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POPULATION DYNAMICS AND EPIDEMIOLOGY OF TERRITORIAL ANIMALS

(JOINT MEETING OF QUANTITATIVE EPIDEMIC MODELLING GROUP AND MATHEMATICAL ECOLOGY GROUP)

edited by

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(* = abstract only)

INTRODUCTION

Denis Mollison

Between 1978 and 1984 meetings on various aspects of the spread of fox rabies were organised by a working group set up jointly by the Institute of Terrestrial Ecology and the Royal Statistical Society (e.g. Bacon & Armitage 1979, Bacon & Macdonald 1981, Ball & Bacon 1984). These meetings were felt to be valuable, particularly in the way they brought theoreticians together with practical ecologists and epidemiologists, and it was therefore agreed that the working party should be continued as an informal 'Quantitative Epidemic Modelling Group'.

The aim of the present meeting was to widen the scope of the working party's discussions by comparing the population dynamics of a number of other territorial animals, under the influence of a variety of diseases. The juxtaposition of papers by ecologists and epidemiologists, practical and theoretical workers, provoked many useful discussions, and will I hope have a similar stimulating effect on the reader.

The meeting was organised by Philip Bacon and myself (mostly Philip), with support from the British Ecological Society and Biometric Society for which we are grateful. I should also like to thank all those who attended, whether as speakers or simply as participants in the discussions; John Jeffers, Director of ITE, the original proposer of the working group in 1977, and our host on this occasion; and Philip Bacon and supporting staff at ITE Merlewood for all their hard work which made possible a smoothly run and enjoyable meeting. Our thanks also go to Winnie Hughes for retyping the papers on my Department's word processor at Heriot-Watt.

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"WILDLIFE RABIES IN EUROPE AND THE BRITISH SITUATION"

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There is a large volume of scientific and documentary literature relating to rabies in domestic and wild animals in almost all parts of the world. There is also much field experience of rabies control in domestic and wild mammals and of rabies prevention in man, but the disease persists, is difficult to eradicate and is endemic in many parts of the world. Two fundamental control problems remain, broadly of persistent canine rabies in under-developed countries and of sylvatic rabies in developed countries.

Dog rabies is the major problem since dogs are responsible for over 90% of human exposures to the disease, whether sylvatic rabies is present or not. In Europe, where sylvatic rabies predominates the ratio of wild to domestic rabies is 77:23, whereas in the Indian subcontinent it is 1:99. In Europe, where human deaths from the disease are rare, rabies is only of minatory importance to man whereas in India, in some teaching hospitals, one in every five hundred to two thousand admissions are rabies cases and twenty thousand deaths annually is estimated.

Our primary objective in Britain is to keep rabies out by strict import controls and by quarantine of imported mammals. An outbreak of rabies in domestic animals would cause considerable concern but less so than confirmation of the disease in wildlife. Domestic animals can be restrained and, if necessary, vaccinated to prevent the disease spreading, but free ranging, unrestrained wild animals present a different and more difficult problem of rabies control and it is this aspect of the epidemiology of rabies that is of interest to this group.

In affected areas of Europe the fox is the main reservoir and vector of rabies. With minor exceptions, despite attempts to control the disease in foxes, rabies in wildlife is out of control.

For the disease to persist a density of foxes above the epidemiological threshold is required, commonly stated to be about one fox per square km. To achieve lasting control it is estimated variously, depending upon fox population density, that a reduction of 60-90% of the "standing crop" of foxes would be required, annually, until the disease expired. This would require coordinated, concerted, highly organised and effective action, internationally. In view of the large area involved, of the ineffectiveness of traditional methods of control involving the removal of potential vectors, and of problems of measuring the degree of reduction of potential vectors achieved, it is not surprising that the prevalence of rabies is high in foxes in all rabies endemic areas of Europe.

Oral vaccination of foxes using a live vaccine preparation offered in baits is being used experimentally in Switzerland, part of West Germany and Italy to prevent rabies spread and to protect rabies-free zones. This technique, locally successful, has not yet reached the stage of an official policy for fox rabies control, but if it were adopted generally in Europe the logistics of its use would be formidable though minor by comparison with attempts to control the numbers of foxes.

If rabies were to occur in Britain the control objectives would have a different accent from those in rabies endemic areas of Europe. Wherever rabies occurs the primary objective of rabies control is to protect man, but in Europe because rabies

is prevalent in wildlife the practical aim is to prevent infection occurring in domestic animals. In Britain, where the same aim would obtain, an additional confrontation would be the prevention of rabies spreading to wild mammals. If the disease were confirmed in foxes in Britain the elimination of the virus by a rigorous reduction of fox numbers over defined areas would become an urgent necessity. Those concerned with the modelling of the epidemiology of wildlife rabies have addressed themselves to the probable course of events following a single focus infection, and whilst adequate data on many aspects of the biology of foxes are available one fundamental parameter is poorly understood, that is, the probability of a rabid fox transmitting the disease to another.

To provide a predictive model the "effective contact rate" would need to be known for a range of circumstances, such as, at different population densities, according to sex and age, at different times of the year, according to the behaviour of healthy foxes confronted by rabid foxes, and vice-versa, among itinerant and dispersing foxes and within and between family groups; knowledge of the movement behaviour of rabid foxes is poorly understood and the probability of transmission from adult foxes suffering dumb rabies to dependent or other offspring, by aerogenic infection perhaps in confined spaces, is not known. There are many examples in Europe of a confirmed rabid fox being found 40 to 60 km ahead of the nearest known case in the advancing rabies front. No cases are known where such a focus of rabies has proliferated independently of the main body of the disease and the evidence suggests strongly that the successful transmission of rabies from individual foxes is not highly assured. The behaviour of foxes while in the rabid phase must clearly be adapted to ensure an optimum probability of spread and survival of the virus in a totally susceptible species. If it were otherwise, the disease could, on the one hand, die out because its transmission was inefficient or on the other hand burn out because it was too effective. Certainly the dumb form of rabies which reduces the probability of further transmission compared with the furious form (the dumb form is variously estimated to occur in 50 to 70% of rabies affected foxes) would be one means of effecting a reduction in the effective contact rate in an endemic situation, but there could well be other less obvious aspects of behaviour of rabid foxes which achieve the same end without impairing the survival of the virus.

The recently developed monoclonal antibody technique enables different strains and variants of rabies virus to be identified and it is now established that the vulpine strain predominates in Europe, with the canine strain also occurring (in dogs and wolves) in Yugoslavia and Turkey. Interestingly, neither dogs nor cats are highly susceptible to the fox strain of virus nor is the quantity of virus present in the saliva as great in these species as when infected by the canine strain. Similar information for foxes relevant to the canine strain of virus is not at present available. In areas of Europe, outside Yugoslavia and Turkey, dog rabies is usually attributed to infection from foxes.

If a dog, illicitly or accidentally introduced into the UK, from France for example, were to develop rabies it would probably be of fox origin and the probability of subsequent transmission could be less than if the dog were infected by the canine virus strain because its bite would be less virulent. Of interest in this context is the provenance of the strain of rabies virus affecting foxes in North America and whether or not it has similar epidemiological characteristics to the vulpine strain in Europe. Simulation models, such as the Ontario model, based upon the characteristics of the disease in one country may not be entirely valid when applied to another area if the causative organisms are geographically isolated and epidemiologically different.

Contingency plans for wildlife eradication in Britain are based upon the removal of as high a proportion as possible of foxes, by whatever means most appropriate to

the location and circumstances. This would involve killing foxes. Oral vaccination of foxes using a live virus preparation would, in Britain, be inconsistent with the aim of eliminating rabies virus, but a dead vaccine might be acceptable and such a development could add a potent weapon to our anti-rabies armoury.

As things are now, anti-rabies action would need to be undertaken and completed swiftly, and though the task would be daunting the example of successful rabies control in Denmark, where an effective but inefficient method (gassing of dens) was used, suggests that our plans to use both effective and efficient methods, mainly involving the use of poison, would prevail.

DISPERSAL OF URBAN FOXES

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This paper describes the patterns of dispersal seen in foxes in Bristol, and the construction of a model of urban fox dispersal.

Between 1977 and 1984, 1098 foxes have been captured, tagged and released in Bristol, with 504 re-captures or recoveries from 419 individual foxes. Sex, age, locality and date of capture were recorded for each animal. Additionally 1240 foxes between 1977 and 1983 have been subject to a post-mortem examination to provide information on productivity and mortality. Both sets of data will be updated as more information becomes available.

Two assumptions were made to simplify the analysis. Firstly, a fox dispersed if it moved 700 m. or more from its point of first capture; this distance is derived from a consideration of the mean home range size of Bristol foxes. Secondly, all foxes first captured up to 26 weeks after the first week in April (taken as the typical date of birth) were considered still to be on their natal home range, and had not yet dispersed.

