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POPULATION DYNAMICS OF THE BADGER (*MELES MELES*) AND THE EPIDEMIOLOGY OF BOVINE TUBERCULOSIS (*MYCOBACTERIUM BOVIS*)

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CONTENTS

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A survey and analysis is presented of the population biology of the badger (*Meles meles*) and the epidemiology of bovine tuberculosis (*Mycobacterium bovis*) within badger populations. Simple mathematical models are used to further our understanding of the processes that control the dynamics of badger abundance and disease transmission. Special attention is given to the identification of areas in which current knowledge is inadequate, and to future research needs.

The badger is shown to have a low intrinsic population growth rate, a not insignificant maturation delay to first breeding, to produce small litters of cubs which experience high rates of mortality in their first year of life but low rates thereafter, and to exhibit limited powers of dispersal. Population abundance is largely determined by habitat type and long term stability appears to arise primarily as a consequence of density-dependent constraints on fecundity. Such constraints are thought to only operate at densities close to the carrying capacity of the habitat. Cyclic fluctuations in abundance, with a period of between six and eight years, may occur in areas of moderate to poor habitat. Such fluctuations will be most apparent with respect to cub abundance as opposed to adult density.

Bovine tuberculosis is endemic within many badger populations throughout regions of Britain but is particularly prevalent in areas of good badger habitat in the southwest of England. Current evidence suggests that badgers play a significant role in disease transmission to cattle. It is argued that the infection is able to persist in high, moderate and low density badger populations. The observed stability of the disease appears to be a consequence of 'pseudo-vertical' transmission (from parent to new born offspring), the long duration of infectiousness of infected animals (low disease-induced mortality rate), the presence of carriers and inactive cases and the social organization and behaviour of the host species. Disease prevalence is likely to be related to badger density although in a nonlinear manner.

Control measures based on the removal of infected social groups of badgers in the southwest of England appear to have reduced the force of infection within badger populations by approximately 50%. The disease, however, remains endemic but at low levels of prevalence. The reduction in the force of infection has reduced the frequency of disease transmission to cattle herds. Eradication of the infection within badger populations may not be necessary for the *short-term* control of the infection in cattle. The persistence of low levels of infection in low density badger populations (suppressed by control measures), in areas of intensive cattle farming activity, however, presents a continual threat to cattle health in the *long term*. The ability of badger populations to recover from substantive reductions in density is poor, with a return time (to the pre-control state) of approximately five years. Small reductions in abundance, however, are likely to enhance net population growth rates as a consequence of the relaxation of density-dependent constraints on fecundity (the natural population regulatory mechanism). As such, rapid population growth to precontrol levels is predicted, following small reductions in density.

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As a consequence of the requirement for continual and substantive suppression of badger abundance (a renewable resource) in areas of intensive cattle farming it is suggested that alternative methods of disease control should be actively sought with a view to the design of more effective *long-term* control policies.

Introduction

The Eurasian badger (*Meles meles*) acts as a reservoir host for bovine tuberculosis (*Mycobacterium bovis*) in southwest England and is thought to play a significant role in the transmission of this bacterial infection to cattle (Zuckerman 1980). Although the subject of a continuing controversy, the circumstantial evidence for badger–cattle transmission is now considerable. It is well established, for example, that *M. bovis* infections are endemic in many badger populations in the south of England, and that outbreaks of infection in previously disease-free cattle herds are most frequent in areas of medium to high badger density (Muirhead *et al.* 1974; Gallagher & Nelson 1979; Cheeseman *et al.* 1981; Little *et al.* 1982; Wilesmith 1983). Direct evidence for transmission in natural agricultural habitats is difficult to obtain but research under experimental conditions confirms that the disease in cattle can arise from contact with infectious badgers (Little *et al.* 1982). In addition, ecological and epidemiological research has established that badgers forage preferentially on cattle pastures at certain times of the year when earthworms are abundant at the soil surface, and that bovine tuberculi bacilli survive in soil and in dung on grass for not inconsiderable periods of time (Kruuk 1978; Williams *et al.* 1930; Maddock 1933).

