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A mathematical model of rinderpest infection in cattle populations

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SUMMARY

A mathematical model for the epidemiology of rinderpest was developed, starting from a simplified descriptive analysis of the disease. A formula for the calculation of the probability of infection of a susceptible animal was first established. A deterministic failure threshold of the infection was then deduced. Deterministic and stochastic approaches were adopted using iterative methods on a computer. These allowed a description of the spread and the variability of an infection process in a population to be made. An illustration of the use of this model showed that, in some cases, variability effects due to stochastic factors were very important. In these particular conditions, the use of the deterministic model alone was not adequate for a good description of the infection. Consequently, improvements of the model were proposed in order to make it more realistic and to allow its use for the evaluation of the efficiency of field operations.

INTRODUCTION

Rinderpest is a non-persistent virus infection of cattle, mostly transmissible by direct contact. This infection still occurs in Africa, the Middle-East and India, and remains one of the most important diseases of bovine species. It should be accounted as a major economic problem [1].

The analysis of the spread of rinderpest is complex due to the interactions between three fundamental factors involved: host population, virus and environment. An intuitive evaluation of the spread of an epidemic or of the efficiency of a vaccination protocol is therefore not easy and a more accurate means of investigation, such as the construction of epidemiological models, seems essential for a better understanding of the dynamics of the infection.

Rinderpest virus belongs to the family of paramyxoviridae, genus Morbillivirus. Because measles virus belongs also to this genus and behaves in the same way as rinderpest in cattle, the work of Anderson and his co-workers on modelling of infectious diseases in general and on measles epidemics in the United Kingdom may be relevant [2, 3]. Some authors have emphasized the role of the computer for developing theoretical mathematical models [4] and recently a computer model for rinderpest was presented [5, 6].

The discrete-time model proposed here begins with a description of the main

characteristics of rinderpest in cattle. A mathematical formulation for the calculation of the probability of infection of a susceptible animal is then elaborated, starting from a simplified view of the phenomenon. A deterministic failure threshold of the infection is then deduced. Deterministic and stochastic computer simulations allow a practical representation of the spread of an infection in a population as a function of the characteristics of the virus strain, host animal and environment. Use of the model shows that, under particular conditions, variability in the progress of the infection can be so important that the deterministic approach alone is not adequate.

This model of rinderpest could be used for the evaluation of the efficiency of field operations or for the study of diseases epidemiologically similar to rinderpest.

DEVELOPMENT OF THE MODEL

Descriptive model

In some respects rinderpest conforms to a simple model. Although there are differences in the pathogenicity of virus strains, only one serotype exists and an efficient vaccine is available [1, 7]. Further, from knowledge of the epidemiology of rinderpest, the following assumptions can reasonably be made [1, 7–11] to elaborate a simplified model: transmission occurs only by direct contact; an alternative virus reservoir does not exist, neither does transmission by biological vectors; immunity is lifelong after vaccination or recovery from disease; there is neither asymptomatic carriage, nor recurrence of disease, and silent excretion if it occurs is extremely rare. These factors imply that epidemics which occur in previously rinderpest-free zones are caused by the introduction of living infected animals.

A simplified descriptive model, which summarizes the essential characteristics of the problem, is presented here. Natural mortality, i.e. depending on other causes than rinderpest, and the introduction of new animals by calving were not accounted for. The five possible states of the animals at time t, with respect to rinderpest, can be considered as follows: susceptible (S_t), infected but not yet infectious (L_t), infectious (I_t), immune (R_t) and dead (D_t). The relation between the different states is summarized in Fig. 1. Susceptible animals are those which belong to a susceptible breed and which are neither infected nor immune. The virus is practically non-resistant in the environment. Consequently, susceptible animals are normally infected via the oronasal route when they come into close contact with one or more infectious animals within the herd. After infection, animals do not excrete virus during a period referred to hereafter as the non-infectious period. At the end of the non-infectious period, the infectious period begins, during which animals excrete virus and may infect susceptible animals. Virus excretion may begin 1 or 2 days before the onset of the first clinical signs [12]. In this case, the infectious period begins before the end of the incubation period. At the end of the infectious period, animals become immune or die. Only susceptible animals can be protected from rinderpest by vaccination.