Of foxes captured up to 26 weeks, 55% of males, and 29% of females, dispersed. This difference is significant, whereas there is no significant difference between the proportion of dispersing male and female foxes first captured when more than 26 weeks old. Dispersal occurs principally during the late autumn and winter of their first year. The mean recovery distance for male and female cubs was also significantly different; males moved on average 1.8 ± 0.4 km. (n=160), females 0.7 ± 0.2 km. (n=151). There was no significant difference for older foxes. Although the maximum recovery distance was 18km. for a male, 58% of male and 81% of female recovery distances were 1 km. or less. There was no significant difference in the average age at recovery for dispersed and non-dispersed foxes. Compared with other studies, the foxes in Bristol moved less far and fewer dispersed. This fits in with a relationship that has been observed between dispersal distance and home range size/density.

There was a positive correlation between the distance a fox moves, and the fox family group density at the point of recapture. However no relationship existed between distance moved and original capture density. The angle of dispersal from the point of first capture was examined to see if there was a trend to move in or out of the city. For both sexes, a Rayleigh test showed that the angle of dispersal was at random. Foxes living near the city centre were less likely to disperse, although this did not affect the range of distances they moved. The city/rural area interface did not act as a boundary to dispersal. There was no significant effect of fox size, litter size, or date of birth on the pattern of dispersal. Radio-tracking studies have shown that the foxes in Bristol exhibit a variety of patterns of dispersal movements.

The mean litter size (excluding barren vixens) was 4.7 ± 0.2 (n=182) at birth, but down to 4.2 ± 0.3 (n=54) for 4-8 week old cubs. From this the mortality during the first two months was calculated to be 12%. From the estimates of the age at death of the post-mortemed sample, survivorship curves were plotted, and mortality rates estimated. On average 61% of foxes died each year. During their first

year of life, 59% of females and 64% of males died. Both litter size and mortality rates are in close agreement with values given in the literature.

A stochastic simulation model is envisaged, based on information obtained from the above analysis, to describe the temporal and spatial aspects of fox dispersal in Bristol. The construction of the model is at a preliminary stage. The spatial aspects will be dealt with by subdividing the city and surrounding area into a regular grid of 500 by 500 m. cells. The same grid was used for fox family group density estimates, so in later forms of the model a measure of habitat desirability could be included in each cell. Observed fox densities will be used to reflect habitat desirability. A continuous pattern of mortality and a seasonal pattern of dispersal are the two main processes in the model. As the majority of fox dispersal occurs in juveniles, the initial simulation will be over a period of one year. The mortality rate will be constant, and estimated from the survivorship curves. Dispersal will occur only in the latter half of the simulation. The probability of individual foxes dispersing will follow the pattern observed in the Bristol foxes. The angle of dispersal will be chosen at random; the distances moved will be described by an exponential decay curve, as this fits well the observed distribution of dispersal distances previously analysed. The simulation will be started with 211 fox family groups (the mean annual density found in Bristol) distributed over the grid, with 4.7 cubs per site on average on a 1:1 sex ratio.

The study on dispersal in Bristol shows that fox density is one of the few factors significantly affecting dispersal. In a simple model of fox dispersal more complex features (animal size, or date of birth for instance) need not be included, as these have little effect on dispersal. Given that productivity and patterns of mortality appear similar in other cities, a model of fox dispersal based on Bristol data should be applicable to other urban areas, if information is available on the fox densities in those areas. A multiple regression model has been devised that generates such estimates of fox density from an analysis of the distribution of habitat types found within an urban area. It should therefore be possible to devise a model of urban fox dispersal that can be applied to most British urban areas.

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"ESTIMATION OF THE DISPERSION DISTANCE OF FOX-CUBS AND THE INCUBATION PERIOD OF FOX RABIES THROUGH A BIOMATHEMATICAL MODEL"

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Two important epidemiological parameters of fox rabies - the dispersion distance of cubs and the incubation period - are difficult to measure in the field. Therefore, we have derived a deterministic compartmental mathematical model which simulates the space-time evolution of the disease and makes it possible to estimate the mean value of those parameters using a Monte-Carlo method.

Three output variables of the model - the rate of survivors of the disease front wave, the speed of the disease displacement and the distance between the first and the second wave - were studied as functions of the two parameters of interest which we use as input variables. One hundred simulations were carried out using FORTRAN on a CDC CYBER 750 computer with representative combinations of those input variables. Estimates of the value of the dispersion distance of fox-cubs and the incubation period of fox rabies in the field were obtained by the computation of the distribution of the input variable values yielding output variables consistent with the data observed in Europe. It is found that the actual European situation of fox rabies is linked to a dispersion distance of fox-cubs less than 8 kilometers and an incubation period greater than 25 days.

"SURVEY OF RSS/ITE GROUP'S WORK ON FOX RABIES"

Frank Ball
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In 1977 the Royal Statistical Society set up a working party on quantitative studies in the epidemiology of fox rabies. This talk will commence with a brief history of the working party, followed by a brief overview of two models of fox rabies.

The first model is an empirical model developed by Sayers, see e.g. Sayers et al. (1985), to analyse the pattern of reported Rabies cases in Europe. After appropriate smoothing of the data, foci of infection are identified and their trajectories can be related to geographical features.

The second model is a "Monte-Carlo" spatial simulation model of fox rabies in Ontario developed by a group of Canadian workers, see Voigt et al (1985). This is a complex model incorporating a high degree of biological realism.

A third model will then be presented in somewhat more detail. This model, see Ball (1985), is a nearest neighbour spatial epidemic model appropriate for the control of a possible fox rabies outbreak in Britain. Emphasis will be placed on the underlying modelling philosophy which led to the particular form of this model rather than on the mathematical details.

The talk will finish with a short discussion of the biological unknowns most urgently required by mechanistic modellers of fox rabies.

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EPIDEMIC MODELS: STRUCTURE AND SENSITIVITY

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For ecological processes such as epidemics, a wide variety of types of model have been proposed, for instance stochastic branching, percolation and diffusion processes, as well as deterministic differential and difference equation models. While the most interesting theoretical advances of recent years have been on simple spatial stochastic processes, most applied workers still rely on nonspatial models, or on over-complex simulation models.

I shall discuss the relations between various simplex epidemic models, with particular reference to the ways in which they include basic epidemiological components and how this affects their qualitative behaviour.

Some aspects of model behaviour, such as the level of prevalence and period of oscillation, appear to be robust; while others, unfortunately including crucial questions of control, are very sensitive to the detailed form of the model components.

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A SIMPLE MODEL FOR THE SPATIAL SPREAD AND CONTROL OF RABIES

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I. Abstract

A very simple partial differential equation model for the spatial spread of rabies is motivated and briefly analyzed. It models the dynamics of the front of an epizootic wave. The model is used to estimate the minimum width (in kilometers) of a break, that is, a region in which a control scheme is employed in order to stop the spatial progression of the rabies wave front. A prediction for the spatial spread of an epizootic in Britain is also obtained. The simple model may be extended to model the endemic state as well as the front of the epizootic. Results of an analysis of the extended model are described.

II. The Model

In this summary we discuss a very primitive model based on the assumption that the migration of rabid foxes determines the dynamics of the epizootic front. A full account of the model and a simple extension modelling endemic behaviour is in Kallen, Arcuri and Murray (1985). The reason we present such a simple model for such a complex problem is that is poses relevant questions that more realistic models must also address. There is, of course, in any modelling a trade-off between complexity and the difficulty of estimating many parameters and a simpler one where values can be reasonably assessed. In this paper, we have opted for the latter strategy.

We use a deterministic approach and divide the fox population into two groups, infective and susceptible foxes; the former consists of rabid foxes and those in the incubation stage. The basic facts (see, for example, the books by Macdonald (1980) and Kaplan et. al. (1977)) and assumptions of our model are:

(i) The rabies virus is contained in the saliva of the rabid fox and is normally transmitted by bite. Therefore a contact between a rabid and a susceptible fox is necessary for the transmission of the disease.

(ii) Rabies is invariably fatal in foxes.

(iii) Foxes are territorial and seem to divide the countryside into non-overlapping home ranges which are marked out by scent.

(iv) The rabies virus enters the central nervous system and induces behavioural changes in its host. If the spinal cord is involved it often takes the form of paralysis. However, if it enters the limbic system the foxes become aggressive, lose their sense of direction and territorial behaviour, and wander about in a more or less random way.