The Ministry of Agriculture, Fisheries and Food (M.A.F.F.) has overall responsibility for the eradication of bovine tuberculosis in cattle in Britain and has implemented direct measures to suppress the degree of badger to cattle disease transmission by the removal of infected social groups of badgers (the overall suppression of badger density) in Britain. Several research programmes have been initiated by M.A.F.F. in areas with badger–cattle disease problems, to provide much-needed scientific information to aid in the design of more effective long term control policies. One such research area is the topic of this paper; namely, the population biology of badgers and the dynamics of disease transmission.

The template of our research is a comprehensive survey and analysis of *quantitative* information on both the population biology of *M. meles* in Europe and the epidemiology of bovine tuberculosis within badger populations. This information, in conjuction with simple mathematical models, is used in an attempt to further our understanding of the processes that control the dynamics of badger abundance and disease spread. We focus specifically on the stability and resilience, of both badger and pathogen populations, to perturbations induced by various factors. A further aim is the identification of gaps in current knowledge of the dynamics of badger abundance and disease prevalence. This objective is of special relevance to the badger–bovine tuberculosis problem, in light of the overall paucity of relevant long-term ecological and epidemiological data.

This paper is organized as follows. The first section reviews the various processes (fecundity, mortality, dispersal and density-dependent factors) that influence badger abundance, and presents a quantitative analysis of the available data. The next section uses this information in the formulation of simple mathematical models and explores their dynamical properties. The third section examines the quantitative information on the epidemiology of bovine tuberculosis

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in badgers and presents a summary of the data relevant to the dynamics of disease transmission. This is followed in the next section by an attempt to construct simple models of disease spread and to examine the factors determining infection prevalence. The final part of the paper turns to the question of control and briefly discusses current progress. Throughout the paper, a primary aim is the identification of future research needs.

1. Population biology of *Meles meles*

Past research on the ecology of the badger has, to a large extent, focused on behavioural factors. Specifically, such studies have centred on the social organization of badger families and groups, the determinants of territory size and territorial behaviour, and feeding plus reproductive strategies (Ahnlund 1980; Graf & Wandeler 1982*a*; Kruuk 1978; Kruuk & Parish 1982; Neal & Harrison 1958; Stirling & Harper 1969; Wandeler & Graf 1982; Wijngaarden & Peppel 1964). Our focus is somewhat different; namely, the factors that determine temporal changes in both the numerical abundance of badger populations, and population age structure. Demographic changes depend on the prevailing rates of fecundity, mortality and dispersal, and the influence of climatic factors plus intra- and interspecific interactions on these processes.

Fecundity

The majority of mature female animals exhibit *post partum* ovulation and are fertilized in the spring and early summer (Ahnlund 1980). There then follows a period of delayed implantation, during which time the females may show renewed ovulation, and further mating may occur (Neal & Harrison 1958). Implantation takes place in December or January (somewhat dependent on the latitude of the habitat) and births occur in the following spring. In southwest England implantation occurs in late December and the majority of births take place between mid January and mid March, peaking in the first three weeks of February (Neal 1977).

The time taken to reach sexual maturity appears to depend on habitat latitude. Most females will first ovulate in the spring of their second year of life (aged 14 months), while the remainder mature the following autumn (aged 20 months) (Neal & Harrison 1958; Ahnlund 1980). In Sweden it has been reported that a few females do not mature until their third year of life (Ahnlund 1980). The mild climate and favourable habitat in southwest England result in some females maturing earlier, in late autumn of their first year (aged nine months) (S. Fargher and P. Morris, unpublished). Similarly, some males in this region may attain sexual maturity at, or before, 12 months old. The majority, however, mature early in their second year (aged 13–14 months) (Ahnlund 1980; Graf & Wandeler 1982) although, like females, a few may not reach maturity until the end of their second year (Neal 1977; Ahnlund 1980).

Estimates of litter size can be obtained at three stages of the reproductive cycle: before implantation, during pregnancy and from observations made after birth (table 1). The average litter size in Europe, calculated from measurements made at all three stages, is 2.7 cubs per female. Both litter sizes and pregnancy rates tend to be lower in yearling animals when compared with older females. Changes in the estimate of litter size during the reproductive cycle suggest that there may be a 15–20% prenatal mortality. It is believed that changes in the pregnancy rate via the cycle either reflect high mortality among pregnant females, reabsorption, or indicate abortion of an entire litter before birth. Tables 1, 2 and 3 present a summary of the quantitative data on litter sizes and pregnancy rates.

