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Bovine Tuberculosis in Badger (Meles meles) Populations in Southwest England: The Use of a Spatial Stochastic Simulation Model to Understand the Dynamics of the Disease

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SUMMARY

A spatial stochastic simulation model was developed to describe the dynamics of bovine tuberculosis in badger populations in southwest England, based on data from the literature and from unpublished sources. As there are no data on intra- and intergroup infection probabilities, estimates of these were obtained through repeated simulations based on field observations of the spread and prevalence of the disease.

The model works on a grid-cell basis, with each grid cell potentially occupied by one badger social group; immigration to and emigration from the main grid are incorporated. Population regulation is assumed to occur at the group level through density-dependent fecundity and cub mortality, and the

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model can be run for various disease-free equilibrium group sizes (which are determined by the carrying capacity of the environment). The model works on a quarterly (three-monthly) basis and processes are stochastic at the individual level. Three classes of individual (adults, yearlings and cubs) and three classes of infection (susceptible, infected-but-not-infectious and infectious) are recognized.

Bovine tuberculosis was shown to persist in badger populations for long periods of time, even in populations with a disease-free equilibrium group size of only four adults and yearlings. However, with standard rates of intergroup infection and movement, the disease only became endemic in populations with a disease-free equilibrium group size greater than six adults and yearlings. In the endemic situation, the prevalence of the disease ranged between 11–22% depending on the combination of inter- and intragroup infection probabilities used. Endemic infection within the homogeneous environment of the grid was characterized by a high degree of heterogeneity. Patches of infection were spatio-temporally unstable, but shifted in location relatively slowly.

Spread of the disease from a point source of infection with standard rates of intergroup movement and infection only occurred to any marked extent in populations with disease-free equilibrium group sizes of eight or more adults and yearlings. Increasing the intergroup infection probability had a significant effect on increasing the probability and rate of spread, and considerably lowered the threshold group size for spread from a point source to around four adults and yearlings. However, increasing the rates of intergroup movement reduced the probability of spread of the disease except at the largest group sizes. When both intergroup infection and movements were increased, the effects of increased infection in enhancing spread were offset to some degree by the increased movements. Perturbation to the badger population, as may be caused by control operations, could therefore increase the probability of persistence or spread of an infection.

1. INTRODUCTION

In England bovine tuberculosis (*Mycobacterium bovis*) is now largely confined to the southwestern counties of Avon, Cornwall, Devon, Gloucestershire, Somerset and Wiltshire, where badger (*Meles meles*) densities are highest (Reason *et al.* 1993) and badgers serve as a reservoir host for the disease. In experimental conditions it has been shown that the transmission of bovine tuberculosis from badgers to cattle is possible (Little *et al.* 1982*a*), and that infectious badgers may excrete bacilli into the environment in their urine, faeces, sputum and pus (Muirhead *et al.* 1974). It is believed that under field conditions, these excretory products constitute the main threat of infection to cattle (Muirhead *et al.* 1974), although the mode of transmission has never conclusively been demonstrated (White *et al.* 1993).

The presence of a wildlife reservoir for bovine tuberculosis in southwest England has complicated attempts to eradicate the disease in cattle, and since 1975 badger control operations have been in force in an attempt to reduce the incidence of bovine tuberculosis in cattle in areas where badgers are believed to be the most likely source of infection. These operations have taken various forms based initially on gassing, and later, on cage trapping (White *et al.* 1993). However, although there have been a few local successes in eradicating the disease from the badger population (Little *et al.* 1982*b*), these control operations have not led to a comparative decline in the number of herds in the southwest of England with infected cattle, nor in the overall number of infectious badgers found in samples examined by the Ministry of Agriculture, Fisheries and Food (MAFF) (MAFF 1993*a*). Furthermore, there has been no attempt until the recent policy amendment (MAFF 1993*b*) to conduct any experimental comparison of the relative success of different control policies.

One of the main problems in attempting to formulate any strategy for the control of bovine tuberculosis in badger populations is the lack of information on the spatial dynamics of the disease. Despite locally concentrated badger control operations, the areas of southwest Britain where the disease is most prevalent in cattle have remained largely unchanged. Furthermore, the distribution of the disease in badgers throughout the southwest, as determined by *post mortem* examination of road traffic casualties, is clumped and tends to be associated with areas in which bovine tuberculosis occurs repeatedly in cattle (MAFF, unpublished data).

Explicitly spatial analyses of the dynamics of bovine tuberculosis in badger populations have been ignored so far in favour of deterministic, non-spatial approaches. Deterministic, non-spatial models are useful for understanding principles such as equilibrium prevalences, threshold population density and cyclicity, and as such are useful steps towards developing more complex models. However, they also have major limitations in that the basic models assume homogeneously mixing populations, do not incorporate stochasticity, and ignore local effects of environmental heterogeneity and individual behaviour. These problems can be countered, but only through external artificial constraints on the operation of the model (e.g. Barlow 1991*a*). For acute diseases in non-structured populations, these limitations may have only a minor effect on the validity of any predictions resulting from the model. However, for chronic diseases in structured populations, such as bovine tuberculosis in badgers, these effects are likely to be of far greater importance.

Thus Anderson & Trewhella (1985) and Bentil & Murray (1993) used a non-spatial modelling approach, and some of the assumptions and predictions of both studies failed to match what is now known of badger population biology and the epidemiology of the disease in real populations. This is discussed in more detail in later sections. A major drawback of non-spatial models is their inability to aid understanding of the spatial aspects of both disease epidemiology and control

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measures. In addition, as a consequence of their structure, they are not easily applicable to practical situations in the field. In contrast, spatial models are able to address such questions more explicitly. For example, the mixed deterministic/stochastic model of Barlow (1993), which operates on a spatial grid, has resulted in modifications to the strategy to control bovine tuberculosis in possums (*Trichosurus vulpecula*) in New Zealand, which was originally based on purely deterministic, non-spatial modelling studies (Barlow 1991*a*,*b*). The resulting modifications incorporated into the control strategy included the effect of the size of the control area on immigration and hence the success of control, and strategies aimed at the prevention of disease spread rather than elimination in areas in which it is endemic.

The badger/bovine tuberculosis system is particularly well-suited to a spatial modelling approach because of the rigid territorial system exhibited by badgers living at moderate to high population densities (Kruuk 1978; Cheeseman & Mallinson 1980; Kruuk & Parish 1982). This territorial system operates where badgers are believed to be a major source of bovine tuberculosis for cattle and badger territories can therefore be considered as occupying distinct spatial cells within a model, with interactions occurring between individuals both within and between these cells.

This paper describes the use of a spatial stochastic model to examine the dynamics of bovine tuberculosis in badger populations. The model is designed to be as realistic as possible and operates according to known biological parameters. The objectives of developing such a model were two-fold. The first was to obtain a better understanding of how various biological and epidemiological parameters combine to determine the observed spatial dynamics of the disease, including an assessment of the sensitivity of the system to changes in certain key parameters that may be induced by badger control operations, and this is addressed here. The second objective was to use the model to evaluate the efficacy of current control policies and the design of future policies, and this is addressed by White & Harris (1995).

2. BADGER POPULATION BIOLOGY

The early literature on badger population biology and the epidemiology of bovine tuberculosis in badgers have been reviewed by Anderson & Trewhella (1985) and so only those aspects that are pertinent to the development of the model presented here are discussed below and in §3–5.

(*a*) *Population structure, density and territory size*

Three age classes of individuals are considered in this paper: adults (> 2 years old), yearlings (1–2 years old) and cubs (< 1 year old). Recorded population densities (defined here as the number of adults plus the number of yearlings) range from 4.7 km^{-2} in Cornwall to 19.7 km^{-2} in Gloucestershire, with mean territory sizes of 0.75 km^2 and 0.22 km^2 respectively (Cheeseman *et al.* 1981; Anderson & Trewhella 1985). However, the very large groups and small territories recorded in Gloucestershire are unusual: the average badger territory in southwest Britain probably measures around 0.7 km^2 (Cheeseman *et al.* 1981) and contains six badgers (adults and yearlings) (Cresswell *et al.* 1990).

(*b*) *Fecundity and sex ratio*

The average litter size, calculated from measurements before implantation, during pregnancy and from observations made after birth, is 2.7 cubs (Anderson & Trewhella 1985). However, only 44% of adult females produce cubs each year; 22% do not become pregnant, and 34% are fertilized but fail to implant their blastocysts (Cresswell *et al.* 1992). Lower productivity rates were calculated for badgers in southwest England by Page *et al.* (1994). However, their data were collected in the early stages of the culling programme and also included a larger proportion of road-killed animals than the more recent sample from the same area examined by Cresswell *et al.* (1992). It is possible therefore that the increased level of productivity reported by Cresswell *et al.* (1992) represents a density-dependent response to the culling programme and/or enhanced badger mortality due to endemic bovine tuberculosis. Whatever the cause, the data of Cresswell *et al.* (1992) were thought to be more representative of the current situation in the problem areas of southwest England and so were used in the present analyses.

Data from a badger population in Gloucestershire indicate that the number of cubs produced per social group is relatively constant irrespective of the number of males in the group, but that it is related to the number of females in their third year or older (Cresswell *et al.* 1992). This relation is significant ($r = 0.97$, $p < 0.001$), and can be expressed by the formula:

$$P_{\text{cubs}} = 0.60 + 0.63\ a_{\text{f}},$$

where P_{cubs} is the number of cubs produced per social group per year and a_{f} is the number of adult females in the group at the start of the year.

The sex ratio among badger cubs is generally close to unity (Anderson & Trewhella 1985; Cheeseman *et al.* 1987; Harris & Cresswell 1987; Kruuk & Parish 1987). Thus, no artificial adjustments were made to the sex ratio of cubs produced in the model, and the sexes of the cubs produced in each group were chosen with a probability of 0.5 for either sex.

(*c*) *Mortality*

(i) *Adults and yearlings*

Based on a variety of studies, Anderson & Trewhella (1985) estimated that the average adult death rate was 25% per annum and that this often remained approximately constant with age. Several studies have shown a preponderance of males in the adult population (Anderson & Trewhella 1985; Cheeseman *et al.* 1987). However, the sex ratio of the adult population is not generally significantly different from 1:1 in any single population (but see Harris & Cresswell 1987). Cheeseman *et al.* (1987) calculated annual mortality in

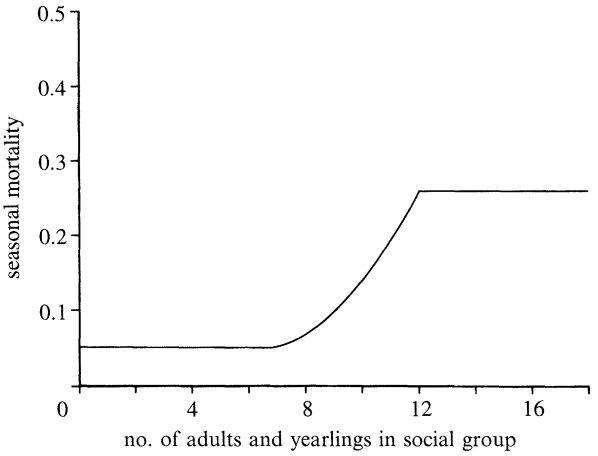

Figure 1. The relation between seasonal cub mortality and group size for a group with an equilibrium size of six adults and yearlings.

a high-density population to be 35% for males and 24% for females (mean of 28%), and Harris & Cresswell (1987) found levels of 40% for males and 28% for females (mean of 35%) in a suburban population. These studies both used trapping data as the basis for their calculations, and this may slightly over-estimate mortality because each animal not caught is assumed to have died: some may have dispersed or may just not have been caught. A more recent study by Harris *et al.* (1992), using known-age animals and tooth wear to estimate age, calculated the mean mortality rate for animals 2–7 years old to be 19%. Badgers may live for 11 years in the wild (Neal & Cheeseman 1991), and it is possible that animals >7 years old and yearlings will experience slightly higher rates of mortality. However, in the absence of any data to substantiate this suggestion, 19% was used as the annual mortality rate for all adult and yearling male and female badgers.