Differences exist in the pathogenicity of different strains of rinderpest virus as well as in the susceptibility of breeds of cattle [8]. The average lengths of the non-infectious and infectious periods and the death rate vary according to the

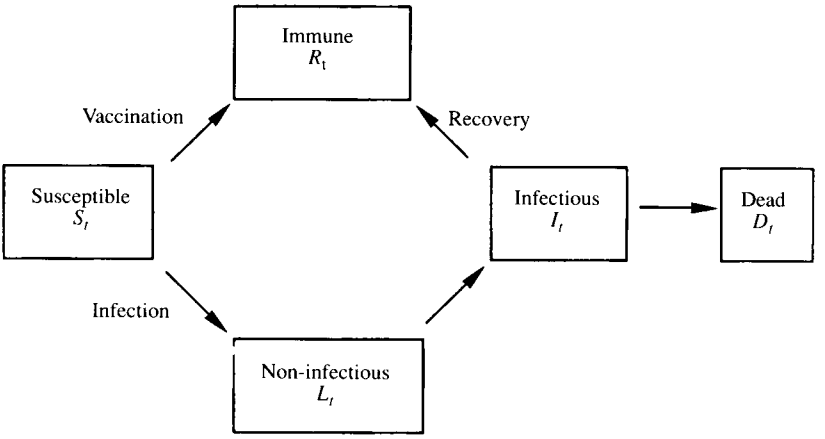

Fig. 1. Simplified descriptive model of rinderpest.

virulence of the virus strain and the resistance of the breed of cattle. Also, the lengths of non-infectious and infectious periods differ among individual animals. These factors affect the speed with which the disease spreads and the model must account for this. In addition, the model must be useful in conditions where the death rate is high and when the number of living individuals in the population varies during the course of the epidemic; it should also take account of the effect of vaccination programmes.

Mathematical model

The step for which a mathematical treatment is to be found is the initiation of infection. As soon as an animal is infected, it will then follow the normal course of the disease, owing to the lengths of non-infectious and infectious periods and the death rate.

Notations

ti = time of introduction of infected animals; ni = number of infected animals introduced into the herd; $Lmin$ = minimum length of the non-infectious period; $Lmax$ = maximum length of the non-infectious period; $Imin$ = minimum length of the infectious period; $Imax$ = maximum length of the infectious period; K = number of contacts an animal undergoes per unit of time; b = rinderpest-related death rate; a = probability of one contact between a susceptible and an infectious animal causing infection.

At time t: S_t = number of susceptible animals; L_t = total number of infected but non-infectious animals; $L(j)_t$ = number of animals ending their jth day of non-infectious status; I_t = total number of infectious animals; $I(j)_t$ = number of animals ending their jth day of infectious status; R_t = number of immune animals; D_t = number of dead animals; N_t = total number of living animals.

Spread of infection within a herd

Consider a herd composed of a mixture of susceptible and immune animals into which ni infected animals are introduced at time ti. Suppose that all the animals are randomly distributed.

An important parameter is the number of contacts (K) which an animal, whatever its state, makes with its neighbours during a unit of time. Consider a single susceptible animal taken at random amongst S_t susceptible animals in the herd at time t. During a given time, this susceptible animal will not necessarily have contact with all the other animals of the herd, but will contact K animals which are closest to it. K will be lower than N_t-1 for large herds or greater than N_t-1 for small herds. The value of K depends on herd characteristics (size, area occupied, age structure), breeding system and climate; this last will affect the density of animals in the vicinity of watering places in the dry season. We note that a model with K random has been developed by Lefèvre and Picard [13].

The contacts between animals are not necessarily contagious even when these contacts involve infectious animals because the required close contact may not always occur. For the present, it may be assumed that, on each contact with an infectious animal, a susceptible animal has a constant probability 'a' of becoming infected. In fact, the contact must be intimate enough so that the virus dose transmitted reaches at least the minimum infection dose. The probability 'a' depends on the virulence of the virus strain and on the resistance to disease of the breed of cattle. In what follows, the herd size will always be supposed to be large enough to replace N_t-1 by N_t. Also, K will be considered as a constant.