TABLE 1. AVERAGE LITTER SIZES FOR BADGERS

method of estimation	source of sample	sample size	age	mean litter size	reference number†
blastocysts	SW England	—	—	2.86	1
		—	—	2.88	1
		25	—	2.8	2
		391	all ages	2.67	3
		265	over 2 years	2.65	3
		26	12–14 months	2.04	3
		15	10–11 months	1.88	3
	central Sweden	257	3+ years	2.92	4
			1–2 years	2.53	4
		183	3+ years	2.9	5
		89	2 years	2.51	5
		41	1 year	2.34	5
	Switzerland	230	—	2.9	6
foetuses	SW England	10	—	3.1	2
	S and SW England	29	—	2.76	2
	central Sweden	12	3+ years	3.0	5
		4	2 years	3.25	5
		11	1 year	2.54	5
	Switzerland	230	—	3.1	6
placental scars	SW England	382	—	2.76	3
		78	—	2.95	3
	central Sweden	132	3+ years	2.31	5
		61	2 years	2.36	5
		54	1 year	1.94	5
foetuses and placental scars combined	S and SW England	37	—	2.92	7
	Germany	7	—	2.71	8
	Sweden	34	—	2.47	9
	SW France	23	—	2.95	10
cubs	SW England	50	—	2.3	2
	S and SW England	97	8–10 weeks	2.37	7
	Holland	15	—	3.3	11
	E Germany	14	—	2.4	12
totals					
blastocyst		1522		2.70	
foetuses and placental scars		1104		2.73	
cubs		176		2.43	

† Reference number 1, M.A.F.F. (1979); 2, Neal & Harrison (1958); 3, R. Page (unpublished); 4, Ahnlund (1980); 5, H. Ahnlund (unpublished); 6, Wandeler & Graf (1982); 7, Neal (1977); 8, Fischer (1931); 9, Notini (1948); 10, Canivenc (1966); 11, Wijngarden & Peppel (1964); 12, Stubbe (1965).

Sex ratio

The sex ratio in badger populations, for both adults and cubs, is in general close to unity (table 4). Variation is sometimes observed, however, between small groups of animals (Kruuk 1978).

Age structure

A wide variety of methods have been used to age badgers. These include techniques based on the fusion of the tibial epiphyses, teeth sectioning, teeth wear, eye lens mass and skull structure (M. K. Hancox, unpublished; Ahnlund 1976). Accurate ageing of adult badgers, however, remains problematic and recorded data must be interpreted with caution.

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TABLE 2. PREGNANCY RATES IN BADGERS IN CENTRAL SWEDEN

age class (years)	pregnancy rate (%) (sample size in parentheses)			
	blastocysts	placental scars	foetuses	lactating
1	51 ($n = 74$)	46 ($n = 107$)	67 ($n = 24$)	
2	93 ($n = 69$)	91 ($n = 66$)	90 ($n = 20$)	26–45 ($n = 26$)
3	93 ($n = 160$)	94 ($n = 143$)		

References: H. Ahnlund (unpublished); H. Ahnlund & Lindahl (unpublished).
Additional information on pregnancy rates (Canivenc 1966): SW France: pregnancy rate (blastocysts), 80–83% ($n = 270$).

TABLE 3. PREGNANCY RATES OF BADGERS IN SOUTHWEST ENGLAND

method of estimation	sample size	age	pregnancy (%)
blastocysts	25	—	92
	16	adult	88
blastocysts–embryos	386	2 years or more	max. 90
	131	all ages	36
	23	2 years or more	52
	99	—	15
	23	2 years or more	78‡
	32	9 months	min. 19, max. 44
placental scars	16	adult	25
lactating	243	2 years or more	32
	37†	adult	38
	16	adult	13

Data from Neal & Harrison (1958); Cheeseman *et al.* (1985); R. Page (unpublished); Gallagher & Nelson (1979); M.A.F.F. (1979); C. Cheeseman (unpublished); Fargher & Morris (unpublished); Notini (1948); H. Kruuk (personal communication).
† This sample from Scotland.
‡ Recolonizing population after gassing.