(ii) *Cubs*

Cub mortality is believed to be one of the density-dependent factors regulating the size of badger populations (Anderson & Trewhella 1985; Cheeseman *et al.* 1987). Mortality of badger cubs is very high. Total annual mortality for cubs may be around 60–70% (Anderson & Trewhella 1985; Cheeseman *et al.* 1987; Harris & Cresswell 1987), and Harris & Cresswell (1987) calculated from capture-mark-recapture data that 38% of cubs in a suburban badger population died before emerging from the sett. However, there are no data to quantify the relation between cub mortality and population density. Thus for the model, the following relation is assumed. Minimum cub mortality occurs where the social group is less than or equal to the disease-free equilibrium size. In these circumstances, it has the same value as adult mortality (19% per annum). However, if there are no adult females in the group during the period January to June inclusive, it is assumed that cubs are almost certain to die and they are therefore subjected to a mortality rate of 90% per annum during this period. A 70% level of mortality is assumed to occur where group size exceeds the carrying capacity by at least 100%. Hence for a standard territory with a carrying capacity of six adults and yearlings, 70% mortality will occur at group sizes of 12 and above. Groups of this size have been recorded in the literature (e.g. Cheeseman *et al.* 1987; Harris & Cresswell 1987), and more than 20 adults and yearlings have been recorded in some groups in Gloucestershire (C. L. Cheeseman, personal communication). It is further assumed that the rate of increasing cub mortality with increasing adult group size will become more rapid as adult group size increases further from the disease-free equilibrium level. Thus the density-dependent function generated, excluding any adjustments for an absence of adult females during the period January-June inclusive, takes the form of two constant components: a minimum at 19% per annum and a maximum at 70% per annum, joined by a semi-quadratic function between the disease-free equilibrium group size and 100% above this equilibrium level. This is illustrated in figure 1 for a disease-free equilibrium group size of six adults and yearlings. The semi-quadratic functions describing these changes in mortality for equilibrium group sizes of four, six, eight and ten adults and yearlings are listed in table 1. These density-dependent cub mortality functions, in conjunction with the relation between fecundity and the number of adult females in the group outlined above, ensured that population size remained approximately constant, close to the equilibrium level in the absence of the disease.

(*d*) *Dispersal*

Most long-term studies of badger ecology have been conducted in areas of high population density where badgers occupy strictly defined territories (Cresswell & Harris 1988). Typically, in such areas, dispersal is rare and generally confined to adult males (Kruuk 1978; Kruuk & Parish 1987; Evans *et al.* 1989). Cheeseman *et al.* (1988*a*) compared the dispersal and other movement patterns of badgers in rural Gloucestershire and suburban Bristol. Group changes were rare in both populations, although they were more common in the lower density population in Bristol. There were five confirmed dispersers (all male) out of 270 badgers recaptured (1.9%) for the Gloucestershire population, with another 42 possible dispersers (15.6%), giving a maximum dispersal rate of 17.5% over nine years (mean of 1.9% per annum). The respective figures for Bristol were 21 confirmed (19.6%) and a further 13 possible (12.1%) out of a total of 107 recaptured, giving a maximum dispersal rate of 31.7% over seven years (mean of 4.5% per annum). Including possible dispersers, 68% of dispersers in Gloucestershire and

Table 1. *Functions describing changes in seasonal mortality (y) with group size (x) between the equilibrium group size and twice the equilibrium group size for equilibrium group sizes of four, six, eight and ten adults and yearlings*

equilibrium group size	function
4	$y = 0.013\,x^2 - 0.105\,x + 0.26$
6	$y = 0.006\,x^2 - 0.070\,x + 0.26$
8	$y = 0.003\,x^2 - 0.052\,x + 0.26$
10	$y = 0.002\,x^2 - 0.042\,x + 0.26$

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71 % in Bristol were male. Mean dispersal distance was small (0.52 km in Gloucestershire and 0.98 km in Bristol), indicating that most animals moved to adjacent groups (see also Kruuk & Parish 1987). In another high density population in Oxfordshire, da Silva *et al.* (1994) also found that dispersal was infrequent and only occurred among adults, with only 6 % of adult males and 8 % of adult females dispersing over a period of two years (means of 3 % and 4 % per annum for males and females respectively). All of the males, and all but one of the females dispersed to neighbouring groups. The relatively high dispersal rate of females in this study may have been due to disturbance, as they all came from one social group (da Silva *et al.* 1994). Combining the Oxfordshire data with the confirmed dispersers from the Gloucestershire study, the mean proportion of confirmed male dispersers for high density populations is 1.6 % per annum. This is a minimum figure as it is probable that some of those animals described as 'possibles' in the Gloucestershire study had in fact also dispersed; inclusion of these animals would increase the figure to 2.5 % per annum. Also, both studies were undertaken in high density undisturbed badger populations, and the available data suggest that dispersal is more common and that females may disperse more readily in lower density populations and in response to increased disturbance (Cheeseman *et al.* 1988*a*; 1993; Roper & Lüps 1993).

In the areas in southwest England with endemic bovine tuberculosis in the badger population, disturbance is likely to be higher and population density lower as a result of the badger control operations that have been in force since 1975. For the initial modelling, the proportions of males and females dispersing were therefore increased slightly over those calculated above, to 6 % and 2 % per annum respectively, and badgers were allowed to disperse only to the immediately adjacent social group. Potential variations in dispersal caused by disturbance (Cheeseman *et al.* 1988*a*; Brown *et al.* 1994) may have important implications for the rate of spread of bovine tuberculosis in badger populations. Therefore, dispersal was one parameter which was later varied as part of the sensitivity analysis.

(*e*) *Colonization of empty setts*

Data on the rates of colonization by badgers are scant, although badgers appear to be poor colonists (Anderson & Trewhella 1985; Kruuk & Macdonald 1985; Cheeseman *et al.* 1993). The rate of successful colonization is likely to depend on the status and relative proximity of any surrounding groups. Based on their modelling work, Anderson & Trewhella (1985) predicted that recovery time would be approximately five years. However, such rapid recovery is extremely unlikely to occur. Field measurements of the recolonization process have only been made for one population in Gloucestershire. In this study, badgers were completely removed from two areas comprising five and six social groups, respectively. This removal had little effect on the territory boundaries of the former neighbouring groups (Kruuk & Macdonald 1985; Cheeseman *et al.* 1993). It took nine to ten years for the population in the cleared areas to recover to near pre-removal levels (Cheeseman *et al.* 1993), with the recovery process taking slightly longer in the more isolated of the two areas. However, this badger population was unaffected by any persecution, and was actually growing over the period of study. Thus, even this measurement is likely to be an overestimate of the true rate of recovery for relatively stable populations, and especially so for reduced or persecuted ones (e.g. Harris 1993). A growing population in the Gloucestershire study would have resulted not only in increased colonization into removal areas, but also increased dispersal movements into groups once they had formed in the former removal areas. Such immigrants were a major contributory factor to the rapid recovery of the population in the more isolated area in the Gloucestershire study, where no cubs were recorded for the first two years post-removal. This pattern of initially slow recovery of adult badger numbers in removal areas was replicated by the model using a colonization rate for animals of the surrounding groups of around 2.5 % per annum. Recent data from Oxfordshire suggest that females may also be important as colonizers (R. Woodroffe, personal communication), and so it was assumed that this probability was equal for both sexes.

(*f*) *Spatial organization of the population*

The dispersion of suitable habitats for badgers is patchy, and this is reflected in a very clumped badger distribution. Of 2455 1 km squares in Britain, 84.2 % had no badger setts, 12.5 % one active main sett and only 3.3 % between two and six active main setts (Cresswell *et al.* 1990). The absence of a sett does not necessarily mean that some or all of a 1 km square is not part of a territory within a population of contiguous groups. However, as the average territory measures 0.7 km^2, such a high proportion of 1 km squares with no setts would indicate that extensive areas of contiguous badger territories are not common. For this analysis, it was assumed that contiguous territories occur in the high density badger populations likely to be found in those areas of southwest England where bovine tuberculosis is most prevalent.

Cheeseman *et al.* (1988*b*) mapped the distribution of 32 badger territories in 9 km^2 in Gloucestershire. Of these, 12 were almost completely enclosed by other badger territories. The spatial arrangement was not based on optimum space use (i.e. hexagonal), but more closely resembled an offset grid of predominantly square territories. The 12 completely enclosed territories had an average of 5.9 ± 0.3 (s.e.) neighbours. However, the vast proportion of each territory boundary was shared with a mean of 4.6 ± 0.3 neighbours. The other 1.3 ± 0.2 neighbours generally shared only a small corner of the territory boundaries, and interactions with these groups would therefore be expected to be minimal. Similar patterns of spatial organization have been observed in smaller study areas in Avon (5.5 km^2) and Cornwall (6.5 km^2) (Cheeseman *et al.*

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1981), over a much larger area in Avon (100 km^2) (M. Hutchings, personal communication), in a 5 km^2 area of Staffordshire (Cheeseman *et al.* 1985), and in a 10 km^2 area of Oxfordshire (Kruuk 1978). Thus for the basic structure of the model it was decided to use a grid of square cells to represent a badger population with contiguous territories. Each grid cell was potentially occupied by a single social group of badgers, and had four main adjacent neighbours plus four corner neighbours with which interactions were minimal.

3. BOVINE TUBERCULOSIS IN BADGERS

(*a*) *Prevalence of infection*

The prevalence of bovine tuberculosis in badger populations shows much variation, suggesting a high degree of clumping of the disease within the population. Outside the southwest, the highest overall prevalence recorded in any county is 2.8% in East and West Sussex (Cheeseman *et al.* 1989), although 17.8% has been recorded locally in a 5 km^2 area of Staffordshire (Cheeseman *et al.* 1985). After local outbreaks of bovine tuberculosis in cattle in four areas in southwest England averaging 3.6 km^2, Cheeseman *et al.* (1981) found that the prevalence of bovine tuberculosis in badgers from these four areas varied from 6.9% to 34.5%. A prevalence of 65% was recorded in badgers killed during a control operation in an area of Somerset (MAFF 1993*a*).

Bovine tuberculosis in badgers appears to be strongly spatially correlated with the incidence of the disease in cattle. Of all the badgers killed during bovine tuberculosis control operations in southwest England to the end of 1991, 15.4% were positive for *M. bovis*, compared with 5.7% in a sample of badgers killed on the roads throughout the whole of the southwest (MAFF 1993*a*). However, this figure of 5.7% does not represent an overall background prevalence as it includes animals from the control areas as well as animals from other parts of the southwest. The prevalence of tuberculosis is generally higher in male badgers than in females (Cheeseman *et al.* 1988*b*; 1989). Based on data from all badgers caught in MAFF control operations over the period 1982–1993 inclusive (R. Clifton-Hadley, personal communication), this difference is significant ($\chi^2 = 39.9$, d.f. $= 1$, $p < 0.001$).

The prevalence of infection does not appear to be related to badger density within specific areas (Cheeseman *et al.* 1989). However, because bovine tuberculosis in badgers is most commonly found in the southwest of Britain, which contains 24.9% of the British badger population but makes up only 10.3% of the land area (Cresswell *et al.* 1989), it is likely that there is some relation between the presence of the disease and population density and that there is a threshold population density below which the disease cannot persist (Anderson & May 1979; May & Anderson 1979). For diseases in most populations, host density in terms of the number of individuals per unit area is a useful means of expressing this density. However, for badgers, the existence of well-defined social groups which interact relatively little makes measures of absolute density less meaningful. Although territory sizes may vary, these variations may not necessarily be reflected in changes in contacts between the groups. The chances of transmission between adjacent groups will ultimately depend on the number of infectious and susceptible animals in each group. Thus the use of the disease-free equilibrium group size as a measure of the threshold density makes more intuitive sense, and this is the approach adopted in this paper.

(*b*) *The course of infection in individual animals*

The progression of bovine tuberculosis infection in badgers appears to be slow, although the data are scant (Gallagher *et al.* 1976). Little *et al.* (1982) reported that the incubation period could last between three and five months. Once animals become infectious, recovery and the acquisition of immunity do not appear to occur (Anderson & Trewhella 1985). However, field data collected in Gloucestershire suggest that the infection may become latent, i.e. previously infectious animals cease to excrete bacilli, although such animals may later become infectious once more (Anderson & Trewhella 1985; Wilesmith 1991; C. L. Cheeseman, personal communication).

Because the probabilities of infections becoming latent and then reverting to infectious states are unknown, they were estimated based on the limited available data. Of eight infected badgers, Cheeseman *et al.* (1985) found that samples taken when alive were positive for *M. bovis* for only two of the animals, whereas post mortem samples were positive for seven. This suggests a ratio of infected-but-not-infectious to infectious animals in the order of 3:1 or 4:1.