In the model, the change with time of the number of infective foxes within a small area (like a territory) is equal to the rate of transitions from the susceptible population minus the mortality rate and the migration from the area. In the short time interval we are interested in (~ 1 year), the temporal variation in the

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susceptible population is simply the rate of loss to the infective population. Assuming that the dispersal of the infective population can be reasonably approximated by random walks (from assumption (iv)) our model equations are

$$\partial S/\partial t = -KIS$$

$$\partial I/\partial t = D\partial^2 I/\partial x^2 + KIS - \mu I \qquad (1)$$

Here S and I are the susceptible and infective population densities respectively and for simplicity only we consider the one-dimensional (x) spatial problem. Thus, our model is very similar to one by Noble (1974) for the spread of the Black Death in the 14th Century. The term KIS comes from (i) KS is the number of new infectives produced per unit time per infective and the constant K is the transmission coefficient. Note that only a fraction of the infective foxes, that is the rabid ones, can transmit the disease. The term $-\mu I$ comes from (ii) with the additional assumption that the probability for an infective fox to survive for at least time T is given by $e^{-\mu T}$, so that $1/\mu$ is the life expectancy of an infective fox. The absence of a migration term in the equation for the susceptibles is motivated by (iii): susceptible foxes do occasionally travel considerable distances but they always return home (with the exception of young foxes leaving their home territory in search of territories of their own, an event briefly considered below). The diffusion term comes from (iv) and represents the random motion of rabid foxes averaged out over the whole infective population: the diffusion coefficient D can be estimated by

$$D = kA \qquad (2)$$

where k is the rate at which infective foxes leave their territories which have average area A, that is, 1/k is the average time until a rabid fox leaves its territory. More accurate estimates of D would be obtained by field observations of net distances travelled by infectives during observation periods.

Several other assumptions in the model are important. A static population of susceptibles is assumed in the sense that deaths are equally balanced by births. This is unrealistic on a long timescale but for the short period of about a year, with which the model is concerned, is perhaps not unrealistic. We are primarily interested in the rate of propagation of the epizootic. We show below that this depends on the susceptible density ahead of the outbreak, where deaths and births balance since the fox population is assumed to be in equilibrium with the carrying capacity of its environment. The extension of the model to include susceptible births is briefly considered in Section IV. We also neglect all routes of infection other than binary contacts between rabid and susceptible foxes.

In leaving out a migration term in the equation for susceptibles we are neglecting the fact that male foxes may leave their territory in the rutting season looking for a mate, and that juvenile foxes leave their home territory in the autumn travelling distances that typically may be 10 times a territory size in search of a new territory. If a fox happened to have contracted rabies around the time of such long-distance movement, it could certainly increase the rate of spreading of the disease into uninfected areas. However, such events represent a very small fraction of the overall life history of the fox, implying the effective rate of diffusion of susceptibles is very small compared to the dynamics incorporated in the simple model equations (1).

We also assume that space is completely homogeneous so that there are no preferred directions to move in. Some of these effects could easily be incorporated in the

model. They have been excluded so as to isolate with simplicity what we believe to be some key features.

The parameters in (1) may be rescaled to give the nondimensional model equations

$$\partial u/\partial t = \partial^2 u/\partial x^2 + u(v - r)$$
$$\partial v/\partial t = -uv \qquad (3)$$

where S_0 is the carrying capacity of the environment, $u = I/S_0 \geq 0$, $0 < v = S/S_0 \leq 1$, and $r = \mu/KS_0$. Note that the qualitative behaviour of the system depends on only one dimensionless parameter, r. Its inverse, $R = 1/r$, is equivalent to the basic reproduction rate of the disease in the initial stages of an outbreak of the disease. Hence, $r < 1$ must obtain for rabies to persist. Thus, given K and μ, there is a typical critical minimum fox density S_c, below which rabies cannot persist, given by

$$S_c = \mu/K = rS_0 \qquad (4)$$

This threshold result is common to a large class of epidemiological models which use multiplicative terms to represent interactions between populations.

An elementary mathematical analysis of the model equations (1) shows that if $r < 1$ and the initial distribution of susceptibles is uniformly equal to 1 (that is, $S = S_0$ everywhere), then in the one-dimensional case a small localized introduction of rabies evolves into two travelling waves going in opposite directions. Figure 1 gives the shape of the wave moving to the right for $r = 0.5$. It is also found that the proportion a of susceptible foxes which remain after the infective wave has passed satisfies $0 < a < r < 1$, and is given in terms of r by

$$a - r\log a = 1 \qquad (5)$$

Figure 2 shows this relationship between a and r.

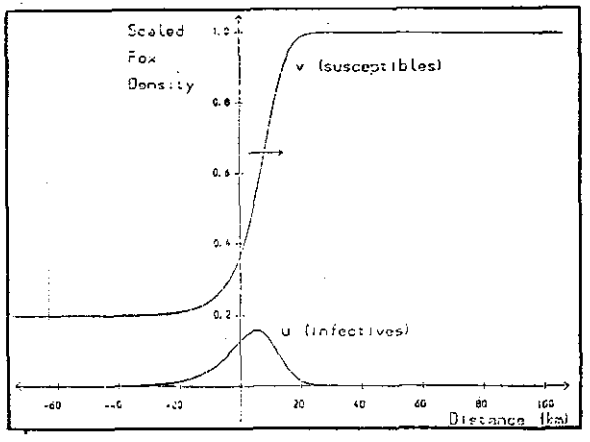

FIG. 1

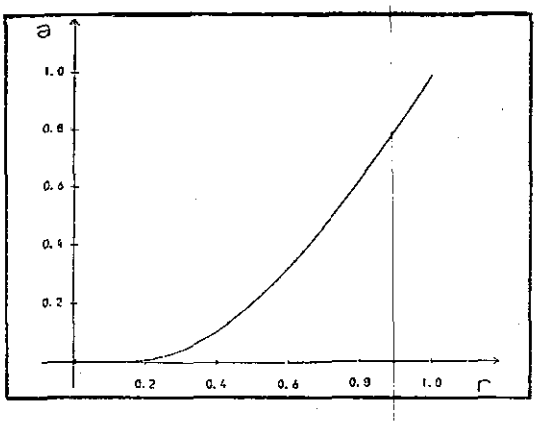

FIG. 2

The waveforms for the two types travel together at the same speed c, which is given by

$$c = 2\sqrt{2 - r} \qquad (6a)$$

or in dimensional terms, by

$$c = 2[D(KS_0 - \mu)]^{1/2} = 2[D\mu(1/r - 1)]^{1/2} \qquad (6b)$$

One way of using our model is to make tentative predictions as to how an epizootic might spread if introduced into a region where the initial distribution of (susceptible) foxes is known: a knowledge of this could prove helpful in combating the epizootic. Taking estimates for the initial distribution of susceptible foxes from McDonald (1980), together with the speed of propagation given by (6) and the results from (fairly crude) computer simulations as a guide, we have drawn by hand the map shown in Figure 3 to illustrate how a small population of infective foxes introduced around Southampton might spread the disease throughout Britain.

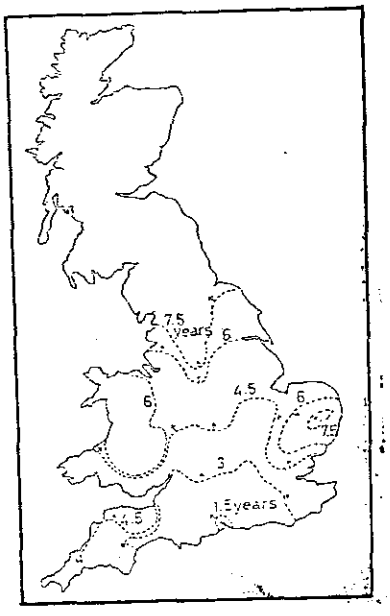

FIG. 3

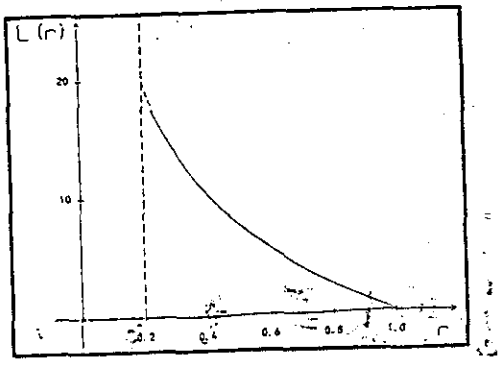

FIG. 4

III. Control Measures

Considering the problem of introducing control measures in a protective break in order to stop the epizootic wave from entering a rabies-free region, we consider the one-dimensional problem and let $0 \leq x \leq L$ be a protective barrier between a rabies free region $x > L$ and an infected region $x < 0$. We may then numerically determine the minimum width L which keeps $x > L$ free of rabies. For a simple (illustrative) control scheme where initially we have a uniform fox density everywhere and that the fox density is successfully reduced within the barrier $0 \leq x \leq L$ to 20% of the at-large density, we find that the width $L = L(r)$ of the barrier varies with r as shown in Figure 4. Here we used the non-dimensional system (3) and took the infective population to be zero in $x > L$ if it was less than

10^{-4} (the result is very insensitive to this choice away from the dashed part of the curve in Figure 4).