Basic formulae

At time t, a susceptible animal will meet an infectious one with the probability I_t/N_t. Thus, the probability that a susceptible animal, after K contacts, undergoes i contacts with infectious animals and $K-i$ contacts with non-infectious animals is given by the binomial distribution

$$\frac{K!}{(K-i)!\,i!}(I_t/N_t)^i(1-I_t/N_t)^{K-i}. \tag{1}$$

For this susceptible animal which has i contacts with infectious animals, the probability that one at least of these contacts will result in infection is equal to

$$1-(1-a)^i. \tag{2}$$

The product of equations (1) and (2) gives the probability $P(i)_t$ of a susceptible animal being infected after i contacts with infectious animals.

The probability of infection P_t of a susceptible animal being infected at time t, is given by the sum of $P(i)_t$, $i=0, \ldots, K$. It follows directly after simplification:

$$P_t = 1-(1-aI_t/N_t)^K. \tag{3}$$

The probability Q_t of a susceptible animal remaining uninfected at time t will be given by the complementary probability

$$Q_t = (1-aI_t/N_t)^K. \tag{4}$$

Approximate formulae

If it can be supposed that the quantity (aI_t/N_t) remains small enough, equation (3) can be simplified by keeping only the two first terms in the development of the Newton binomial. This leads to the following approximate expression:

$$P_t = aKI_t/N_t. \tag{5}$$

The approximate probability of a susceptible animal being infected depends on the product 'aK' and on the proportion of infectious animals in the herd. This term 'aK' may be understood as being the number of contacts intimate enough for the transmission of the disease, that an animal undergoes per unit time.

Stochastic model

Let $L(1)_{t+1}$ be the random variable which represents the number of susceptible animals that will be infected at time $t+1$, given S_t and P_t. The distribution of $L(1)_{t+1}$ is binomial with parameters

$$L(1)_{t+1} = BIN(S_t, P_t). \tag{6}$$

This means that the probability of j individuals becoming infected at time $t+1$, given S_t and P_t, can be computed by

$$P[L(1)_{t+1} = j] = \left[\frac{S_t!}{(S_t - j)!\,j!}\right] P_t^j (1-P_t)^{S_t - j}.$$

Deterministic model

The deterministic model is constructed on the basis of the mathematical expectation of the binomial distribution (eqn 6). The random variables S_t, L_t, I_t, R_t and N_t are replaced by the mathematical variables s_t, l_t, i_t, r_t and n_t. Let $l(1)_{t+1}$ be the mathematical variable which represents the average number of susceptible animals that will be infected at time $t+1$, given s_t and p_t. Clearly,

$$l(1)_{t+1} = s_t p_t = s_t[1-(1-ai_t/n_t)^K]. \tag{7}$$

For this deterministic model, some interesting properties can be derived rather easily:

(1) If the probability of infection p_t is replaced by its approximate expression (formula 5) in equation (7): then

$$l(1)_{t+1} = aKs_t i_t/n_t. \tag{8}$$

Equation (8) is similar to the continuous-time formulation [14, p. 304] which suggests that susceptible individuals at time t will produce new infected cases at the rate $\beta x_t y_t/n_t$ where x_t and y_t are the numbers of susceptible and infectious individuals at time t, respectively, n_t is the total population size at time t, and the parameter β represents the rate of infection.

In the particular case when the population size remains roughly constant during infection, the value of n_t can be included into the parameter β and the rate of production of new infected individuals becomes $\beta x_t y_t$, the formula used by Bailey [14] and referred to as a conventional hypothesis [2]. This relationship, viewed here as a particular case of equation (7), is valid providing that the quantity (ai_t/n_t) is small and the population size remains stable during the progress of the infection. Also, an interpretation can be given for the rate of infection β which can be split up into a 'population effect' represented by the parameter 'K' and a 'virus strain effect' characterized by 'a'.