(*c*) *Disease-induced mortality*

Gallagher & Nelson (1979) reported that tuberculosis infection was responsible for 40% of all natural deaths in one badger population in Gloucestershire and Cheeseman *et al.* (1989) found that tuberculosis accounted for 30% of natural deaths in the same population over a 15-year period (1972–1987). As a cause of mortality for the population as a whole, bovine tuberculosis was of only minor importance due to the preponderance of road accidents (which accounted for 58% of all deaths). However, of 149 badgers autopsied from this population over the period 1977–1985 inclusive, 39 animals were found to be infected and tuberculosis had been the direct cause of death in 15 cases (Cheeseman *et al.* 1988*b*). In addition, four of the infected animals were found *in extremis* and it was believed that tuberculosis would soon have caused the death of these animals. If these four cases are included, tuberculosis accounted for 48% of all deaths of infected animals. It is assumed in the model, as in others describing the dynamics of bovine tuberculosis in wildlife hosts (Anderson & Trewhella 1985; Barlow 1991*a*, 1993), that disease-induced mortality acts only on infectious individuals. Although a badger has been recorded as surviving for over three years while actively excreting *M. bovis* (Little *et al.* 1982), this is probably a rare occurrence. Therefore, it is assumed that 95% of infectious badgers die before reaching the end of their

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third year of infection, and that disease-induced mortality acts uniformly throughout this period. This equates to a disease-induced mortality of 63% per annum for infectious individuals.

(*d*) *Transmission*

(i) *Intragroup transmission*

The probability of intragroup transmission will be much higher than intergroup transmission due to individuals of the same group sharing setts. It is likely that the respiratory route is the main route of infection in badgers (Cheeseman *et al.* 1989), as many animals sharing the same limited volume of enclosed air in a sett is an ideal situation for the spread of a respiratory disease such as bovine tuberculosis (Gallagher *et al.* 1976). Some workers have argued that there may be a relatively high level of pseudo-vertical transmission of bovine tuberculosis from sows to their cubs (e.g. Cheeseman *et al.* 1981, 1988*b*) and if this was the case, transmission from sows to their cubs would be expected to peak in February-April when they are still maintaining close contact. However, there are no data to suggest that a substantial degree of pseudo-vertical transmission occurs. Data from badgers caught as part of MAFF control operations over the period 1982–1993 inclusive (R. Clifton-Hadley, personal communication) show a positive relation between the prevalence of bovine tuberculosis in cubs and females caught in the same year ($F = 8.80$, d.f. = 1,10, R^2(adjusted) = 41.5%, $p < 0.05$). However, there is also a strong relation between prevalence in cubs and adult males caught in the same year ($F = 13.60$, d.f. = 1,10, R^2 (adjusted) = 53.4%, $p < 0.01$), and no relation between prevalence levels in cubs and those in females caught in the previous year. Transmission to and from subdominant females may be at a minimum during the period February-April because subdominant females use annexe setts in preference to the main sett at this time of year (Cresswell *et al.* 1992). Thus due to the many different (possibly counteracting) transmission combinations, for these analyses all intragroup dyads were assumed to have the same probability of infection and no quarterly variations were incorporated.

(ii) *Intergroup transmission*

It is likely that boundary demarcation by scent-marking constitutes the main form of territory defence, and that costly agonistic encounters between groups generally will be avoided (Parker 1974; Maynard Smith & Parker 1976; White & Harris 1994). Most boundary patrolling is done by males, with adults patrolling much more than yearling males (Brown 1993). Adult and yearling females rarely patrol territory boundaries, and so most intergroup contacts at or near territory boundaries are likely to be between adult males, although there will be some contacts between adult and yearling males. Adults of both sexes may also make contact with adult males from other groups during the main mating period (February-March) when males visit adjacent territories. The relative importance of each of these transmission pathways is unknown, but overall adult male-adult male transmission is probably of greatest importance, followed by adult male-adult female transmission, and finally adult male-yearling male transmission. The higher prevalence of infection in males than females led Cheeseman *et al.* (1989) to suggest that male badgers may be more immunologically sensitive to *M. bovis* than females. However, there are no data to support this suggestion and differences in immunological sensitivity have not therefore been specifically included in the model: higher prevalences found in males probably simply reflect their greater involvement in activities likely to promote the transfer of the disease such as fighting. For the purposes of estimating actual figures for these different transmission processes, a ratio of 4:2:1 for adult male-adult male to adult male-adult female to adult male-yearling male pathways was assumed.

The extent of any intra-annual variation in intergroup contacts is unknown, but appears more marked than for intragroup contacts. Intergroup contacts are likely to peak in January-March, with a subsidiary peak in July-September. This largely reflects mating activity and mirrors the occurrence of large pre-ovulatory follicles recorded by Cresswell *et al.* (1992). Intergroup contacts are likely to be at a minimum in October-December, when badgers are less active (Brown 1993).

Thus to reflect these behavioural patterns in the model, the year was divided into the following quarters: January-March (winter/spring), April-June (spring/summer), July-September (summer/autumn) and October-December (autumn/winter). The different quarters were then assigned the following proportions of the annual infection probability: winter/spring, 0.4; spring/summer, 0.2; summer/autumn, 0.3; autumn/winter, 0.1.

(iii) *Contacts and infection*

With so many opportunities for transmission to occur between members of the same group underground, and with the chronic nature of the disease in badgers, it is perhaps surprising that the prevalence of tuberculosis in badger populations is generally low. This fact and the spatially clumped distribution of the disease in badger populations (e.g. Cheeseman *et al.* 1988*b*), suggest that bovine tuberculosis infection is not easily acquired or spread by badgers. Thus only a small proportion of contacts of susceptible animals with infectious ones will result in the transmission of infection. The probability of contacts between individuals is unknown, as is the probability of infection arising after a potentially infectious contact. Thus for the model, these values were estimated by matching the predictions of the model to field measurements of disease prevalence and spread. The choice of these values will be discussed in §5 after the description of the structure of the model.

4. THE MODEL

(*a*) *Framework*

The basic framework for the model was a main grid of 100 square cells, arranged in a 10 × 10 square. This size was chosen because it is probably close to the maximum

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number of badger groups that would occur contiguously in an area. Each grid cell represents a territory that contains a single social group of badgers. Each territory has four neighbouring groups sharing its long borders, and four at each corner. Permanent transfer of individuals between social groups occurs by dispersal, and infectious contacts are made by predetermined probabilities. Intergroup infectious contacts, dispersal and colonization can only occur between fully adjacent territories, not ones bordering at the corners. The main grid is surrounded by a further ring of 44 boundary cells, the dynamics of which are controlled by the same processes as operating on the rest of the grid. These boundary cells serve as a source of immigrants to the grid and a sink for emigrants, and are susceptible to disease in the same way as those on the main grid.

(*b*) *Classes of infection*

Three classes of infection are considered in the model: susceptible (X), infectious (Y), and infected-but-not-infectious, i.e. latent infection (H). Because processes operate on a group level, if G is the group size:

$$G = X + Y + H.$$

Susceptible individuals becoming infected pass to class H. Individuals of class H may become Y individuals based on a predetermined probability, and Y individuals may revert to H once more, again according to a predetermined probability:

$$X \rightarrow H \rightarrow Y.$$

(*c*) *Model structure*

The disease-free equilibrium group size in each cell on the grid is predetermined. It is assumed that it is dependent on the availability of resources, and is the level around which density-dependent effects operate. The model stores information in terms of the number of constituent individuals of each of the three age classes, their sex, and disease status within each social group.

The model consists of a main program and a series of subroutines which represent specific biological processes e.g. intergroup transmission, dispersal, disease-induced mortality. These subroutines are called from the main programme for each individual from each badger social group in turn according to specific conditions laid down in the main program. The subroutines therefore constitute transitional processes i.e. the means by which the composition of each group can be altered. After each subroutine, running totals of group composition are calculated in the main program. When the main program has finished for all the grid cells in a specific time period x, these running totals are converted to the new group composition totals at time $x+1$, before the main program is run again.

The model runs on a quarterly basis using the quarters described above. Cubs are introduced to each group in the winter/spring quarter according to the function describing fecundity. At the start of this quarter, any cubs remaining from the previous year become yearlings, and any yearlings remaining from the previous year become adults. Cubs are only born into groups containing one or more adult or yearling females.

The second process is intragroup infection. If the number of susceptible and infectious individuals are both greater than zero, whether each infectious individual makes an infectious contact with a susceptible individual is calculated according to the intragroup infection probability. This is done for each susceptible individual in turn. The third process is intergroup infection. This works in a similar way as that for intragroup infection, but only takes account of adult male and female and yearling male behaviour. Yearling females and cubs are ignored because they rarely participate in territorial defence. These transmission processes result in a non-linear relation between infection rate and the density of infectious individuals, because as the number of infectious animals in the population increases, the number of susceptibles decreases; this non-linearity is exacerbated by the high degree of spatial heterogeneity of the badger population. After the processes of infection, the infected individuals in each group are subjected to a probability of transfer in their infectious state, i.e. infected individuals becoming infectious and infectious ones becoming latent.

Dispersal is considered next; only adult males and adult females can disperse. Dispersal from a specific group can be to any of the four fully adjacent groups and the number of animals dispersing from a group is not limited. Colonization of empty territories is considered next. As with dispersers, colonizers can only come from the four fully adjacent groups, and the number of animals colonizing an empty range in any time period is not artificially limited. Colonizers may be infected or uninfected animals.

The final processes are disease-induced mortality and natural mortality, and these are again calculated on an individual basis. Only infectious (Y) individuals are subject to disease-induced mortality. All classes of individuals are subject to natural mortality; for infectious individuals, natural and disease-induced mortalities are additive.

The parameters used in the model are listed in table 2 and the structure of the model is illustrated in figure 2.

(*d*) *Comparison of assumptions with those of previous models*

Anderson & Trewhella (1985) found no quantitative evidence of any association between fecundity and/or mortality with population density, and assumed that density-dependence acted on fecundity rather than adult or cub mortality. The loss of reproductive potential among groups in high density populations (Cresswell *et al.* 1992), combined with the very high cub mortality rates recorded, suggest that fecundity and cub mortality interact to regulate the population density at the group level. However, the reduction in *per capita* fecundity in high density populations does not appear to constitute true regulatory density-depen-

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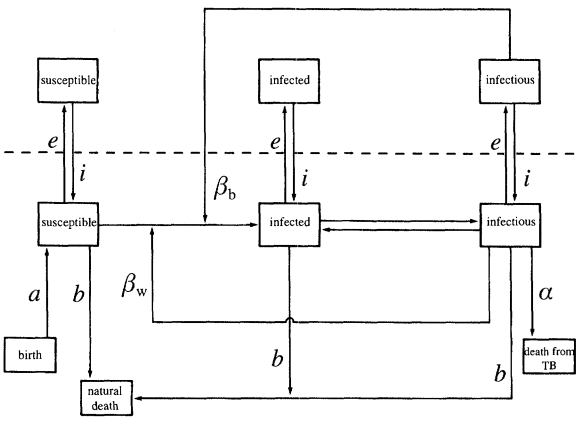

Figure 2. Diagrammatic representation of the structure of the model. The territory boundary between adjacent groups is represented by the dotted line: a, birth; b, natural mortality; α, disease-induced mortality; β_w, intragroup infection; β_b, intergroup infection; i, immigration; e, emigration.

Table 2. *Functions and parameter values used in the standard form of the model*

(Percentages are per capita per annum. P_{cubs} is the number of cubs produced per social group, and a_f is the number of adult females in the group.)

fecundity	$P_{cubs} = 0.60 + 0.63\,a_f$
sex ratio of cubs	1:1
standard adult mortality	19%
standard cub mortality	density dependent, 19–70%
dispersal probability	males, 6%; females, 2%
adult colonization probability	2.5%
disease-induced mortality	63%
transfer from infectious to latent disease state	59%
transfer from latent to infectious disease state	48%

dence. Anderson & Trewhella (1985) also assumed that any density-dependent processes exerted a strong influence on population behaviour. However, the fact that group sizes fluctuate considerably from year to year (e.g. Cheeseman *et al.* 1987) suggests that any density-dependence is relatively weak. The assumptions made by Anderson & Trewhella (1985) concerning the course of infection in individual animals are generally the same as those made here, although in one of the more complex versions of their model they incorporated a carrier class of individuals in addition to the susceptible, infected-but-not-infectious (latent infection) and infectious classes. Carrier individuals show no evidence of any symptoms, and are therefore subject to natural mortality alone (not disease-induced mortality) and yet actively excrete infective bacilli. The existence of a carrier state for badgers and bovine tuberculosis is virtually impossible to verify, and is therefore omitted from this model. However, it does occur in many bacterial infections and, due to the absence of disease-induced mortality, it will act to enhance the ability of such infections to persist endemically in low density populations.