Note that no such barrier exists for $0 < r < 0.2$, since under those circumstances the susceptible population in the protective barrier ($0.2S_0$) is sufficient to sustain the epizootic (see equation (4)). To get the actual length in km we need to know the various constants μ, a, and D. From the literature we obtain the estimates

a = fraction of surviving susceptibles = 0.2

μ = death rate = 1/(life expectancy of infectives) = 10/year

D = rate of diffusion of infectives = $60 km^2$/year

These values give r = 0.5, implying the disease reduces the population by roughly 50%, a frequently cited figure. These values also give the wavespeed c = 50km/year which is in good agreement with the empirical data from Europe. The predicted break width, in dimensional terms, is L = 14 km.

IV. Modelling Endemic Rabies

If we want to model more than the front we must take into account reproduction in the fox population. We expect this to affect the tail of the front. Assuming that S_0 is the carrying capacity in a particular rabies-free habitat, we can add a logistic population growth term to the equation for the susceptible foxes to obtain in place of the first of (1)

$$\partial S/\partial t = -KIS + \beta S(1 - S/S_0) \qquad (7)$$

where β is the (linear) birth rate. We assume that the rabid foxes do not reproduce. The non-dimensionalized equations, equivalent to (3), are then

$$\begin{aligned} \partial u/\partial t &= \partial^2 u/\partial x^2 + u(v - r) \\ \partial v/\partial t &= -uv + bv(1 - v) \end{aligned} \qquad (8)$$

where $b = \beta/KS_0 = r\beta/\mu$. The travelling epizootic 'wave' which results is illustrated in Figure 5, at least near the front. One suggestion from this is that the observed periodic outbreak of rabies in endemic areas could perhaps be a late effect of the 'over-kill' of the front. Anderson *et. al.* (1981) have speculated that it is primarily an effect of the incubation period.

Dunbar (1983) has studied the system (8) analytically and has shown the oscillations are damped exponentially in time if $b \leq b_c$ for some critical value b_c. If $b > b_c$, no oscillations occur, and (u,v) approaches (b(1-r),r) monotonically.Dunbar also gives the estimate $b_c < 4(1-r)/r$. Since $b = \frac{\beta}{KS_0} = \frac{r\beta}{\mu} = \frac{r}{\mu}\frac{S_c}{S_0}$, the condition for oscillations $b \leq b_c$ is equivalent to $S_0 > \alpha S_c$ (here $\alpha = \frac{\mu}{\beta b_c}$), that is, the system (8) will give oscillations if the carrying capacity of the environment S_0 is sufficiently greater than the minimum fox density required for the persistence of rabies, S_c. Mollison (1985) has shown that a large class of epidemic models which do not include dispersal exhibit a similar threshold for (non-damped) oscillatory behaviour.

Because of the high probability that rabies will reach disease-free countries such as England and Sweden, it is essential to have as full an understanding as possible of

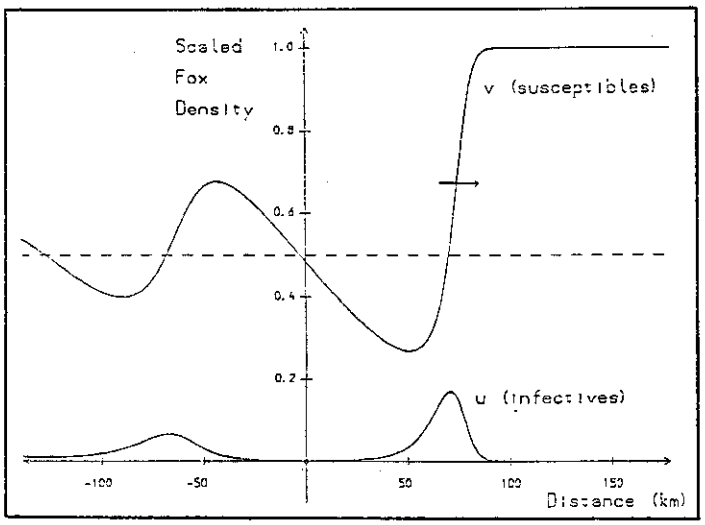

FIG. 5

both fox ecology and the spatial spread of the disease into disease-free areas. We have tried to highlight some essential quantitative questions that have to be answered before claiming to understand the gross dynamics of the rabies epizootic front.

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POPULATION DYNAMICS OF BADGERS

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Badger ecology is being studied as part of the MAFF investigation into the occurrence of bovine tuberculosis in badgers and cattle.

Two main aspects of badger ecology are presented in this paper : (i) badger territorial organisation and movements and (ii) population dynamics. In addition the paper will summarise the results of investigations of *Mycobacterium bovis* infection in the badger population under study.

The study area lies in the Cotswolds where badger territory size is small averaging approximately 30 ha and the distribution of territories is stable from year to year. Some extra-territorial movements occur, but these are mostly short forays associated with mating, movements between related social groups and movements caused by disturbance. Very few badgers have either emigrated from the study area or moved permanently away from their parental social group. An analysis of habitat utilisation shows that badgers spend 60% of their foraging time in permanent pasture which comprises only 25% of the available habitat.

Badger density in the undisturbed part of the study area fluctuated little with approximately 16-20 badgers per km^2 from 1979-1984. There is some evidence of a population regulatory mechanism in the proportion of adults and cubs seen from year to year. Mortality in adults averages 30% per annum and an estimated 65% of cubs die in their first year of life. Recolonisation of cleared areas is also under study. Modelling work by Anderson and Trewhella (1985) has predicted that it will take 5-7 years for badgers to recover to carrying capacity following removal. The speed of recolonisation will depend on a number of variables. Data being accumulated in the Cotswold study area indicates that the badger population will take at least 7 years to reach pre-removal density.

There is no reliable test for diagnosing tuberculosis in live badgers. In order to investigate the epidemiology of tuberculosis infection in this badger population two relatively crude methods of sampling are employed. These methods have nevertheless yielded some valuable data on both the prevalence and incidence of disease and the characteristics of tuberculosis infection in wild badgers. The first of these methods involves regular systematic sampling of badger faeces. Results have demonstrated that in the last four years infection has been confined to one part of the study area. It therefore appears that the transmission of tuberculosis infection from one badger social group to another probably occurs at a low rate. The second method of investigating tuberculosis infection involves clinical sampling of live badgers. The results obtained so far have enabled certain tentative conclusions to be drawn:

1. Estimated TB prevalence in the study area was relatively low when sampling began in 1981 and by the end of 1984 had declined to an even lower level.

2. No apparent relationship has been revealed between estimated TB prevalence and badger population density in the social groups regularly sampled.

3. A high proportion of tuberculous badgers have survived for long periods.

4. TB has not been a major cause of mortality.

5. The badger is an ideal maintenance host for *M. bovis*.

Examination of badgers found dead during the period of study has confirmed that the motor car is a relatively important cause of death in badgers and that TB is a relatively minor cause of mortality. A greater prevalence of tuberculosis infection is seen in male badgers than in female badgers.

It is hoped that in time this long term prospective study will give a better understanding of the relationship between tuberculosis infection in badgers and cattle, and eventually reveal any association between the prevalence of tuberculosis in badgers and their population dynamics.

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"EPIDEMIOLOGY OF BOVINE TUBERCULOSIS IN WILD ANIMALS"

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For the bacteriologist and public health worker there are three main aims of epidemiology in the study of infectious disease. The first is the positive identification of the source of the infectious agent that is being dealt with. Not only is the identity of the infectious agent necessary, the accurate identification of the strain involved may be equally important (*vide infra*). The second aim is to study and identify reasons for the chain of infection that has occurred and resulted in the outbreak in the human or animal population at risk. This may or may not be important. In the past, for example, the realization that polluted water spread typhoid fever was instrumental in its control. Conversely, the knowledge that close contact and the presence of large numbers of individuals creates ideal conditions for measles and other respiratory diseases has not helped in its control while immunization has. The third aim is to mimic the spread of the disease by using models and, if these are accurate enough, to explore the most effective ways of controlling the disease.

As a bacteriologist I want to examine the first and third aims in relation to bovine tuberculosis in badgers in more detail, leaving the second aim to the environmental health officer and the animal ecologist.

There is no doubt that high levels of bovine tuberculosis (*Mycobacterium bovis* infection) exist in badgers. This reservoir of infection is a very great potential risk to both domestic animals and man. The public health risk has, I believe, been underestimated and the effect of exposure to adults and children in the South West has not been fully assessed. This is difficult in a country where the majority of people are Mantoux positive or Heaf positive.

Bovine tuberculosis in badgers is largely a respiratory disease although fulminating infections following infected bite wounds occur. The disease is thought to spread between badgers by close contact as in man (particularly siblings).