(2) For a population of susceptible animals with a number of infectious individuals i_t introduced at time t, the ratio

$$y_{t+1} = l(1)_{t+1}/i_t = s_t p_t/i_t \tag{9}$$

can be considered as the average infecting capacity of an infectious animal at time $t+1$. As the value of s_t is $n_t - i_t$, equation (9) can be rewritten as

$$y_{t+1} = (n_t/i_t - 1)[1-(1-ai_t/n_t)^K]. \tag{10}$$

When the proportion i_t/n_t is increasing (or decreasing), the value of y_{t+1} decreases (or increases) slightly. If many infectious animals are introduced simultaneously into a disease-free herd, the infecting capacity of a particular infectious individual is lowered because several of the infectious animals may contact the same susceptible animals.

(3) If in equation (10), i_t/n_t is small enough, the corresponding limit value of y_{t+1} reduces to

$$y_{t+1} \simeq aK.$$

Thus, in a very large herd, the experimental determination of the average infecting capacity (i.e. the average number of animals which are infected at time $t+1$ per infectious animal introduced at time t in a susceptible population) can therefore be taken as an estimation for the product aK, the rate of infection.

(4) In order to study the conditions required to prevent the infection from persisting in the herd, let animals, just beginning their first day of infectious status, be introduced at time $t = 0$ into a disease-free population. As suggested by Anderson and May [2], a necessary criterion to prevent the disease becoming established is the non-renewal, at the end of the infectious period, of the number of infectious individuals initially present.

Consider the approximate expression (eqn 8), which is adequate in the beginning of the infection, and suppose that the infectious individuals initially present remain infectious during an average period of ip days, $ip = (Imin+Imax)/2$. Then, $aKs_{j-1}i_{j-1}/n_{j-1}$ new infected individuals will appear at time j, $1 \leqslant j \leqslant ip$.

Let us make the additional assumption that during these ip days, infection is not transmitted by the new infected animals to secondary cases, which means that $ip \leqslant Lmin+1$. This implies that the number of infectious individuals is constant during these ip days.

Now, a rate of infection, g, can be defined as the average number of new infected animals per infectious animal during the average length of its infectious period. Under the conditions above, g becomes

$$g = aK \sum_{j=1}^{ip} (s_{j-1}/n_{j-1}).$$

So, if $g < 1$, the renewal of infectious individuals by newly infected individuals cannot take place. In this case and denoting by s_m/n_m the arithmetic mean of s_{j-1}/n_{j-1}, then

$$s_m/n_m < 1/(aKip). \tag{11}$$

Moreover, if s_m/n_m can be approximated by s_o/n_o, equation (11) becomes

$$s_o/n_o < 1/(aKip). \tag{12}$$

This approximation is valid when the infection starts, provided that the virus strain is not too virulent.

Observe here that s_o is equal to the initial population size n_o minus the number of immune animals initially present r_o. Thus, equation (12) is equivalent to

$$r_o/n_o > 1-1/(aKip). \tag{13}$$

Equation (13), defined here as the deterministic 'failure threshold density', shows how to compute, from the initial conditions, the minimum proportion of individuals that must be vaccinated in order to prevent the renewal, after the average length of the infectious period, of the infectious individuals initially present.

Computer program

A computer program is used to simulate the spread of infection in host populations, after entering in the following parameters: initial size of herd, time of introduction and number of infected animals introduced, initial number of immune animals, minimum and maximum lengths of both non-infectious and infectious periods, number of contacts an animal makes per unit of time, death rate of rinderpest and probability of one contact between a susceptible and an infectious animal causing infection.

For a given virus strain, it is supposed that non-infectious and infectious periods vary according to the individuals and are distributed between two limits (minimum and maximum) with constant probability functions. Algorithms used for the state transitions are different depending on the considered version.