Bentil & Murray (1993) based the values of the initial parameters they used in their model on those of Anderson & Trewhella (1985). The priority of Bentil & Murray's (1993) approach was to demonstrate how numerical methods might help in generating values for parameters that were not available from field data rather than construct the most realistic model possible. However, they were forced to make several assumptions to replicate the findings of Anderson & Trewhella (1985); many of these were either unfounded or unrealistic. They ignored density-dependence, and artificially maintained the population at a constant size by setting the birth rate equal to the death rate. They also assumed that disease-induced mortality was negligible compared with natural mortality, and effectively disregarded it in their model. In addition, they included an immune class of animals, for which there are as yet no convincing data.

5. ESTIMATION OF UNKNOWN EPIDEMIOLOGICAL PARAMETERS

(*a*) *Probabilities of an infection becoming latent and reverting to an infectious state*

The only quantified data on transfers between the different infected states are presented by Wilesmith (1991). Of seven individuals that were captured and identified as excreting *M. bovis* in 1989 and recaptured the next year, three were still excreting *M. bovis*. However, four animals had ceased to excrete the bacilli, and the infection had transferred to an inactive state. If this proportion (60%) is taken as an approximate annual rate of transfer from an infectious to an inactive state, then the equivalent seasonal probability of transfer is 0.20. Because the ratio of inactive to infectious individuals is around 3:1 or 4:1, the seasonal probability of transfer from the inactive to the infectious state was derived from this figure.

Although there are more animals with inactive infections than infectious animals, this does not necessarily imply that the rate of transfer from an infectious to an inactive state exceeds that from an inactive to an infectious one due to the higher death rate of infectious animals. However, the fact that infected badgers do not continually fluctuate between infectious and inactive states indicates that the probabilities of transfer in either direction are low. To derive the probabilities of transfer between the different states, simulations were run using a variety of different inter- and intragroup contact probabilities over a range of seasonal probabilities of transfer in either direction between 0.01–0.50. Over these ranges of values, the combination of seasonal probabilities of 0.20 and 0.15 for infectious to inactive transfers and inactive to infectious transfers respectively consistently gave the closest ratio of infected to infectious individuals of between 3:1 and 4:1. These probabilities were therefore used in the model.

(*b*) *Intra- and intergroup infection probabilities*

The intra- and intergroup infection probabilities used in the model include both the probability of contact between two individuals and the probability of infection occurring during a contact. Intra- and intergroup infection probabilities interact to produce the observed disease prevalence, and thus where both

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are unknown, they must be considered together in any attempt at quantification.

As bovine tuberculosis in badgers is chronic in nature and does not spread rapidly between groups, prevalence may show much local variation and be very dependent on the initial pattern of infection. The only data on the spatial dynamics of bovine tuberculosis in a badger population come from one long-term study in Gloucestershire, where the temporal spread of infection between social groups was slow and restricted (Cheeseman *et al.* 1988*b*). Infection was initially present in four groups in the southwest of the study area; five years later the infection was still present in just four or five groups. It had not spread significantly beyond those groups originally infected, and the prevalence of infection over the ten most studied groups never exceeded 8.1%. Disease-induced depression of the population was negligible, probably because of the low overall prevalence levels resulting from the restricted spread of the disease.

To estimate a range of probable values for inter- and intragroup infection probabilities, the initial pattern of infection in the Gloucestershire population was replicated by introducing one infectious male and one infectious female in each of the central four groups on the grid. The model was then run for five years at different combinations of inter- and intragroup infection probabilities, each simulation being repeated 20 times. The intergroup adult male-adult male infection probabilities ranged from 0.005–0.05, and the intragroup infection probabilities ranged from 0.05–0.5. The annual mean size of the ten most intensively studied groups in the Gloucestershire study area over this period ranged from 6.8–8.1 (Cheeseman *et al.* 1988*b*), so an equilibrium group size of eight adults and yearlings was used in the model. Cheeseman *et al.* (1988*b*) expressed population size and disease prevalence over the ten most studied groups. Thus to make our figures approximately comparable, we expressed population and prevalence in terms of the central nine groups (i.e. the four initially infected ones plus five adjacent groups to make a square, with the originally infected groups occupying the bottom left-hand corner). The following measures of the dynamics of the disease in the simulations were quantified for comparison with the observational data to obtain an indication of the probable range of combinations of inter- and intragroup infection probabilities for the disease in badgers.

1. The mean number of infected groups within the central nine groups after five years (maximum nine).
2. The mean number of infected groups outside the central nine groups after five years (maximum 91).
3. The percentage of simulations in which the disease persisted throughout the five years in the central nine groups and in which five or less groups outside these central groups were infected after five years.
4. The mean prevalence of infection in the infected groups within the central nine groups after five years.
5. The mean overall prevalence of infection in the central nine groups after five years.
6. The mean population size in the central nine groups after five years.

The relations between these variables and the different combinations of intra- and intergroup infection probabilities used are shown in figures 3*a*-*f*. The numbers of infected groups inside and outside the central nine groups after five years were both largely determined by the intergroup infection probability (see figures 3*a*,*b*), with higher intergroup infection probabilities resulting in greater numbers of infected groups. The probability of localized persistence of the disease with only limited or no spread to surrounding groups (see figure 3*c*) was also determined largely by the intergroup infection probability. It was less than 30% for an intergroup infection probability above 0.01, and less than 10% for an intergroup infection probability above 0.03. The mean prevalence of infection in the infected groups within the central nine groups after five years (see figure 3*d*) was controlled by the intragroup infection probability, being greater than 50% with an intragroup infection probability greater than 0.2. However, the mean total prevalence throughout the nine central groups (see figure 3*e*) was determined by both the intra- and intergroup infection probabilities, being highest where both inter- and intragroup infection probabilities were greatest. The mean population size over the nine central groups (see figure 3*f*) was also determined by both inter- and intragroup infection probabilities, but showed the reverse of the relation in figure 3*e*, population depression due to the disease being greatest at the highest infection probabilities.

The simulations showed that combinations of an intergroup adult male-adult male infection probability of 0.001 or 0.005 and an intragroup infection probability of 0.2 were most likely to replicate the precise disease pattern recorded by Cheeseman *et al.* (1988*b*), resulting in 30% and 35% probabilities respectively of the disease persisting with some limited spread (to less than five groups outside the central nine) over the five-year period, and 90% and 70% probabilities of the disease persisting with limited or no spread. However, the precise values of inter- and intragroup infection found in this high density, well-structured population are unlikely to be the same as those found in lower density, less structured populations, or in those populations which have been subjected to disturbance. These are all likely to be characteristics of the badger populations in those areas subjected to control operations since 1975. Such populations are likely to have relatively higher rates of intergroup contacts involving fighting, and therefore increased net infection probabilities. For these reasons, a wider range of infection probabilities was used to investigate disease dynamics. The chosen combinations were an intergroup adult male-adult male infection probability of between 0.005–0.01 and an intragroup infection probability of between 0.05–0.2. With these combinations, there was still some probability (between 5–20%) of the pattern of disease in the Gloucestershire population being replicated. However, the higher overall probability of disease spread is likely to be more

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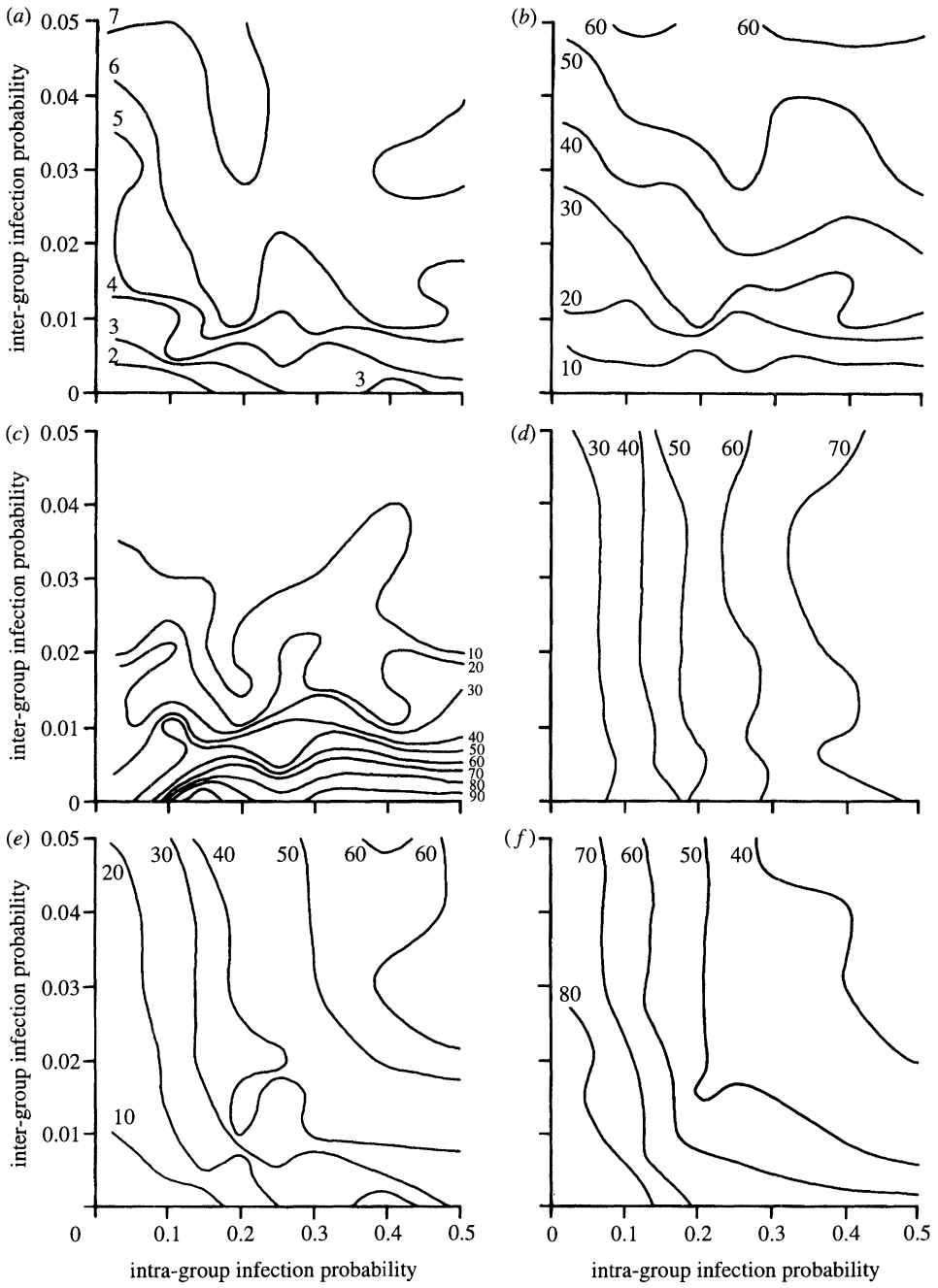

Figure 3. Isopleth maps showing the passage of bovine tuberculosis infection through a badger population with an equilibrium group size of eight adults and yearlings over five years after the introduction of one infectious male and one infectious female in each of the central four groups on the grid. The model was run at different combinations of inter- and intragroup infection probabilities. The isopleths represent the mean values from 20 simulations for (*a*) the mean number of infected groups within the central nine groups after five years (maximum nine), (*b*) the mean number of infected groups outside the central nine groups after five years (maximum 91), (*c*) the percentage of simulations in which the disease persisted throughout the five years in the central nine groups and in which five or less groups outside these central groups were infected after five years, (*d*) the mean prevalence of infection (%) in the infected groups within the central nine groups after five years, (*e*) the mean overall prevalence of infection (%) in the central nine groups after five years and (*f*) the mean population size over the central nine groups after five years.

representative of the situation in less structured populations. It is also important to consider the potential effects of disturbance, since this may decrease social stability and hence increase both intergroup movements and infective contacts. Both these effects are examined in later sections.