Most of the disease in badgers occurs in the South West of England where most cattle reactors now occur. This can be seen by the proportionately large amount of money spent in this region on compensation payments (Barrow, 1982). The presence of badgers and cattle, both with disease in the area, suggested a causal relationship. This has been backed up by the sampling of other animals by MAFF which reveals by far the most infection in badgers with very little in other species.

A closer look at MAFF sampling reveals some inadequacies. Sampling should be carried out by population removal or random sampling. Neither of these has been used strictly. Analysis of such figures and of estimated required sample sizes for populations with different levels of infection by using the Binomial expansion reveals that some of the samples have been very inadequate in the past (Barrow, 1983) and some other species cannot be excluded from investigations.

However, an attempt was made to remove the populations of some mammals on two farms where outbreaks in cattle had been associated with infected badgers (Barrow & Gallagher, 1981). Although removal was obviously not attained no isolations of *M.bovis* were made except from badgers.

The ideal situation would be a combination of both methods combined with assessing the susceptibility of species at risk to *M.bovis* infection. In this way

sampling can be concentrated on species known to be susceptible.

The badger then is probably the most important reservoir of infection. Is there then proof that they have transmitted infection to cattle? This is difficult to prove. A method for identifying strains within the species *M.bovis* is necessary. Until recently there was no method available. Such "typing systems" allow the investigator to ascertain whether the characteristics of the isolated bacterium are identical from the various sources. This is immensely important so that, for example, in the case of food poisoning, although *Salmonella typhimurium* may have been the cause of an outbreak, its isolation from a chicken farm does not mean it was the source of infected chickens since this bacterium is so common. If however, the organism isolated from the outbreak had characteristics different to the organism isolated from the chickens one can say with definition that the chicken farm was not involved. However, if the types are identical, it still, unfortunately, is not proof of a source of infection.

A typing system for *M.bovis* was developed (Barrow, 1981) which was used to analyse strains from cattle and badgers on one farm in the Cotswolds. This suggested that transfer of infection had occurred from badgers to cattle or *vice versa* and between badgers of adjacent social groups. However, much more work needs to be done on this sytem before it can be used extensively.

Mathematical models have been used to describe various infectious diseases of man. These can be used to study the effects on the levels of infection of different control measures such as immunization and chemotherapy. These diseases differ from tuberculosis in badgers in several respects. First and foremost, there is a paucity of data on such essential characteristics as morbidity, mortality, infectivity, incubation period and the duration of infection and also whether immunity exists in any form. Secondly, most of the models relate to a population of fixed size and certainly of standard density. This does not occur in wild animal populations. Thirdly, the disease has an extremely long duration compared with the life of the animal. Last, the models all imply random mixing of the population to produce cross infection. Such models cannot therefore be applied to populations which exist in discrete, discontinuous groups. The epidemiology in such populations is obviously more complex. The spread of disease can be studied using simple functions to describe spread from one group to another. Using such a crude representation it can be deduced that spread of infection between groups in areas of high badger density will be at a greater rate and more diffuse than in areas of low density where infection may follow one pathway from social group to social group.

The model however is too simplistic and is based on the assumption that most inter-group transmission occurs at boundaries between males. This may not be the case: because of frequency of contact and probable increased susceptibility of cubs, the cub-dam group may represent a nucleus of infection which is spread to other groups as females mature and join another social group. If these breed they then infect their own cubs. Many adults may be partially immunized by other environmental mycobacteria. Thus it is unclear whether maximum transmission will occur at boundaries to adjacent groups with a high local density or by infected animals, particularly females, moving preferentially to areas of relatively low population density.

Using the presently available deterministic models we can deduce some probable characteristics of the epidemiology of the disease in badgers.

The disease is unlikely to regulate the population as do diseases where the mortality rate is very high compared with the rate of population growth. The mortality rate of tuberculosis in badgers is probably low and the potential population growth rate, in the Cotswolds at least, is high.

The threshold population to maintain the disease is likely to be very low because "pseudo-vertical" transmission occurs from dam to cubs, the period of infectivity is very long and the mortality rate is low.

The disease is unlikely to be cyclic. Diseases such as measles affecting large cities fluctuate because of the short duration of infection compared with the individual's life span, the presence of long lasting immunity and the slow appearance of a new susceptible population. The apparent cyclicity of infection observed in badgers in some areas may reflect spatial fluctuations as a result of sampling, population change etc.

For an adequate model much more information on the disease in badgers is needed including infectivity (trapped infected animals should be kept with healthy ones), morbidity (a seriological test should be developed), mortality (difficult to measure with some animals dying underground) and the level of immunity. Thus much more information is needed for a working model of *M.bovis* infection in badgers to be developed.

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KEY CATEGORIES IN THE REGULATION OF RED DEER POPULATIONS

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The effects of increasing population density on survival are unlikely to be uniform for all individuals in the population. Just as changes in population size can be caused principally or exclusively by changes in survival at one stage of the life history, so too can they be caused by changes in the survival of one category of animals within the population. This paper considers to what extent different categories contribute to changes in population size, in a wild herd of red deer studied on the Isle of Rhum from 1971.

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RABBIT REPRODUCTIVE RATES IN RELATION TO LENGTH OF THE BREEDING SEASON

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Rate of population increase depends on the balance between recruitment and death or emigration. In mammals which reproduce seasonally and have several litters per seasons, such as the wild European rabbit, recruitment will depend on a combination of litter size, the length of the reproductive season and neonatal survival. This paper examines the way in which the length of the reproductive season may affect recruitment.

Importance of length of breeding season

Rabbits caught from the wild or bred in captivity from wild stock, were kept in male-female pairs in separate enclosures over 3 consecutive years. All rabbits had food excess to normal requirements. The frequency of littering and the size of litters was recorded. Mean litter size showed no significant difference between years (Table 1). Year 1 had the lowest production of young per female which appears to have been due mainly to the low littering frequency which, in turn, correlated with the length of the breeding season (Table 1). The shorter breeding season in year 1 was mainly due to a delayed start. This demonstrates that production of young is probably more dependent upon when breeding begins each year than on either the litter size or the time when breeding terminates. Observations of rabbit populations tend to confirm that the time when breeding terminates is quite constant compared with the time of onset (Andersson *et al.* 1979a; Andersson *et al.* 1979b; Boyd 1985). Hence, it is reasonable to view the length of the reproductive season as a major constraint on the rate of recruitment.

Year	Number (female)	Number of young/ female	Number of litters/ season	Mean size litter	Week 1st conception	Week last birth	Difference (weeks)	Weeks not pregnant
1979	47	7.1±0.4	1.7±0.1	4.3±0.1	14.0±0.8	27.3±0.9	13.3±1.0	3.1±0.8
1980	43	11.0±0.9	2.8±0.2	3.8±0.2	8.7±0.4	25.0±0.9	16.3±1.0	4.4±0.7
1981	43	10.4±0.8	2.5±0.2	4.1±0.1	8.4±0.9	24.6±1.2	16.2±1.3	5.5±1.0
$F_{(2,130)}$		9.021	16.92	2.420	15.87	1.35	1.55	11.40
P		<0.001	<0.001	NS	<0.001	NS	NS	<0.001

Table 1 Breeding results of rabbits kept in an enclosure over 3 consecutive years

Influences on the length of the reproductive season

The breeding season of wild rabbits in northern Europe usually begins during February and ends in early August. Termination of breeding is most probably controlled by declining daylength after the summer solstice (Boyd 1985) which would account for the consistency in its timing. The start of breeding is also primarily under photoperiodic control (Boyd 1985) but greater variation in this time is introduced by the modifying influence of factors such as temperature, nutrition and social circumstances. Little detailed information is available about the effect of these other factors but most variation in the start of the reproductive season is probably due to their combined effect.

Effect on rate of population increase

No studies have examined the relationship between population growth and the length of the reproductive season. However, from Table 1, on average, the number of young born to a female per week of the breeding season was 1.87, 1.48 and 1.56 for each year respectively. Considering that between year 1 and year 2 there was a 50% increase in the birth rate and, over the same period, there was a 21% decline in the birth rate per week, then birth rate per week provides an indicator of reproductive rate with which to measure the effect of fluctuations in the length of the breeding season on the reproductive output of a population.

Consequences for epidemiology

Juvenile rabbits appear to be particularly susceptible to disease (Lloyd 1981). Myxomatosis may still kill 50% or more of the annual production of young although what proportion of these animals are already weakened by coccidial infection, malnutrition and social stress, which would in any case have led to an early death, is unknown. The annual pattern of reproduction in rabbits leads to a peak population density in late summer while many individuals are still juvenile and immunologically naive. These conditions are ideal for the establishment and spread of disease. By improving our knowledge of the environmental factors which influence the start of the breeding season and thence of productivity, in tandem with our knowledge of the dynamics of disease, we may be in a position to predict the expected eruption of a disease epidemic by measuring several environmental variables in advance.

