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.