Deterministic version

The deterministic model makes use of the following relationships:

$$s_{t+1} = s_t - l(1)_{t+1} \quad \text{and} \quad l(1)_{t+1} = s_t p_t.$$

If $Lmin \leqslant j \leqslant Lmax$, infected individuals ending their jth day of non-infectious status may still remain in the same status during a maximum of $Lmax - j$ days. Therefore, we suppose that a proportion of $1/(Lmax+1-j)$ of these individuals will become infectious, so that:

$$\begin{aligned} l(j+1)_{t+1} &= l(j)_t \quad \text{if} \quad 1 \leqslant j < Lmin \\ &= l(j)_t[1 - 1/(Lmax+1-j)] \quad \text{if} \quad Lmin \leqslant j \leqslant Lmax \\ i(1)_{t+1} &= \sum_{j=Lmin}^{Lmax} l(j)_t \, 1/(Lmax+1-j). \end{aligned}$$

Similarly for infectious animals

$$\begin{aligned} i(j+1)_{t+1} &= i(j)_t \quad \text{if} \quad 1 \leqslant j < Imin \\ &= i(j)_t[1 - 1/(Imax+1-j)] \quad \text{if} \quad Imin \leqslant j \leqslant Imax \\ r_{t+1} &= \sum_{j=Imin}^{Imax} i(j)_t(1-b)/(Imax+1-j) \\ d_{t+1} &= \sum_{j=Imin}^{Imax} i(j)_t \, b/(Imax+1-j). \end{aligned}$$

Stochastic version

The binomial distribution (eqn 6) of the random variable representing the number of susceptible animals that will be infected at time $t+1$ can be simulated in the following way. For each susceptible animal present at time t, a random

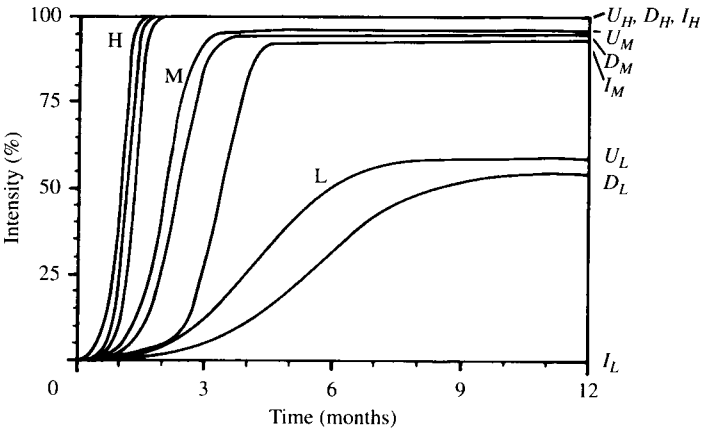

Fig. 2. Infection of a disease-free cattle population by three virus strains of different virulence (H: high, M: moderate, L: low). Characteristics of the virus strains are presented in Table 1. No vaccination has taken place. Characteristics of the population are: $N_o = 1000$, $ti = 0$, $ni = 2$, $K = 20$, $R_o = 0$. U, I: upper and inferior limits of the zone where 90% of the stochastic observations are taking place; D: deterministic simulation.

number is selected with value in the range 0 to 1 and its value is compared to the probability of infection P_t. If this random number is lower or equal to P_t, the susceptible animal becomes infected; otherwise the animal remains susceptible. The total number of infected animals at time $t+1$ is obtained by the summation of all these state transitions.

Consider now an infected animal ending its jth day of non-infectious status. If $j < Lmin$, the animal remains non-infectious. Otherwise, for each non-infectious animal, a random number is selected with value in the range 0 to 1, and if this number is lower than $1/(Lmax+1-j)$, the animal becomes infectious.

In the same way, for an animal ending its jth day of infectious status, if $j < Imin$, this animal remains infectious. Otherwise, for each infectious animal, a random number is selected with value in the range 0 to 1. If this number is lower than $1/(Imax+1-j)$, this animal undergoes a state transition; it dies if another random number is lower or equal to the death rate or becomes immune in the opposite case.

Use of the model

For illustration, and in order to emphasize the effect of variability in the virulence of virus strains and the consequence of vaccination programmes, two typical cases were selected, results of which are presented in Figs. 2 and 3. These examples allow illustration of to what extent the spread of infection can be properly represented by the deterministic model.