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COCCIDIOSIS IN RABBITS

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Introduction

Coccidial infections can be the cause of death in domestic rabbits (Chapman 1929); and in young wild rabbits, in New Zealand (Bull 1958), Australia (Mykytowycz 1962) and Britain (Stephens 1952). An investigation of coccidial infections in a wild rabbit population in Southern England is reported here. The prevalence of the disease is considered with respect to three variables which reflect the density of young animals, namely peak summer population size, time of year and breeding group size.

Methods

During 1979 and 1980 samples of faeces were collected from 184 cage trapped rabbits belonging to a population living on a 10 ha bowl of chalk downland. Cowan (1983) gives details of the trapping, marking and observation techniques used to measure changes in population size and patterns of mortality. The faeces samples were maintained at $17^{o}C$ for 48h to encourage sporulation (Mykytowycz 1962) and then placed in 10% formalin. One to two grammes of faeces from each sample was macerated in 5ml of distilled water and washed through a 1.5mm sieve with a further 40ml of water. A count of the total number of coccidial oocysts in 0.5ml of the resulting solution was made with further dilution if the number present exceeded 500. Up to 100 of the oocysts in each sample were identified using the descriptions given by Levine and Ivens (1972). For each species the number of oocysts present per gramme of faeces was calculated. The counts of the most pathogenic species, *Eimeria stiedai*, which invades the bile duct and causes hepatic coccidiosis were pooled with those of *E. media*, one of the more pathogenic intestinal species, because their oocysts could not be distinguished in unsporulated form.

Coccidial infections, particularly by the more pathogenic species, are highly age specific and oocyst counts decline rapidly from 16 weeks of age onwards (Cowan 1983). Hence, only samples taken from animals captured when below this age are included in the following analyses. All counts were $\log_{10}$ transformed to conform to parametric assumptions.

Results

The peak summer rabbit population was 221 in 1978 and 124 in 1979 (Cowan 1983). Both the total oocyst and *E. stiedai/media* counts were higher in 1978 than 1979 (Table 1).

Table 1. Variation in oocyst counts (log oocysts/g faeces) between years.

		1978	1979	F
	n	51	63	
All species	$\bar{x}$	4.97	4.70	11.84
	SD	0.47	0.38	p=0.0008
E. stiedai/media	$\bar{x}$	3.86	2.44	18.10
	SD	1.42	2.02	p=0.0001

The number of young rabbits present on the study site increased during the course of each breeding season (Cowan 1983). The oocyst counts also increased amongst successive cohorts of young born during each breeding season (Table 2).

Discrete breeding groups were recognised on the basis of shared access to burrow systems: groups contained one to 12 adult females and the number of young born per group was positively correlated with female group size (Cowan 1983). The *E. stiedai/media* counts were higher amongst young born into multi-female groups than those born to single female groups (Table 3).

Table 2. Variations in oocyst counts (log oocysts/g faeces) amongst young born during different periods of the breeding season.

		Time of birth			
		Early (February to mid April)	Mid (mid April to mid June)	Late (mid June to September)	F
	n	42	57	15	
Total oocysts	$\bar{x}$	4.67	4.89	4.99	4.73
	SD	0.40	0.37	0.65	p=0.01
E. stiedai/ media	$\bar{x}$	2.29	3.39	4.06	6.96
	SD	1.95	1.82	1.34	p=0.001

Table 3. Variations in oocyst counts (log oocysts/g faeces) amongst young born into single female and multi female breeding groups.

		Natal breeding group		
		Single female	Multi-female	F
	n	17	97	
Total oocysts	$\bar{x}$	4.72	4.84	1.12
	SD	0.31	0.46	NS
E. stiedai/ media	$\bar{x}$	2.09	3.25	5.56
	SD	2.05	1.84	p=0.02

In a three-way ANOVA the between year and time of birth effects were significant for both total oocyst (F=9.65 df=1,103 P=0.002; F=4.28 df=2,103 P=0.016) and *E.stiedai/media* counts (F=14.13 df=1,103 P=0.000; F=6.96 df=2,103 P=0.001). The effect of female group size on the *E. stiedai/media* count was only significant at the 10% level (F=2.99 df=1,103 P=0.087).

In 1978 seven juveniles were found dead in cage traps. Four had enlarged livers and yellowish lesions typical of tubercles caused by infection with E.stiedai (Chapman 1929). The three other individuals showed signs of diarrhoea and the contents of their large and small intestines were mucoid with occasional flecks of blood. One individual had an enlarged small intestine with a number of distinct white lesions. Microscopic examination revealed large numbers of coccidial oocysts in the intestines of all seven individuals.

Discussion

The severity of coccidial infections in young rabbits reflected variations in population density between years, within seasons and between breeding groups. Cowan (1983) found that the patterns of juvenile mortality were also density dependent with particularly high mortality in 1978, higher mortality amongst successive cohorts born in a given season, and higher mortality amongst young born to multi-female breeding groups. While these trends do not necessarily reflect cause and effect coccidiosis was strongly implicated in the deaths of at least seven individuals. These findings suggest that further investigations of the relationships between density dependent patterns of coccidial infection and juvenile mortality are warranted.

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THE INFLUENCE OF MYXOMATOSIS IN REGULATING RABBIT NUMBERS

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Summary

The changes in the relationship between myxomatosis and the wild rabbit and the effect of these changes on rabbit numbers are reviewed. The main factors influencing these changes (the transmission of the disease, the virulence of field strains of virus and resistance and immunity in rabbits) are discussed.

Introduction

Myxomatosis has been the single most important factor influencing rabbit numbers in Britain since its appearance in 1953. During the first sweep of the disease between 1953 and 1956, it is estimated that rabbit numbers were reduced by 99% (Lloyd, 1970). Two major changes in the relationship between the disease and the rabbit allowed rabbits to survive and to increase again in numbers to roughly 20% of pre-myxomatosis populations.

The original outbreaks involved a virus strain that killed >99% of infected rabbits, but within 2 years weaker strains of virus were found and by 1962, the most commonly occurring field strains were of moderate virulence (killing 70-95% of infected rabbits). The reason for the predominance of moderately virulent strains was that such strains were transmitted more effectively by rabbit fleas (Mead-Briggs & Vaughan, 1975).

By about 1970, rabbits from one site were shown to have developed a significant degree of resistance to a moderately virulent virus strain (Ross & Sanders, 1977), and it was later demonstrated that this resistance occurred in three other widely scattered rabbit populations (Ross & Sanders, 1984).

When resistance to myxomatosis was detected in Australia (much earlier than in Britain), it was predicted (Fenner & Ratcliffe, 1965) that the virulence of field strains of virus would increase since, in resistant rabbits, the most effectively transmitted strains would be of higher virulence. Such a change in the virulence of field strains of virus was recorded in Britain between 1962 and 1975 and again between 1975 and 1981 (Ross & Sanders, in prep.).

There appear to be three possible courses for the future development of the relationship between virus and rabbit:-

1. Resistance may continue to increase and virulence of field strains may continue to increase in response.

2. Resistance may continue to increase but there may be a limit to selection of more virulent strains.

3. There may be a limit to the development of resistance and the increase in virulence would stop also.

It appears that 2. is occurring now in Britain since rabbits from one site have been found to be resistant to a fully virulent virus strain (Ross & Sanders, 1984), while fully virulent field strains continue to be rare (Ross & Sanders, in prep.). However, the possibilities outlined above are not mutually exclusive; selection for

resistance in rabbits could reach its limit, and virulence could increase again due to mutation. Investigation of the consequences of the different possibilities is one area where mathematical modelling could be useful. A relatively simple model of myxomatosis may allow predictions of the future importance of the disease. Some of the factors influencing the effects of the disease are discussed below.

Transmission

The main vector in Britain is the rabbit flea, and it is unlikely that the number of fleas is ever a limiting factor since the maximum number of fleas per rabbit at any time of year was found to be 10-20 (Mead-Briggs, Vaughan & Rennison, 1975). Approximately 42% of fleas from an infected rabbit are capable of infecting another rabbit (Mead-Briggs & Vaughan, 1975) and 45% of fleas leaving a rabbit find a new host (Mead-Briggs, 1964).

Incubation period and infectious period

For the strains most effectively transmitted, the incubation period (time from initial infection until sufficient virus for effective transmission is present in skin lesions) is 6-7 days, and the infectious period (during which fleas can pick up sufficient virus to infect another rabbit) is 10-20 days (Mead-Briggs & Vaughan, 1975).

Rate of spread

The rate of spread of myxomatosis in the original outbreaks was relatively high, 5.5km. per month (Armour & Thompson, 1955), because of the high population density and lack of any resistance or immunity. More recently (Ross & Tittensor, 1981), rates of spread have been similar to the 200-400m. per month reported by Chapple & Lewis (1964).