Although the intragroup infection probability used may be only five times greater than the intergroup adult male-adult male probability, these infection probabilities represent a combination of contact probability and the probability of disease transfer after such a contact. It is probable that the difference between the inter- and intragroup probabilities is less pronounced than might be expected due to the nature of the most likely contacts. Within a group, most contacts will be social and within the sett, although there may

be some aggressive encounters, especially between dominant and subdominant animals. In contrast, although intergroup encounters as a whole will be much rarer, a greater majority of those that do occur would be expected to be aggressive ones, and bite wounds are likely to be relatively more frequent. Transmission by bite wounds is likely to be considerably more efficient than inhalation or ingestion of bacilli during grooming and other non-aggressive contacts.

6. RESULTS

(*a*) *The existence of a threshold mean group size for endemic infection*

When bovine tuberculosis was widespread in cattle in the early part of this century, it is likely that badgers were exposed to the infection throughout the whole of the southwest, as well as in much of the rest of Britain. Anderson & Trewhella (1985) predicted that the disease could exist at equilibrium in the badger population. Their model suggested that the prevalence showed damped oscillations until an equilibrium was achieved after 30–40 years, after which disease prevalence was constant at approximately 18%. On this basis, and the fact that tuberculous badgers have been recorded from most counties of Britain, Cheeseman *et al.* (1989) argued that bovine tuberculosis was endemic in the British badger population.

The first part of the current study was to investigate whether it was theoretically possible for the disease to persist endemically within the badger population in view of the spatial structure of the badger population and to estimate the theoretical threshold equilibrium group size required for this to occur. A true endemic disease equilibrium will never occur in a spatially confined stochastic model such as this one, because eventually the host population and/or the disease will decline to extinction. However, for the purposes of the current study, endemic infection was deemed to exist where the relation between the prevalence of the disease and the abundance of the badger population had reached an apparent equilibrium (i.e. minimal temporal changes in both parameters) within a time period of 100 years.

To investigate whether such an apparent equilibrium could theoretically exist, the infection was introduced into all groups on the grid in the form of one infected male in each group. This precise pattern

Table 3. *The effects of differences in the infection probabilities and equilibrium group sizes on the rate of extinction of bovine tuberculosis after homogeneous infection of the grid.*

(Fifty simulations of the model were run over a period of 100 years for each combination of intra- and intergroup infection probabilities, using disease-free equilibrium group sizes of four, six, eight and ten adults and yearlings.)

Equilibrium group size	Intergroup infection probability	Intragroup infection probability	Year class in which infection died out					
			0–19	20–39	40–59	60–79	80–99	100+
4	0.005	0.05	8	31	0	0	0	11
6			0	15	5	0	1	29
8			0	0	2	0	0	48
10			0	0	0	0	0	50
4	0.01	0.05	6	19	2	0	0	23
6			0	4	4	1	0	41
8			0	0	0	0	0	50
10			0	0	0	0	0	50
4	0.005	0.10	0	19	4	0	0	27
6			0	1	7	4	1	37
8			0	0	0	0	0	50
10			0	0	0	0	0	50
4	0.01	0.10	0	7	5	1	1	36
6			0	0	0	0	0	50
8			0	0	0	0	1	49
10			0	0	0	0	0	50
4	0.005	0.15	0	9	9	4	11	17
6			0	0	3	5	10	32
8			0	0	4	3	5	38
10			0	0	1	4	3	42
4	0.01	0.15	0	2	6	8	8	26
6			0	0	0	1	11	38
8			0	0	1	5	8	36
10			0	0	0	4	3	43
4	0.005	0.20	0	7	14	11	11	7
6			0	1	5	12	6	25
8			0	2	4	7	9	28
10			0	1	5	6	10	28
4	0.01	0.20	0	5	10	7	15	13
6			0	0	6	14	14	16
8			0	0	5	5	11	29
10			0	0	3	6	14	27

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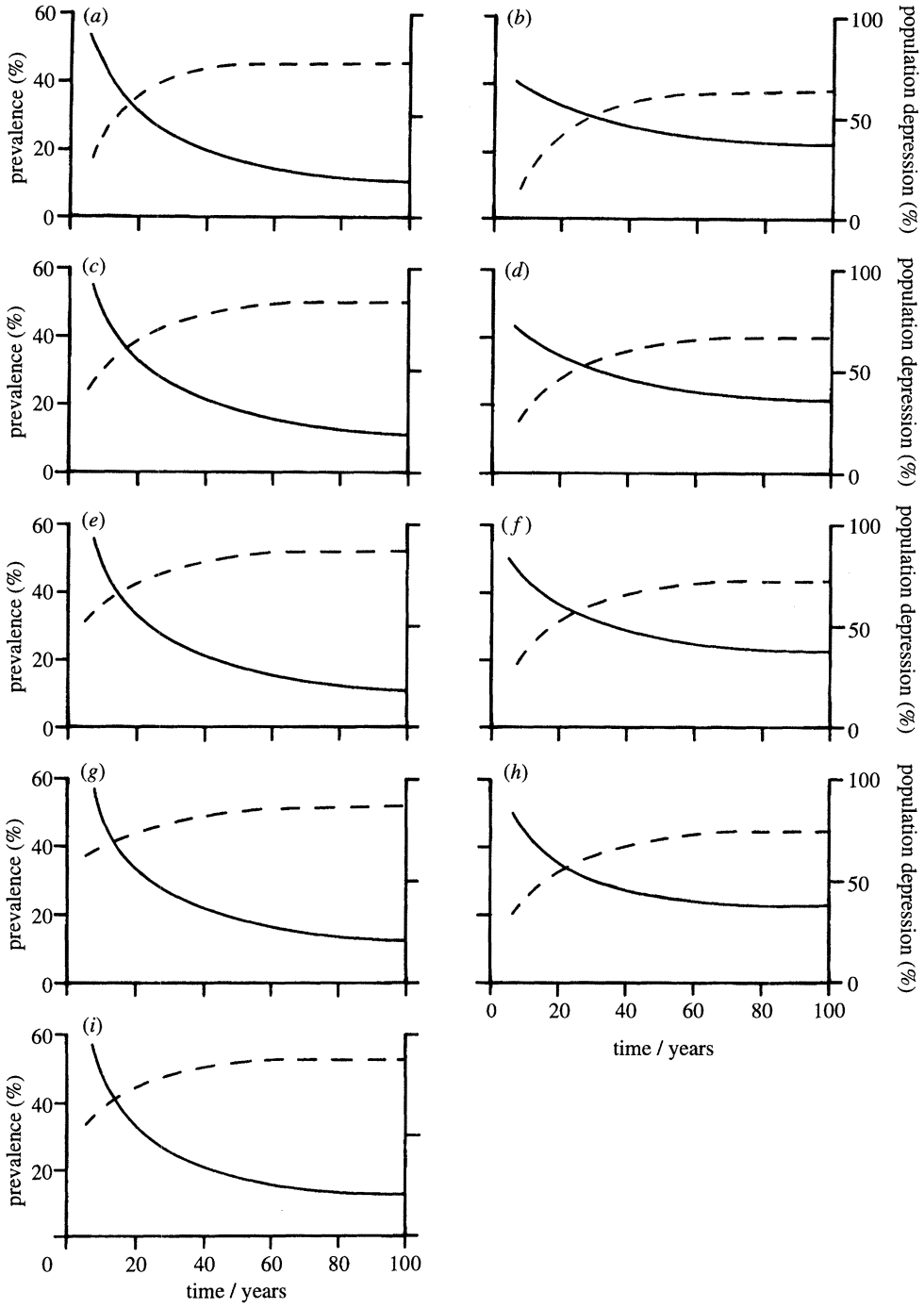

Figure 4. The relation between disease-free equilibrium group size, disease prevalence and population depression (below disease-free level) in the presence of the disease at apparent equilibrium for those combinations of group size, intergroup and intragroup infection probability for which the disease was likely to persist throughout the 100-year period after an initial homogeneous infection of the grid ($p \geqslant 0.95$) using the standard parameter values (see table 2). The lines represent the means of 50 simulations for (*a*) $G_{eqm} = 6$, $\beta_b = 0.01$, $\beta_w = 0.10$, (*b*) $G_{eqm} = 8$, $\beta_b = 0.005$, $\beta_w = 0.05$, (*c*) $G_{eqm} = 8$, $\beta_b = 0.005$, $\beta_w = 0.10$, (*d*) $G_{eqm} = 8$, $\beta_b = 0.01$, $\beta_w = 0.05$, (*e*) $G_{eqm} = 8$, $\beta_b = 0.01$, $\beta_w = 0.10$, (*f*) $G_{eqm} = 10$, $\beta_b = 0.005$, $\beta_w = 0.05$, (*g*) $G_{eqm} = 10$, $\beta_b = 0.005$, $\beta_w = 0.10$, (*h*) $G_{eqm} = 10$, $\beta_b = 0.01$, $\beta_w = 0.05$, and (*i*) $G_{eqm} = 10$, $\beta_b = 0.01$, $\beta_w = 0.10$, where G_{eqm} is the disease-free equilibrium group size (adults and yearlings), β_b is the standard intergroup infection probability (see text) and β_w is the intragroup infection probability. Disease prevalence is indicated by the solid line, population depression by the dashed line.

of infection will never be seen in the field. However, it is an approximation to the initial widespread exposure of the badger population to infection from cattle, and so is appropriate for initial investigations into the theoretical possibility of the disease becoming endemic within the badger population.

Standard intergroup infection probabilities of 0.005–0.01 were used in combination with intragroup infection probabilities of 0.05, 0.10, 0.15 and 0.20, and 50 simulations of the model were run over a period of 100 years for each combination of intra- and intergroup infection probabilities, using disease-free equilibrium group sizes of four, six, eight and ten adults and yearlings. Table 3 shows the effects of differences in the

infection probabilities and equilibrium group sizes on the rate of extinction of the disease. The probability of disease persistence increased with increasing group size and decreased at an intragroup infection probability of 0.15 or above. Larger group sizes resulted in a greater probability of disease persistence at higher inter- and intragroup infection probabilities. However, these results indicate that bovine tuberculosis has the potential to persist in the badger population for a long time, even at small group sizes and at low prevalences, without any reinfection being required.

(*b*) *Badger density, disease prevalence and badger population depression*

Two of the key predictions from the model of Anderson & Trewhella (1985) were that the prevalence of bovine tuberculosis in the badger population would be related to badger density and that the disease would significantly depress the badger population below disease-free levels. These authors found that the extent of this effect varied depending on the threshold population density and the disease-free environmental carrying capacity, although it was similar for all densities above ten badgers (adults and yearlings) km^{-2}, being between 40 and 85% at equilibrium.

The relation between mean disease-free equilibrium group size, disease prevalence and population depression in the presence of the disease at apparent equilibrium, for those combinations of inter- and intragroup infection probabilities for which the infection persisted at apparent equilibrium at the end of the 100-year period in more than 95% of the simulations, are shown in figures 4*a–i*. Disease prevalence was controlled mainly by the intragroup infection probability, being lower at higher intragroup infection probabilities for a certain intergroup infection probability. However, there was no clear correlation between disease prevalence and population depression.

Although it was possible for the disease to reach an apparent equilibrium within a badger population with a mean disease-free equilibrium group size of six adults and yearlings, an apparent equilibrium was most likely to occur in populations with mean disease-free equilibrium group sizes of eight or more for intragroup infection probabilities between 0.05–0.10 and intergroup infection probabilities between 0.005–0.01. Under these circumstances, prevalence levels ranged between 11–22%, and the population density was depressed by between 66–86% below disease-free levels. Population depression was greater for higher intragroup infection probabilities, but was unaffected by equilibrium disease-free group size or the level of intergroup infection probability.

(*c*) *The spatial dynamics of endemic infection*

Although previous non-spatial models have been able to show that bovine tuberculosis can persist endemically in badger populations, they were unable to reveal anything of the spatial dynamics of the disease in an endemic state. This can be done with the current model by observing the spatial distribution of infected individuals throughout the grid over time.