Intensity of the epidemic at time $t(INT_t)$, can be defined, in the sense of Bailey [14], as being the proportion of susceptible animals initially present which have contracted the disease at time t.

$$INT_t = 100(S_o - S_t)/S_o. \tag{14}$$

In a herd containing 1000 animals, 2 infectious animals ending their second day

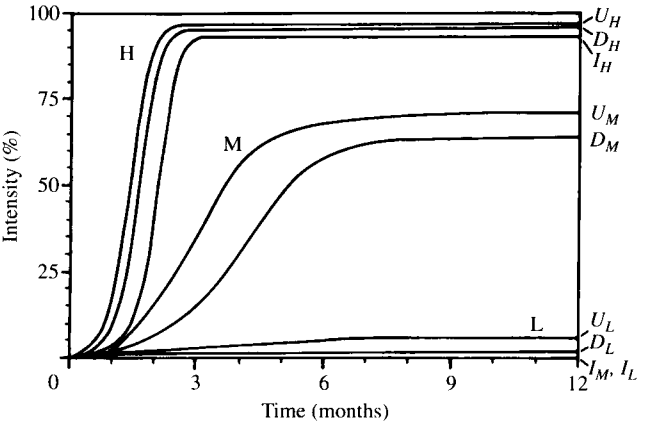

Fig. 3. Same conditions as in Fig. 2 except that vaccination has taken place, 40% of animals being immune when the epidemic starts ($R_0 = 400$).

Table 1. *Characteristics of virus strains used to prepare models of rinderpest illustrated in Figs. 2 and 3*

	Virulence		
	High (H)	Moderate (M)	Low (L)
Lmin, *Lmax* (days)*	3, 8	5, 10	6, 12
Imin, *Imax* (days)†	4, 6	5, 7	6, 8
Rinderpest-related death rate	0·9	0·5	0·1
Probability of one contact causing infection‡	0·04	0·02	0·01
Failure threshold density (%)§	75·0	58·3	28·6

* *Lmin*, *Lmax*: minimum and maximum length of the non-infectious period.
† *Imin*, *Imax*: minimum and maximum length of the infectious period.
‡ Probability of one contact between a susceptible and an infectious animal causing infection.
§ Failure threshold density (%) (eqn 13): minimum proportion of individuals that must be vaccinated in order to prevent the infection from persisting in the herd.

of non-infectious status are introduced. The number of contacts an animal undergoes per day is constant and equal to 20. Figs. 2 and 3 represent the evolution, during 1 year, of the above defined intensity in the herd, for three virus strains of different virulence. Characteristics of these virus strains are given in Table 1. These parameters have been chosen from published data on the epidemiology of rinderpest [12, 15–17]. When the virulence of the virus strain increases, it was supposed that the lengths of non-infectious and infectious periods decrease, because disease progresses more quickly. In Fig. 2, no previous vaccination has taken place and in Fig. 3, 40% of the animals are immune by vaccination when the epidemic starts.

Simulations of infection were achieved on an IBM PS/2 computer. After introduction of the characteristics of the virus strain and the cattle population, the program computes a first deterministic simulation giving the average herd composition as a function of time. One hundred stochastic simulations then follow.

For each virus strain, the deterministic curve is surrounded by two curves which define a zone where 90% of the stochastic observations take place. These two curves display the variability existing in the stochastic process.

Fig. 2 shows how a decrease in virulence of the virus strain is accompanied by a decrease in intensity of the epidemic, but that virus persists longer in the population and the variability of the process increases considerably. For the highly virulent strain, all susceptible animals are infected during the first 2 months. Deterministic simulation gives a satisfactory description of such an infection process. The moderately virulent strain remains for more than 4 months in the herd, reaching an average final intensity of 95%. Variability of stochastic simulations increases slightly but the deterministic simulation remains sufficiently valuable to represent the global process. Strains of low virulence can subsist in the population for 1 year. The hundred stochastic simulations cover a large field and final intensities of the infections vary between 0 and 60%. In these conditions, the use of the deterministic model alone is not adequate for a good description of the infection.