Estimates of mortality

The extent of immunity, particularly in adult rabbits, and the proportion of rabbits which escape infection during an outbreak, influence the prevalence of the disease. The virulence of field strains and the development of resistance influence the case mortality. The best estimate of the mortality due directly or indirectly to myxomatosis during the 1970's is 40-60% (Ross & Tittensor, 1981).

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THE EPIDEMIOLOGY OF HYDATID DISEASE AND THE CONTROL SCHEME IN SOUTH POWYS, WALES

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Echinococcosis or hydatid disease is a cyclozoonotic infection (i.e. development takes place in two species of animals) of world wide importance, caused by the closely related cestode parasites *Echinococcus granulosus* and *E. Multilocularis.* The adaptability of these parasites to an unusually wide variety of host species has made possible their broad geographical distribution throughout the world. Hydatid disease is a medical and, to a lesser extent, an economic problem on all the inhabited continents.

Epidemiology

LIFE CYCLE

E. granulosus is a tapeworm 3-9mm in length and consists of three or four proglottides. The terminal segment becomes gravid and is the broadest and longest. In the United Kingdom the dog and fox are the normal definitive hosts; eggs released from proglottides are passed in their faeces directly onto pasture or carried by the wind onto water supplies and then picked up by cattle, sheep, pigs and horses.

Man can be infected by ingesting eggs attached to inadequately washed vegetables or accidently by handling an infected dog. It is also possible that flies may carry eggs to uncovered food or that eggs may be inhaled in dust.

Each egg consists of an embryo with six hooks, the hexacanth embryo, which is surrounded by an egg case. When an egg is swallowed by an intermediate host the shell is dissolved away and the liberated hexacanth embryo burrows into the intestinal wall until it gets into a blood vessel, usually a branch of the portal vein, in which it is transported to the liver. In the liver the embryo may be retained by the filtering action of the blood capillaries or it may pass on, to be held up by the lung capillaries, or may even pass into the arterial system to other parts of the body. The embryo grows into a hydatid cyst and by 5-6 months may contain brood capsules and scolices (tapeworm heads).

A dog may ingest one or a number of fertile hydatid cysts with its meal of raw offal from a slaughtered sheep, cow, pig or horse or from one of these animals lying dead in a field or on hill grazings. The invaginated scolices are carried along to the duodenum, where, stimulated by the presence of bile each scolex evaginates and, become aggressively mobile, attaches itself to the dogs intestine by means of four muscular suckers and a double row of hooks. Each tiny scolex grows rapidly and by the time three or four proglottides have been produced it is mature. The whole development may occur within 6 weeks, thus completing the life-cycle and releasing eggs to be passed in the dog's faeces and infect new intermediate hosts.

Prevalence of *Echinococcus granulosus* in the definitive hosts

DOGS

In most parts of the world the prevalence of echinococcal infection in dogs is the most readily obtainable index of the extent of infection in a local area and of the relative degree of risk to man. The only diagnostic procedure that can be used on

a living dog is the administration of arecoline, restraint of the dog until it purges and careful examination of the complete purge for echinococcus. It is necessary to sample other dogs, as well as farm dogs, in an area and particularly packs of hounds. In Mid-Wales surveys have revealed one in four of farm dogs and a similar proportion of foxhounds to be infected with *E. granulosus.*

FOXES

A survey by necropsy of the wild red fox in Mid-Wales has indicated that 7% of them carry *E. granulosus.* Whereas there may be many thousands of worms in a dog, the fox has a relatively light infection, rarely more than 20 or 30 worms.

Prevalence of Hydatid cysts in the intermediate hosts

ANIMALS

Hydatid cysts occur in the intermediate hosts and surveys in abattoirs indicate their prevalence in cattle and sheep. This gives an overall picture of the disease because even when the results of specific abattoirs are considered they do not give an accurate picture of local prevalence, since not all animals are slaughtered in the area where they are born and reared. The prevalence rate for sheep slaughtered in the United Kingdom varies from 2.5 to 3.5 per cent each year. In Wales the number of infected animals examined in specific surveys has been as high as 37% of those slaughtered.

HUMANS

Evidence of human infection is often more difficult to obtain as the disease is not notifiable in the United Kingdom. Hospital records and the results of serological surveys indicate that in Wales people living in Powys and Glamorgan show a higher incidence of hydatid disease than other parts of Wales or the rest of the United Kingdom.

Symptoms of Disease

DEFINITIVE HOSTS

Dogs are unlikely to show any symptoms of disease even when infected with many thousands of *E.granulosus.* The proglottides are much smaller than those of the other cestodes found in dogs and owners will not be aware of the potential danger.

Intermediate Hosts

ANIMALS

It is generally assumed that hydatid cysts do not exert clinical effects on sheep and cattle, though little work has been done on this aspect. One survey claimed that animals infected with hydatid weighed 17% less than uninfected animals and it is possible that heavily infected old ewes are more susceptible to stress especially if they are also pregnant.

HUMANS

Hydatid cysts occur mainly in liver and lungs, though they occasionally occur in other parts of the body such as spleen, kidney, brain and in bones. All have to be considered to be potentially very serious and their surgical removal can be a hazardous operation. Recently anthelmintics have been used to treat hydatid cysts with, so far, variable results.

CONTROL SCHEME IN SOUTH POWYS

Control Authority

Experience in the setting up of control programmes has shown that one of the first steps to be taken is the appointment of a control authority. This has been the case in Wales, when it was decided that work initiated by the South Powys Hydatid Liaison Group, and subsequently, by the Powys District Health Authority, should be continued by the provision of funds by the Welsh Office and extended to an Hydatid Control Scheme in South Powys.

The philosophy and approach to the control of hydatidosis is comparable to that for diseases, such as tuberculosis and brucellosis both of which have been successfully controlled in the United Kingdom. The control scheme is operated on a voluntary basis since no national legislation exists, by which it can be made compulsory.

Functions of the Control Authority

There are four important functions for the Control Authority:-

1. **Funding of the programme and its planned expansion**

The control campaign is funded by the Welsh Office with veterinary staff costs met by the Ministry of Agriculture. For the next three years the control scheme will be confined to South Powys.

2. **Selection and training of Personnel**

Veterinary personnel in the State Veterinary Service will be responsible for implementing the control measures. Technical staff in the State Veterinary Service will be trained to assist in the control measures to be adopted.

3. **Collection of Baseline Data and Continuing Surveillance Data**

Surveys are of fundamental importance to:

A. Establish the importance of echinococcus compared with other diseases.

B. Obtain basic data and insight into the process of transmission.

C. Provide baseline data for the subsequent establishment of control measures.

Surveys have established the high incidence of *E.granulosus* in definitive hosts in the area and that of hydatid cysts in man and animals.

4. **Educational Component and the Technical Measures to be adopted**

Educational Component

Community health education must be regarded as a basic component of all echinococcus control programmes.

The initial visit to all dog owners in South Powys is being made by a veterinary surgeon. These visits are being supplemented by explanatory leaflets, articles in the local press and posters. Meetings with local community organisations include Young Farmers Clubs, National Farmers Unions and local Women's Institutes.

Control Measures

The important cycle of infection in South Powys is that of the sheep and sheep dog and it is necessary to understand the epidemiology of the disease in order to establish control measures.

Sheep dog Management

Dogs in the area are used to scavenging and the hill grazings where many sheep die and remain unburied are within one or two miles of the farm buildings. Sheep are also killed by motor vehicles on the unfenced roads passing through the area. Up to one third of the sheep in the area have been found to be infected with hydatid cysts. The majority of dogs are housed or tethered at night and when released have been seen to defaecate within a few minutes of leaving farm buildings. Infected dogs will contaminate the nearby fields resulting in a heavy build up of worm eggs on the pasture.

Sheep Husbandry

The majority of sheep farms in Powys have hill grazings where sheep spend long periods of the year and many die and are likely to remain unburied. When the sheep are collected from the hills they will be concentrated in the fields around the farm buildings. Fields which have been heavily contaminated with tapeworm eggs.

There are two main control measures -

A. **Prevent dogs gaining access to raw offal**
The disposal of infected raw offal from abattoirs and knackers yards is controlled by the Meat (Sterilization and Staining) Regulations 1982. Hunt kennels do not come within the scope of the regulations. Epidemiological surveys have shown that the main sources of raw offal to the sheepdogs in the area are the sheep which die on the hill grazings and remain unburied. There is no obvious practical method of ensuring all sheep carcases are removed before being scavenged, but publicity will encourage the farming community to reduce this source of infection.

B. **Reduction in the number of E.granulosus**

(i) **Reduction of the dog population**
There are few stray dogs in the rural areas, the majority of dogs being either pet dogs or working sheep dogs.

(ii) **Dog Treatment Programmes**
All successful programmes have placed emphasis on dog registration and this will be included in the Scheme.