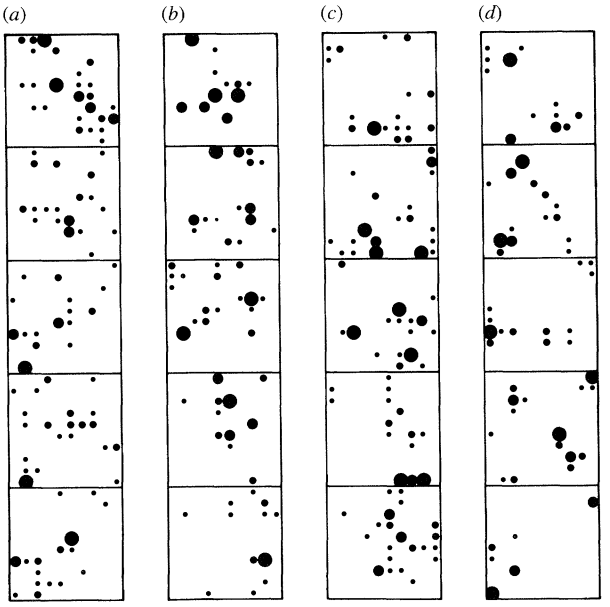

Figure 5. The spatio-temporal dynamics of endemic bovine tuberculosis infection in a homogeneous badger population. The figures show the distribution of badger groups with more than one infected individual throughout the standard 10 x 10 grid after 60 (row 1), 70 (row 2), 80 (row 3), 90 (row 4), and 100 (row 5) years after an initially homogeneous infection across the population. Infected groups are shown by solid circles; increasing circle size denotes 1, 2, 3, 4 and 5 or more infected animals in the group. The examples shown are for a disease-free equilibrium group size (G_{eqm}) of eight adults and yearlings with (*a*) $\beta_b = 0.005$, $\beta_w = 0.05$, (*b*) $\beta_b = 0.005$, $\beta_w = 0.10$, (*c*) $\beta_b = 0.01$, $\beta_w = 0.05$, and (*d*) $\beta_b = 0.01$, $\beta_w = 0.10$, where β_b is the standard intergroup infection probability (see text) and β_w is the intragroup infection probability.

In all of the above simulations, the disease followed a common spatial pattern. From the initial homogeneous state, it gradually became clustered over time to form discrete pockets of infection. These pockets of infection formed in different spatial locations throughout the grid in different simulations, and shifted gradually in location over time. Patches of disease were more distinct and more spatio-temporally stable where the intragroup infection probability was higher relative to the intergroup infection probability. Some examples of this phenomenon for an equilibrium group size of eight adults and yearlings under different combinations of inter- and intragroup infection probabilities are shown in figures 5*a–d*.

(*d*) *Effects of population density on disease spread and persistence*

Because it is believed that bovine tuberculosis is endemic in the badger population in parts of southwest England, and the results above indicate that this is only likely to occur with an intragroup infection probability between 0.05–0.10 and an intergroup infection probability between 0.005–0.01, these infection probabilities were used as the standard values for further investigations into the likely rate and pattern of spread of the disease within the badger population. To investigate the effects of population density on disease

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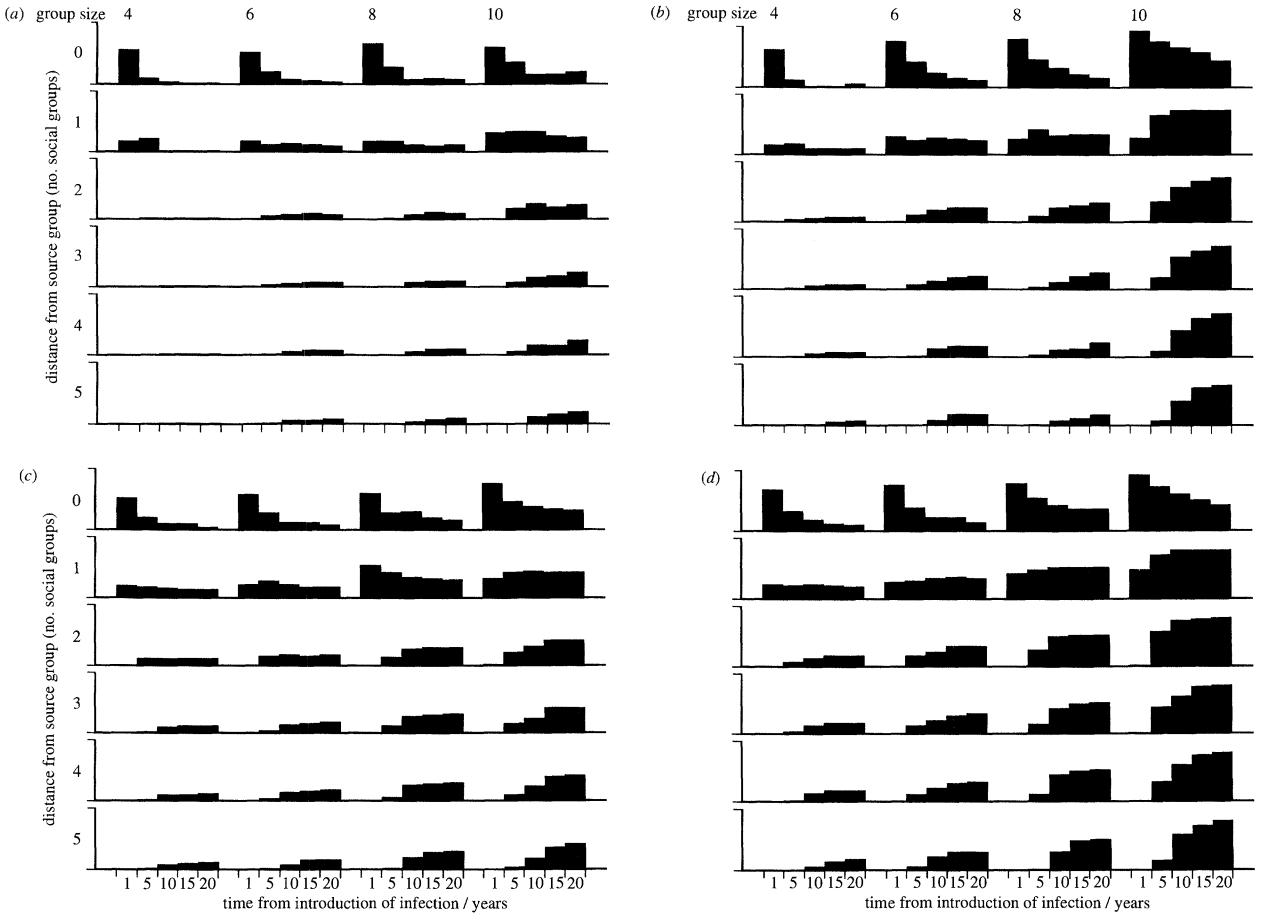

Figure 6. The effects of group size (adults and yearlings) on the spread of infection from a point source under different inter- and intragroup infection probabilities. The bars represent the probability, $p(\mathrm{i})$, of the infection being present in a ring of groups surrounding the source group at a given distance (measured in social groups away from the source group) in a particular year following the introduction of the infection. The full height of the vertical axis on each graph represents a probability, $p(\mathrm{i})$, of 1.0. Results are the means of 50 simulations for (*a*) $\beta_b = 0.005$, $\beta_w = 0.05$, (*b*) $\beta_b = 0.005$, $\beta_w = 0.10$, (*c*) $\beta_b = 0.01$, $\beta_w = 0.05$, and (*d*) $\beta_b = 0.01$, $\beta_w = 0.10$, where β_b is the standard intergroup infection probability (see text) and β_w is the intragroup infection probability.

spread, a point source of infection was set up in a central cell on the grid, and the spread of infection from this point was monitored. As bovine tuberculosis is no longer widespread in either badgers or cattle in the southwest, a point source rather than a uniform infection represents the most probable initial form of exposure of an uninfected badger population to the disease that is now likely to occur.

At the start of each simulation, one male badger in a central grid cell was infected. Simulations were conducted over a period of 20 years using combinations of inter- and intragroup probabilities of 0.005 and 0.05, 0.005 and 0.10, 0.01 and 0.05, and 0.01 and 0.10 respectively, for group sizes of four, six, eight and ten adults and yearlings. Fifty simulations were repeated for each group size.

The spread of infection was measured by examining infection in concentric rings of social groups around the group initially infected (the source group). Thus there were eight groups in the first concentric ring (immediately adjacent to the source group), 16 in the second, 24 in the third, 32 in the fourth, and 40 in the fifth. This method of analysing the rate of spread of the infection from a point source was chosen in preference to a simple assessment of the number of intergroup contacts away from the source group because of the offset-grid pattern of territories found in most badger populations. For each population density, the number of simulations in which each ring of grid cells was infected in each particular year was used as a measure of the probability of spread and persistence of the infection.

The results of these simulations are illustrated in figures 6*a–d*. At the lowest combination of inter- and intragroup infection probabilities considered (0.005 and 0.05 respectively, see figure 6*a*), the probability of the disease persisting to year 20 was 0.10 or less for disease-free equilibrium group sizes of four, six and eight adults and yearlings, although this rose to 0.24 for an equilibrium group size of ten. For the other three combinations of inter- and intragroup infection probabilities, it was clear that as group size increased, the probability of spread of the disease from the source group into the surrounding groups also increased. The most marked difference between the group sizes considered occurred between a group size of six and eight adults and yearlings. For a mean group size of six adults and yearlings, the mean probability of the disease persisting to year 20 was 0.16 ± 0.04 ($n = 3$) for the first three combinations of inter- and intragroup

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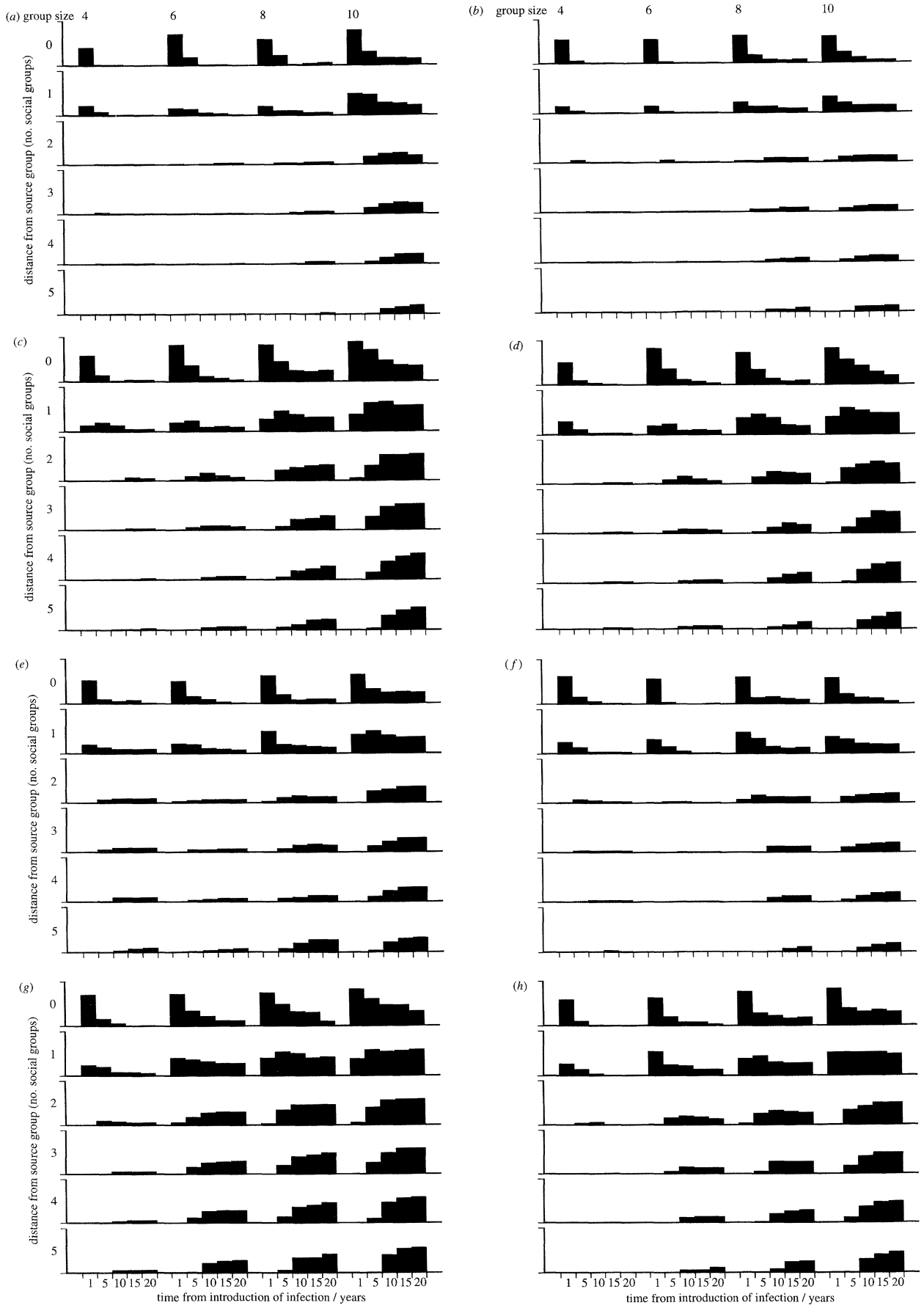

Figure 7. The effects of increased intergroup movements on the spread of infection from a point source for different equilibrium group sizes under different inter- and intragroup infection probabilities. The bars represent the probability, $p(\mathrm{i})$, of the infection being present in a ring of groups surrounding the source group at a given distance (measured in social groups away from the source group) in a particular year after the introduction of the infection.