Fig. 3 shows some effects of vaccination. With the virus strain of moderate virulence, the epidemic variability increases strongly compared to the case with no vaccination. In the case of the strain of low virulence, the percentage of immune animals in the herd ($R_0/N_0 = 40\%$) is greater than the failure threshold density (eqn 13) which specifies that at least 29% of the population must be immune when the epidemic starts in order to prevent the infection persisting in the herd (Table 1). Nevertheless, some infected individuals appear because reaching the deterministic failure threshold density would only prevent the renewal of infectious individuals initially present but does not give any information about the average number of individuals that will be infected at the end of the epidemic.

A decrease in the virulence of the virus strain or an increase in the number of immune animals initially present in the herd has a similar effect; it becomes more difficult to forecast the spread of the infection because the variability in the progress of the infection increases but also to diagnose the disease in the herd because prevalence and mortality decrease.

DISCUSSION

To be realistic an epidemiological model of rinderpest has to take into account important and variable non-infectious and infectious periods as well as an occasionally high death rate. Because of the complexity of the corresponding equations, iterative methods with simulation on a computer appeared to be the most adequate. A preliminary but essential step in the construction of the model was the description, involving some simplifying assumptions, of the main characteristics of the spread process of rinderpest and the derivation of an equation for the calculation of the probability of infection of a susceptible animal. The main originality was the use of two parameters defining independently the characteristics of the population and the characteristics of the virus strain. The equation established here can be simplified, in certain particular conditions, to give the form commonly used in the papers devoted to epidemiology [2, 14].

The opportunity for joint use of both deterministic and stochastic approaches

allows a comparison between the two procedures. The deterministic method provides an average display of the infection process by means of quite simple relationships; these may also be used to compute a failure threshold density for epidemic spread. The stochastic method, more complex from a computational standpoint, gives more realistic results which take into account random effects which are naturally present in biological processes. These random effects are important at the beginning of the infection, because of the low disease prevalence in the population, and mostly when few infectious animals are introduced in the herd, a large number of animals are vaccinated, contacts between them are reduced and the virulence of the virus strain is lowered. Indeed, when the expected number of infected animals is small, random effects will be dominant to determine if the disease persists or not.

The stochastic approach shows that the great variability which may appear in the process will make a forecast for the development of the infection difficult. The study of the spread of the disease by reference to the deterministic approach only is consequently insufficient. Because of this limitation of the deterministic results, further developments remain to be made for the derivation of an accurate stochastic threshold. These would quantify the problem with probabilities to take into account the random character of the process, i.e. of the type: 'there is a level of 90% probability that the infection will be limited to less than 2% of susceptible animals of the herd'.

In order to perform simulations over a time period of several years, it would be necessary to take into account the natural and neonatal mortalities as well as calving. Other improvements of the model would require research in cattle ethology and climatic conditions. Further, it would be necessary to consider a possible reduction in the number of contacts occurring between infected animals and the remainder of the herd, and to consider several subsets of the population in order to study the effect of cattle gathering around watering-places in dry seasons. Parameter 'K', here taken as constant, could vary according to some probability laws or could depend on a periodical term, function of the breeding system and local climate conditions, a maximum number of contacts being observed during the watering of animals, mainly during the dry season. Furthermore, adequate field experiments need to be done in order to obtain a correct evaluation of the basic parameters used in the model.

This model should then be validated to ensure that it adequately fits real infection data. Different levels of complexity might be tested to choose the more adequate in the different conditions (epidemic or endemic situations). Indeed, if a simple model does provide an adequate fit to some epidemic data, it better summarizes the important characteristics of the disease and is more likely to yield to mathematical analysis.

A final purpose of the present study would be the derivation of necessary conditions for the spread of rinderpest virus in a population. A realistic model would allow the evaluation, from the standpoint of relative costs, of the efficiency of different control methods (vaccination programmes and sanitary control, such as lowering in number of contacts between animals). The model would therefore provide an objective tool for the choice of a strategy in control operations. Moreover, the epidemiological model described here for rinderpest could further be

used to study other diseases similar to rinderpest from an epidemiological point of view.

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