The introduction of new anthelmintics with improved efficacy against *E.granulosus* has resulted in a shift in emphasis, from 'arecoline surveillance' to the mass treatment of dogs. In the South Powys Control Scheme the generally agreed interval of six weeks has been adopted for treatments. They will be supervised by the technical staff of the Ministry of Agriculture. At such visits, advantage will be taken to update dog registration and maintain the educational component of the Control Scheme.

As control of the parasite improves, it may be possible to restrict the treatments to the animals most at risk. There is evidence that dog owners tend to see the dog-dosing programme as the *only* important measure required in Echinococcus control, but the introduction of highly effective drugs, such as praziquantel, does not reduce the need for other control measures.

TICKS AND VIRUSES AT A SEABIRD COLONY

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Seabirds, 98 per cent of which are colonial, constitute an interesting model for studying the quantitative relationships between host and tick, and between host, vector and virus. Great Saltee Island, Co. Wexford, Ireland, the study area of this investigation holds 14 species of breeding seabird, and four tick species, three of which, in turn, harbour at least three different viruses. These inter-relationships have been studied quantitatively under the general headings of

A) spatial aspects (a proposed index of coloniality),
B) frequency of infestation (negative binomial distribution), and
C) host specificity (niche breadth and overlap).

Preliminary data are presented and discussed.

IS GROUSE DISEASE REGULATORY ?

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Is grouse disease, trichostrongylosis, one of the biological mechanisms causing the near-regular cycles in numbers of red grouse? Currently there are two schools of thought. The English school, represented by Potts, Tapper and Hudson (1984) argue that the disease plays a key role in causing the regularity of the fluctuations: thought not the sole, or even a major cause of the fluctuations, they nonetheless claim that the fluctuations could not occur without the presence of the parasite. Contrary-wise the Scottish school, represented by Moss and Watson (1985), maintain that the worm is not an essential ingredient and that the fluctuations are caused by factors other, though perhaps in concert with, the worm.

Potts *et al.* argue that breeding success, as measured by the average brood size in August, is inversely related to worm burden. Hudson, in his paper given to the present meeting, reports his subsequent experimental confirmation of this inverse relationship of breeding success and worm burden. Potts *et al.* contend that this effect is sufficient and indeed necessary to explain the cyclic pattern in population numbers.

The ITE grouse team, in their intensive studies of three fluctuations, have shown that in two of the three fluctuations there was a delayed density-dependent relationship of worm burden to grouse breeding density. In the third fluctuation at Kerloch Moor in Kincardineshire there was insufficient evidence to establish any relationship. Even so breeding success was lower during the decline phase of the fluctuation than in the increase phase, and we cannot exclude the possibility that this was caused by the variation in the worm burden.

Watson, Moss, Rothery and Parr (1984) show that changes in population numbers are brought about by changes in the following factors:

i. Sex-ratio

   In the decline phase many territorial cocks are unmated. It is possible that the debilitating effect of the worm burden reduces the ability of a territorial male to maintain a mate.

ii. Brood-size

   There is a density-delayed inverse relationship of brood-size with density in the second fluctuation at Kerloch.

iii. Overwinter loss

   About half of the autumn stock are lost over winter; for the most part the loss is through emigration.

iv. Spring and summer emigration

   In the second fluctuation at Kerloch many of the spring stock emigrated either in spring before hatching of eggs, or in summer taking their young with them. Taken together, spring and summer emigration constitute a major cause of demographic change: from the peak year in the second fluctuation and throughout the decline phase more than a half of the potential broods were lost to the population through emigration.

Changes in the sex-ratio and in breeding success may well be the result of worm infestation. However the effect of the worm burden on incidence of spring, summer and winter emigration is not so clear. It is possible that as the worm load increases the birds are persuaded to emigrate so as to escape from the increasingly unhealthy conditions. On the other hand the data collected at Glen Esk in the 1956-61 fluctuation show that much the same numbers of worms were carried by territorial birds as were carried by non-territorial birds.

The explanation for the cyclical changes put forward as a possibility by Moss and Watson (1985) is that changes in tolerance occur as a consequence of neighbours tending to be closely related during the increase phase. They quote the findings of Lance (1968). Using radio-telemetry, Lance monitored the competition for territory by young red grouse cocks. He found that the young cocks returned to their birthplace, the territory of their father, to seek a territory of their own: thus a group of neighbouring cocks might comprise father, sons and brothers. Moss and Watson argue it is reasonable to suppose that kindred neighbours should tolerate each other. At low densities a father could increase his genotype's fitness by allowing his sons to take parts of his old large territory. At peak densities many territories, particularly those of young cocks, are too small to support a hen. At this stage the fitness of a family's genotype is no longer increased by successive generations taking yet smaller territories. Tolerance then changes to intolerance. The strongest, the most competitive, individuals force the weaker brethren to emigrate.

The fluctuation in density may also be induced, to some extent, by the variation in age-structure. As said before, at peak densities many territories, particularly those of young cocks, are too small to support a hen. The birds that actually beget offspring then tend to be the older birds. In fact in both of the Kerloch fluctuations during the years of increase the proportion of young cocks in the territorial population was about 50%; at peak densities and in the years of decline the proportion of young cocks in the territorial population fell to 20%-35%. This seesawing in the age-profile could help to bring about the cyclic fluctuations: as the breeding population becomes older the numbers decline; as the population recovers to a young age-structure the numbers increase.

Moss and Watson make the important point that insight and understanding will be forthcoming only if the study is pitched at an appropriate level of abstraction. As the fluctuations have resulted from changes in the birds' spacing behaviour, an understanding can be gained only by studying this behaviour. The biological mechanisms are unlikely to be uncovered by a study of aggregate properties of the population. Though useful for predictive purposes, average values provide little information on why one individual rather than another succeeds in joining the breeding population or what are the biological mechanisms underlying emigration.

It will be only from detailed studies that an understanding will be obtained of the regulatory process.

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POPULATION CYCLES IN RED GROUSE: DO PARASITES PLAY A ROLE ?

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The causes of the regular fluctuations in some animal numbers have fascinated population biologists ever since Charles Elton first described the ten year cycle in Canadian wildlife. Interest in the fluctuations of red grouse populations grew when Lack and Chitty independently analysed data from an early study of grouse and came up with conflicting conclusions. Lack suggested that regulation was due to parasitism and starvation whereas Chitty thought it was due to intrinsic changes within the birds. Since then the idea that intrinsic changes in the birds' behaviour could cause population cycles have been developed further and investigated in detail in north-east Scotland (Moss, Watson & Rothery 1984, Watson *et al.* 1984).

Interest in the role of parasites in the regulation of animal numbers has grown through the development of theoretical models (Anderson & May 1978, May & Anderson 1978) which demonstrate that certain features of a host-parasite relationship can generate cyclic fluctuations in host numbers. By applying one of these models to the red grouse and its caecal nematode *Trichostrongylus tenuis*, Hudson, Dobson & Newborn (1985) have shown that there are several essential features which must be met if the parasite is to cause cycles in grouse numbers. The more important of these is a reduction in grouse fecundity through the effects of the parasite and a high survival rate in the free-living stages. The first feature has been investigated in field experiments on red grouse in the north of England. By reducing the parasite burdens in wild grouse Hudson (in press) has demonstrated that parasites reduce the condition and breeding production of hen grouse. These experimental findings are consistent with correlations between worm burdens and breeding success both within and between estates, demonstrating that parasitism has a role to play in influencing the fecundity of grouse.

Potts, Tapper & Hudson (1984) have produced a simulation model of a grouse population as an explanation for the cyclic bag records in the north of England. In the model the burden of parasites in breeding grouse is determined by the density of birds in the previous year. The level of infection reduces the breeding production of the grouse and subsequently the number of birds harvested. Sensitivity analysis of this model shows that this delayed density dependent relationship between parasites and breeding success generates cyclic bag records similar to those observed on estates. The relationship between breeding production and parasite levels used in the model conforms with the experimental findings of Hudson (in press) indicating that the measured effect of parasites on breeding success can account for the fluctuations observed.

Not all grouse populations produce regular cyclic bag records. Hudson, Dobson and Newborn (1985) have looked at differences in parasite burdens from cyclic and non-cyclic populations and found cyclic populations are associated with greater levels of infection and higher annual rainfall than non-cyclic populations. They propose that low humidity reduces the survival of the free-living stages, resulting in lower levels of infection and reduced effects on host fecundity in non-cyclic fluctuations.

These results indicate that parasitism has a role to play in the population cycles of red grouse in the north of England. Nevertheless, they can not refute the hypothesis that inherent factors may be influencing population cycles in north-east Scotland. Indeed, since rainfall in north-east Scotland is relatively low compared to the central Pennines these studies would not have expected parasitism to have played a key role in causing cycles. It seems likely that in the absence of the effects of extrinsic factors such as parasitism on breeding production then intrinsic factors influencing spacing behaviour can cause population cycles. This explanation was originally proposed by Watson & Moss (1979) and encompasses many of the findings from all studies.

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