefore in host populations where antibody prevalence is too high to allow epidemic rinderpest and too low to prevent completely spread of the virus there will be selection of mild and persistent virus sub-populations. By contrast in potential epidemic situations where the antibody prevalence in the population is low there will be enough susceptibles to allow the virus to transmit as rapidly as possible. Consequently the most rapidly transmitting strains will increasingly dominate, which adhering to the BA, would account for the reversion to virulent strains so typical of epidemics.

There are two reasons why this mechanism based on virus as opposed to host variation could be more efficient in the field. Firstly, during an outbreak the virus replication cycle is very much more frequent than that of the host providing more

opportunity for different strains to be generated. Secondly, because of its smaller genome the virus will be more susceptible to major changes caused by minor genetic variation than the host.

The simulations of herd immunity following different vaccination regimes show that attempted immunisation of all calves under two years old is effective at producing high levels of herd immunity only when the efficiency of vaccination is high. Where the efficiency is low high levels of herd immunity cannot be obtained. Under such circumstances only the use of a total herd vaccination policy would produce the high levels of herd immunity required to prevent "take". Such an approach would prove cost-effective since the cost of the extra RV vaccine would be very low compared to the manpower and transport costs which would be similar whether calfhood or whole-herd vaccination regimes were employed.

Despite the potential pitfalls associated with the assumption of homogeneous populations implicit in the model and the lack of published data for most of the virus parameters this epidemiological model has several potential uses:

1. With serological evidence of vaccination campaign efficiencies the efficiency of vaccination programmes can be improved.
2. With serological evidence of herd immunity it should be possible to assess the risk of RV causing epidemic or endemic outbreaks in a host population.
3. Since it is relatively simple to use the model is a suitable tool for teaching aspects of epidemiology, disease control and computer simulation.
4. Validation of the model using new data will improve the theoretical understanding of rinderpest epidemiology.

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UN MODELE EPIDEMIOLOGIQUE POUR LA PESTE BOVINE. II. SIMULATION DU COMPORTEMENT DU VIRUS DE LA PESTE BOVINE DANS LA POPULATION

Résumé–On décrit les paramètres fixes de différentes souches hypothétiques de virus de la peste bovine (RV) dans différentes populations sensibles, avec des détails sur leur dérivation. Des simulations ont été réalisées en modèle informatisé pour déterminer l'effet que la variation de ces paramètres aurait sur le comportement du virus de la peste dans différentes populations. Les résultats de RV ont plus de chances de se comporter de manière épidémique alors que des souches plus atténuées tendent à persister et établir l'endémicité. Des niveaux élevés d'immunité des troupeaux préviennent la transmission du virus, et les niveaux bas d'immunité des troupeaux facilitent une transmission sur le mode épidémique. Les niveaux intermédiaires d'immunité aident à l'établissement de l'endémicité. Le virus est capable de persister plus longtemps dans les grandes populations que dans les petites. Différentes stratégies de vaccination ont également été étudiées. Dans les régions où la vaccination est inefficace, la vaccination annuelle de l'ensemble du cheptel peut s'avérer le meilleur moyen d'induire de hauts niveaux d'immunité de troupeau. Dans les zones d'endémie et dans les troupeaux récupérant d'épizooties la proportion d'animaux cliniquement atteints peut être très réduite. Dans ces situations, les agents vétérinaires ont plus de chances de trouver les cas cliniques par recherche des lésions buccales qu' en se basant sur la diarrhée ou des taux élevé de mortalité.

UN MODELO EPIDEMIOLOGICO DE RINDERPEST. II. SIMULACION DEL COMPORTAMIENTO DEL VIRUS DE RINDERPEST EN LA POBLACION

Resumen–Se describen parámetros fijos para diferentes cepas hipotéticas del virus de rinderpest (VR) y diferentes poblaciones susceptibles, junto con detalles de sus derivaciones. Se llevaron a cabo entonces simulaciones en un modelo computarizado, para determinar los efectos que la variación de estos parámetros tendría, sobre el comportamiento del VR en las diferentes poblaciones. Los resultados indicaron, que las cepas virulentas del VR se comportarían en forma epidémica, mientras que las cepas atenuadas establecerían un estado de endemicidad. La inmunidad alta en el hato impediría la transmisión del virus, mientras que bajos niveles de inmunidad impulsarían la transmisión epidémica. El virus tiende a persistir más en poblaciones grandes, que en pequeñas. Se investigaron también, diferentes estratégias de vacunación. En las áreas donde la vacunación es ineficiente, la vacunación anaul de todo el efectivo ganadero, podria ser la política apropiada para inducir altos niveles de inmunidad. En áreas endémicas y en aquellos hatos en períodos de recuperación de brotes epidémicos, la prevalencia clínica de animales afectados podría ser baja. En estas situaciones, los veterinarios oficiales tendrían más oportunidad de encontrar casos clínicos mediante el examen de la cavidad oral, que esperando encontrar diarrea o alta mortalidad.