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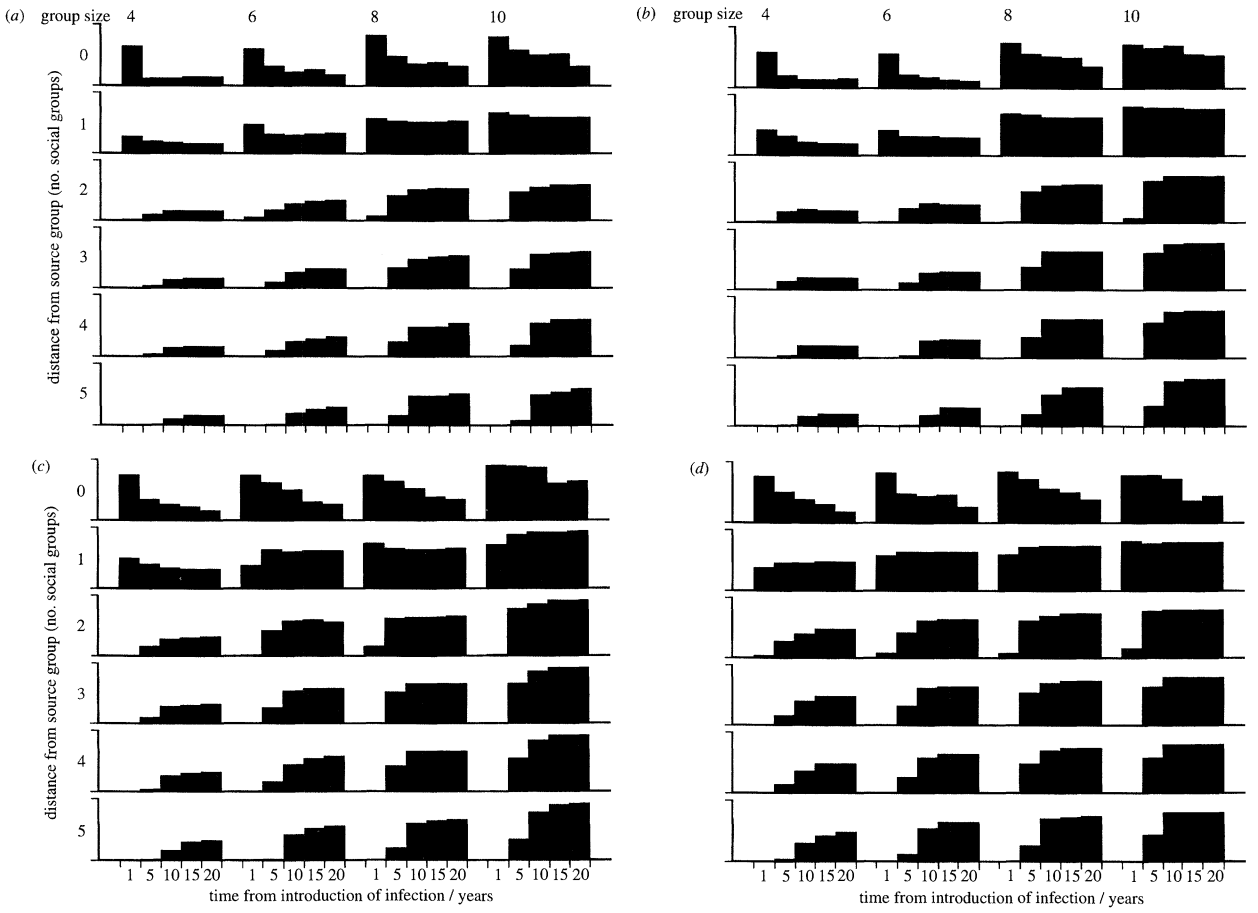

Figure 8. The effects of increased intergroup infection on the spread of infection from a point source for different equilibrium group sizes (adults and yearlings) under different intragroup infection probabilities. The bars represent the probability, p(i), of the infection being present in a ring of groups surrounding the source group at a given distance (measured in social groups away from the source group) in a particular year after the introduction of the infection. The full height of the vertical axis on each graph represents a probability, p(i), of 1.0. Results are the means of 50 simulations for (*a*) $\beta_b = 0.021$, $\beta_w = 0.05$, (*b*) $\beta_b = 0.031$, $\beta_w = 0.05$, (*c*) $\beta_b = 0.021$, $\beta_w = 0.10$, and (*d*) $\beta_b = 0.031$, $\beta_w = 0.10$, where β_b is the standard intergroup infection probability (see text) and β_w is the intragroup infection probability.

infection probabilities, although this rose to 0.33 for an intergroup infection probability of 0.01 and an intragroup infection probability of 0.10. However, for a group size of eight adults and yearlings, for intermediate combinations of infectious probabilities, the mean probability of the disease persisting to year 20 was 0.29 ± 0.02 ($n = 2$), and for the highest combination (see figure 6*d*) it reached 0.52. This difference in the probability of disease spread between equilibrium group sizes of six and eight adults and yearlings reflected the threshold disease-free equilibrium group size for disease spread and persistence. For a group size of ten adults and yearlings, the probability of the disease persisting to year 20 was further increased. For the three highest combinations of infection probabilities for a group size of ten adults and yearlings (see figures 6*b–d*), the probability of persistence averaged 0.65 ± 0.12 ($n = 3$), ranging between 0.40 and 0.80.

(*e*) *Effects of increased intergroup movement on disease spread and persistence*

To examine the effect of increased intergroup movements on disease spread, the model was run as in the previous section but the probabilities of dispersal were increased to two and three times the standard rates for males i.e. 30% and 45% (8.5% and 13.9% per quarter). Annual dispersal probabilities for females were maintained at one third of the level of those for males. The effects of these changes were modelled for populations with mean group sizes of four, six, eight

The full height of the vertical axis on each graph represents a probability, p(i), of 1.0. Results are the means of 50 simulations for (*a*) $\beta_b = 0.005$, $\beta_w = 0.05$, $d_m = 0.085$, $d_f = 0.026$, (*b*) $\beta_b = 0.005$, $\beta_w = 0.05$, $d_m = 0.139$, $d_f = 0.040$, (*c*) $\beta_b = 0.005$, $\beta_w = 0.10$, $d_m = 0.085$, $d_f = 0.026$, (*d*) $\beta_b = 0.005$, $\beta_w = 0.10$, $d_m = 0.139$, $d_f = 0.040$, (*e*) $\beta_b = 0.01$, $\beta_w = 0.05$, $d_m = 0.085$, $d_f = 0.026$, (f) $\beta_b = 0.01$, $\beta_w = 0.05$, $d_m = 0.139$, $d_f = 0.040$, (*g*) $\beta_b = 0.01$, $\beta_w = 0.10$, $d_m = 0.085$, $d_f = 0.026$, and (*h*) $\beta_b = 0.01$, $\beta_w = 0.10$, $d_m = 0.139$, $d_f = 0.040$, where β_b is the standard intergroup infection probability (see text), β_w is the intragroup infection probability, d_m is the male dispersal probability and d_f is the female dispersal probability.

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Figure 9. The effects of increased intergroup infection and movement on the spread of infection from a point source for different equilibrium group sizes (adults and yearlings) under different intragroup infection probabilities. The bars represent the probability, $p(\mathrm{i})$, of the infection being present in a ring of groups surrounding the source group at a given distance (measured in social groups away from the source group) in a particular year after the introduction of the infection. The full height of the vertical axis on each graph represents a probability, $p(\mathrm{i})$, of 1.0. Results are

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and ten adults and yearlings; the results are shown in figures 7*a–h*.

The effects of increased dispersal on the rate of disease spread can be seen by comparing figures 7*a–h* with figures 6*a–d*. Increased dispersal probabilities had no effect on the threshold group size for disease spread and persistence. Higher probabilities of dispersal resulted in a higher potential rate of disease spread, but this was more than compensated by a decrease in the probability of disease persistence, especially at higher movement probabilities. A twofold increase in dispersal (see figures 7*a*, *c*, *e*, *g*) resulted in probabilities of disease spread and persistence on average 0.06 ± 0.02 ($n = 8$) lower than those obtained from the standard dispersal rate for the lower intragroup infection probability considered, and 0.09 ± 0.03 ($n = 8$) lower than those from the standard rate for the higher intragroup infection probability. A threefold increase in dispersal (see figures 7*b*, *d*, *f*, *h*) led to a mean reduction in the probability of disease persistence of 0.10 ± 0.02 ($n = 8$) for the lower intragroup infection probability and 0.18 ± 0.03 ($n = 8$) for the higher intragroup infection probability.

(*f*) *Effects of increased intergroup infection on disease spread and persistence*

To examine the effect of increased intergroup infection on disease spread, the model was run as in § 6*d* with the same seasonal probabilities of dispersal and intragroup infection, but with seasonal intergroup infection probabilities increased two and three times to 0.021 and 0.031. The effects of these changes in the intergroup infection probabilities were modelled for populations with mean group sizes of four, six, eight and ten adults and yearlings; the results are shown in figures 8*a–d*.

For both intragroup infection probabilities considered, the greater the intergroup infection probability, the more likely the disease was to persist and spread to adjacent groups (compare figures 6*c* and 8*a–b* with 6*d* and 8*c–d*). The extent of this increased persistence was similar for both the intragroup infection probabilities considered. A twofold increase in the intergroup infection probability (see figures 6*a* and *c*) resulted in a mean increase in the probability of persistence of 0.15 ± 0.05 ($n = 8$) at the lower intragroup infection probability and 0.16 ± 0.02 ($n = 8$) at the higher one. The equivalent figures for a threefold increase in the intergroup infection probability (see figures 6*b* and *d*) were 0.22 ± 0.08 ($n = 8$) and 0.21 ± 0.07 ($n = 8$). The probability of disease spread and persistence from a point source for a mean equilibrium group size of four adults and yearlings at the higher intragroup infection level was raised from 0.18 to between 0.32 and 0.48, and that for a group size of six adults and yearlings from 0.33 to between 0.56 and 0.64. Thus an increase in the intergroup infection probability effectively decreased the threshold equilibrium group size and increased the probability of the disease persisting at lower mean group sizes to levels comparable with group sizes of eight and ten adults and yearlings under standard conditions of intergroup infection.

(*g*) *Effects of increased intergroup movement and infection on disease spread and persistence*

To examine the effect of both increased intergroup infection and movement on disease spread, the model was run as in the above section with seasonal intergroup infection probabilities of 0.021 and 0.031, but for each combination of intra- and intergroup infection probabilities considered, dispersal probabilities of two and three times the standard rates were used. Again, this was only done for the higher standard intergroup infection probability.

The results of these simulations are shown in figures 9*a–h* (compare figures 6*c* and *d*, 7*e–h* and 8*a–d*). For both the intragroup infection probabilities considered, an increase in intergroup movement and infection combined resulted in an increase in the probability of disease spread and persistence in all cases except when the probability of intergroup infection was increased twofold and that of movement was increased threefold (figures 9*b* and *f*). In these instances, the mean probability of disease spread and persistence was reduced by 0.03 ± 0.02 ($n = 8$).The mean increase over all other combinations of group size, inter- and intragroup infection and movement probabilities was 0.11 ± 0.02 ($n = 24$). The greatest increase of 0.18 ± 0.04 above that for the standard infection and movement probabilities was obtained for a threefold increase in infection combined with a two fold increase in movement (see figures 9*c*,*g*). For this particular combination of movement and infection, the increase is slightly greater than that obtained for an increase in the intergroup infection probability alone. However, for most combinations of inter- and intragroup infection probabilities, the increases in spread and persistence caused by increased movements and infection were not so pronounced as for increased infection alone.

7. DISCUSSION

One of the primary objectives in the construction of this model was to make it as biologically realistic as possible, using parameters that are relatively simple to

the means of 50 simulations for (*a*) $\beta_b = 0.021$, $\beta_w = 0.05$, $d_m = 0.085$, $d_f = 0.026$, (*b*) $\beta_b = 0.021$, $\beta_w = 0.05$, $d_m = 0.139$, $d_f = 0.040$, (*c*) $\beta_b = 0.031$, $\beta_w = 0.05$, $d_m = 0.085$, $d_f = 0.026$, (*d*) $\beta_b = 0.031$, $\beta_w = 0.05$, $d_m = 0.139$, $d_f = 0.040$, (*e*) $\beta_b = 0.021$, $\beta_w = 0.10$, $d_m = 0.085$, $d_f = 0.026$, (*f*) $\beta_b = 0.021$, $\beta_w = 0.10$, $d_m = 0.139$, $d_f = 0.040$, (*g*) $\beta_b = 0.031$, $\beta_w = 0.10$, $d_m = 0.085$, $d_f = 0.026$, and (h) $\beta_b = 0.031$, $\beta_w = 0.10$, $d_m = 0.139$, $d_f = 0.040$, where β_b is the standard intergroup infection probability (see text), β_w is the intragroup infection probability, d_m is the male dispersal probability and d_f is the female dispersal probability.

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measure in the wild. In their earlier modelling study, Anderson & Trewhella (1985) outlined several important areas for future research. Unfortunately, our understanding of the biology of bovine tuberculosis infection in badgers has not advanced significantly over the succeeding decade. There are still few published data on the ratio of infected-but-not-infectious to infectious individuals in badger populations, and the course of infection in individual animals. Another key component in understanding the epidemiology of bovine tuberculosis in badgers is the contact rate between healthy and infectious badgers, but no data for this currently exist. In this model the need for such data was circumvented by using infection probabilities that were estimated indirectly from real data. However, field measurement of contact rates between badgers is a priority, as is obtaining better quantified data on the behaviour of infectious animals, as there are always uncertainties when using contact rates between uninfected animals as a substitute for those between infectious and uninfected ones (White *et al.* 1995).

The validity of the predictions resulting from any model rely heavily on the quality of the available data. Although many of the data sets used to generate parameters for this model are quite extensive, a number of parameters originate from a single extremely high-density non-disturbed badger population. The wider applicability of the model, especially to low density populations, would undoubtedly benefit from any improvement in these basic data, either in terms of sample size or by the availability of data from different populations. Yet despite these potential limitations, the model replicates many of the observed spatial and temporal aspects of the disease in badger populations, and has greatly advanced our understanding of the disease epidemiology in the high-density badger populations in southwest England.

Anderson & Trewhella (1985) stated that there were no quantitative data for the existence of a threshold population density for bovine tuberculosis in badgers, although the structure of their model forced them to use some value, which they varied. However, by using group size as the basic measure of population density, this model has shown that a threshold badger density can be identified. For the range of most probable values of inter- and intragroup infection, this threshold group size was around six adults and yearlings. Although the disease would persist at this density for long periods of time, it was always in decline. The probability of spread from a point source of infection at this density was small, and at a lower mean group size the disease would neither spread nor persist after an initial homogeneous infection throughout the population. In contrast, at a mean group size of eight adults and yearlings, the probability of disease spread and persistence was much increased and the disease could become endemic.

One phenomenon not found in this modelling exercise was any evidence for cyclicity of the disease, as was initially proposed by Anderson & Trewhella (1985). Although one set of field data have been cited as conforming to such a pattern (Cheeseman *et al.* 1989), the evidence for cyclicity is as yet equivocal. Cyclic patterns of disease prevalence are often found for many short-lived infections, and seasonality in transmission rates is one of many complex factors potentially contributing to the persistence of such cycles (May & Anderson 1979). There is almost certainly considerable seasonality in intergroup infection rates for badgers, and this has been incorporated into this model. However, the chronic nature of the disease would be expected to mask these fluctuations and marked cyclicity, especially with a regular periodicity, would not therefore be expected. The high degree of spatial and social structure in badger populations also results in a system of mixing and contacts which is far removed from the homogeneous mixing assumed in deterministic non-spatial models, and this would also act against regular cyclicity. This effect would be exacerbated by any clustering of the population into supergroups as has been argued for a high-density population by Evans *et al.* (1987). The marked cyclicity predicted by Anderson & Trewhella (1985) is probably, in part, an artefact caused by the freely mixing structure of the population in their deterministic model, something which would not be observed in reality.

The levels of badger population depression caused by the disease at apparent equilibrium, and with the standard values used in this model, were similar to those predicted by Anderson & Trewhella (1985), but are not supported by a long-term field study (Cheeseman *et al.* 1988, 1989; Wilesmith 1991). However, this field study was of a single undisturbed badger population in which the geographical extent of *M. bovis* infection was very limited, and this model was able to replicate the results of these studies after the adoption of an appropriate initial pattern of infection (see figure 3*f*). There are no field data on the extent of disease-induced population depression from populations with more widespread infections.

The measures of population depression obtained from the model were calculated after an initial homogeneous infection of a large grid of contiguous badger groups. Although this is thought to approximate to the initial seeding of infection into the badger population, it is no longer a realistic scenario. Depression of badger population density after point-source infections was generally much lower due to the limited rate of spread of the disease and the large number of groups remaining uninfected, and this is the pattern of infection most likely to be observed now in the southwest. An additional complicating factor could be an inadequate representation in the model of the dynamics of colonization and intergroup movements, in relation to changing group size and occupancy of territories. If there are many empty territories, as would arise when the population is greatly depressed, and if disturbance is significant, then colonization might be expected to increase. This would reduce the overall impact of endemic disease on population density, as well as increasing the prevalence levels at apparent equilibrium. However, even very considerable variations in colonization and movement behaviour would be insufficient to reduce the impact of

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endemic disease on the badger population to the minimal levels suggested by Cheeseman *et al.* (1988, 1989) and Wilesmith (1991).

One factor known to increase intergroup movements in badger populations (Cheeseman *et al.* 1988*a*) is perturbation. Badger control itself may result in sufficient disturbance to decrease social stability and thereby lead to greater movements and more disease spread (Brown *et al.* 1994; MAFF 1994). Anderson & Trewhella (1985) suggested that the persistence of the disease at low badger densities would be aided by a degree of movement between social groups. However, the current analyses have indicated that this is not necessarily the case; increases in intergroup movement may actually decrease the probability of disease spread and persistence. This is due to the heterogeneous nature of the badger host population, the chronic nature of the *M. bovis* infection in badgers and the low rates of infection of susceptible animals by infectious ones. When the probability of intergroup movement is increased considerably, a core of infection is able to persist only when the mean group size is large and/or the intragroup infection probability is high. Thus, although there is still the potential for more rapid spread of the infection with a high degree of intergroup movement, the probability of it persisting may be reduced. Increases in the normal dispersal distance caused by disturbance may therefore act to decrease the probability of persistence of the disease. However, they might also increase the initial rate of spread, thus complicating any attempts to predict the spatial dynamics of the disease over time.

As well as potentially increasing intergroup movements, disturbance is also likely to weaken the existing badger social structure. The inevitable result of this will be an overall increase in intergroup contacts, and specifically more contacts of the type most favourable for the transfer of infection i.e. aggressive. Thus, disturbance would be expected to result in higher overall intergroup infection probabilities as well as increased movements. These analyses have shown that increased intergroup infection probabilities alone will have a significant effect in increasing the probability and rate of disease spread and reducing the threshold group size, and this is therefore the most important aspect of disturbance from the epidemiological perspective. Increased intergroup movements may offset the effects of increased intergroup infection to some extent, but a combination of increased intergroup infection and increased intergroup movement will still result in a marked increase in the probability and rate of spread of bovine tuberculosis through a badger population and is likely to reduce the threshold group size to well below six adults and yearlings.

The greater importance of intergroup infection compared with dispersal in spreading the disease can be demonstrated as follows. The probability of an infectious animal transmitting the infection to the surrounding ring of social groups by dispersal is d, the probability of it dispersing. No further transmission of the disease is required since by moving the animal has itself become part of a neighbouring group. The probability of an infectious animal transmitting the infection to a neighbouring group by intergroup infection can be expressed as:

$$1-(1-\beta_{\mathrm{b}})^{nG},$$

where β_{b} is the intergroup infection probability, n is the number of adjacent groups, and G is the mean group size. For dispersal to be a more important transmission process:

$$d > 1-(1-\beta_{\mathrm{b}})^{nG}.$$

Thus, as group size and/or the probability of intergroup infection increase, the probability of dispersal being a more important process in disease spread declines. The overriding importance of intergroup infection in the dynamics of bovine tuberculosis infection in badger populations is due to the rigid spatial structure of the population, the large group sizes and the limited spatial extent of dispersal. Thus for a fixed intergroup probability of 0.01, for dispersal to be more important in disease spread in a population with a mean group size of eight adults and yearlings, the probability of dispersal must exceed 0.28, yet for a mean group size of four adults and yearlings it only has to exceed 0.15. However, for animals which live in groups where dispersal is less restricted and dispersing animals can move over larger distances, dispersal will assume a relatively more important role in disease spread. This may be the situation in populations which have been disturbed by repeated control operations. A better understanding of the precise effects of control operations on badger social organization and behaviour would considerably enhance the value of any predictions regarding the spread or control of the disease.

A characteristic of the disease in an endemic state was its heterogeneous nature. Distinct pockets of infection were formed despite the homogeneity of the imposed environment. A similar theoretical pattern for endemic disease has been demonstrated for rabies by Mollison & Kuulasmaa (1985). These authors showed that a rabies epidemic which began by advancing in a fairly regular manner could break up into an endemic pattern of wandering patches of infection. Thus, heterogeneous behaviour can arise in a homogeneous environment. Any environmental heterogeneity that might lead to local increases in either host density or contact rates, and hence decreases in the threshold density for disease transmission would act to enhance the probability of formation of disease patches. However, it would also increase the spatio-temporal stability of these patches. Increased environmental heterogeneity has been shown to be associated with an increased risk of transmission of the infection from badgers to cattle (White *et al.* 1993), and it may also result in an increased potential for disease spread through the badger population. Any heterogeneities in the environment or the risk of disease transmission could therefore favour the formation and persistence of spatial pockets of the disease in badgers.

Although bovine tuberculosis has been found in badgers from southern Scotland southwards, it is predominantly in southwest England that it continues

to persist locally at levels high enough to pose a risk to cattle. The extent of regional variations in mean badger social group size in Britain are currently unknown (Cresswell *et al*. 1990), although they may be considerable. For example, Skinner *et al*. (1991) reported that an average social group in Essex probably contained three adults and yearlings, and that areas with many contiguous social groups were rare and that the badger population in Essex was becoming increasingly fragmented. Any degree of fragmentation in the badger population will decrease the probability of bovine tuberculosis persisting or spreading. One reason for the concentration of bovine tuberculosis in badger populations in the southwest may be because there are extensive areas of contiguous badger social groups with a mean group size close to or greater than the threshold level. Kruuk & Parish (1982) found that badger group size was positively related with the biomass of earthworms (*Lumbricus terrestris*) per badger territory. Earthworms are particularly abundant in permanent pasture and their surface activity i.e. when they are available to foraging badgers, is greatest on warm, humid nights (Edwards & Lofty 1972). These climatic conditions are more frequent in southwest Britain, thereby tending to increase the number of nights during which badgers can feed on earthworms. Microclimatic effects are also likely to be important, and the highly heterogeneous nature of the landscape in parts of the southwest could also increase the probability of food availability on a specific territory on any one night (Hofer 1988). The relations between landscape heterogeneity, the available earthworm biomass (and hence mean badger group size), the opportunities for the transmission of bovine tuberculosis both between badgers and from badgers to cattle, and the prevalence and spatial distribution of bovine tuberculosis in the badger population, need to be investigated further to help understand the patchy distribution of bovine tuberculosis in badgers in southwest England.

This analysis has concentrated on examining the spread of bovine tuberculosis within and between contiguous social groups of badger subpopulations. The spatial distribution and connectivity of these subpopulations will be major factors influencing the likelihood of disease spread throughout the meta-population. Thus a proper management strategy for the control and management of the disease in badger populations in southwest Britain needs to consider the environmental determinants of badger distribution and density in the problem areas. Once these data are available, spatial simulation models such as the one presented here can be used to estimate the likely rate and pattern of spread of the disease in specific areas, and then to evaluate the effectiveness of different control strategies in specific circumstances. As a first step towards achieving this longer-term goal, in the next paper White & Harris (1995) used this model to compare the effectiveness of the various control strategies implemented to date.

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