Badgers show different activity patterns according to age, sex and season. Comparisons between samples, collected in different months, may therefore be misleading with respect to age composition. Certain workers believe that an autumn sample is the most representative of a population's true age distribution. At this time, on average in Europe, roughly 25–35% of the population are cubs, about 25% are juveniles (second year of life, not yet attained sexual maturity) and the remainder are mature adults. H. Ahnlund (unpublished) found no significant difference in the age structure between samples collected by different methods. Recent studies by M.A.F.F. scientists in Britain, however, suggest that results obtained by trapping for short periods tend to provide underestimates of the proportion of cubs and juveniles in the population (C. Cheeseman, personal communication). A summary of data culled from published and unpublished literature is present in table 5. This information on population age

Table 4. The sex ratio of badger populations

sample size	age	sex ratio (male:female)	reference number†
75	—	1.0:0.9	1
86	—	1.0:1.0	2
1150	all ages	1.0:1.0	3
(286)	0–1 years	1.0:1.0	3
571	—	1.0:1.0	4
31	—	1.0:1.1	5
20	—	1.0:0.8	6
83	all ages	1.0:1.1	6
(42)	0–1 year	1.0:1.0	6
515	—	1.0:0.6	7
526	—	1.0:1.0	7
68	—	1.0:0.9	8
757	all ages	1.0:1.3	9
(676)	0–1 year	1.0:1.4	9
136	all ages	1.0:0.9	10
(36)	0–1 year	1.0:0.4	10
51	all ages	1.0:1.3	11
(20)	0–1 year	1.0:1.0	11
1990	—	1.0:0.9	12
45	all ages	1.0:1.3	13
(13)	0–1 year	1.0:2.3	13
total **6087**		**1.0:1.0**	

† References: 1, Stubbe (1970); 2, H. Ahnlund and Lindahl (unpublished); 3, H. Ahnlund (unpublished); 4, Neal (1977); 5, Kruuk (1978); 6, Little *et al.* (1982); 7, M.A.F.F. (1979); 8, Gallagher & Nelson (1979); 9, J. Gallagher (unpublished); 10, Cheeseman *et al.* (1981); 11, Cheeseman *et al.* (1985); 12, R. Page (unpublished); 13, Cheeseman *et al.* (1984).

structure is divided into three subsets, which reflect various degrees of refinement in the subdivision of populations by age. Note that few studies have been able to provide precise information on age in the older segments of badger populations.

Mortality

Observed mortality rates of *M. meles* vary according to age, habitat type and geographical location of the population (table 6). Cubs have a particularly high mortality rate (not unusual for mammalian species; see, for example, Fowler 1981) and by the end of their first year of life between 50% to 70% may have died. Up to 25% of this loss may occur while the cubs are still underground in the sets since a large proportion of adult females, examined in the spring, show fresh placental scars but are not rearing cubs (Neal 1977; H. Ahnlund, unpublished; Wandeler & Graf 1982; H. Ahnlund and Lindahl, unpublished). The likely causes of such mortality include starvation, respiratory disease and aggressive behaviour by adult badgers (H. Ahnlund, unpublished; M. K. Hancox, unpublished; Stubbe 1970). Starvation may also be a major cause of deaths for newly weaned cubs, notably in periods of dry weather (with

TABLE 5. THE AGE STRUCTURE OF BADGER POPULATIONS

(*a*)

area...	E.(SW)†	E.(SW)	E.(SW)	E.(SW)	E.(M.)
source...	T	T	T	T	T
date...	Aug.	Nov.	June	Oct.–Nov.	June
sample size...	29	38	40	29	45
age class	percentage in each age class				
cubs	10	24	38	31	29
adults	90	76	62	69	71
references...	Cheeseman *et al.* (1981); Cheeseman *et al.* (1983)				

(*b*)

area...	G.D.R.	G.D.R.	E.(SW)	E.(SW)	Sweden	E.(SW)	E.(SW)	E.(SW)
source...	obs.	S, n.d.	r.t.a., M.A.F.F.	r.t.a., M.A.F.F.	H	r.t.a., M.A.F.F.	r.t.a., M.A.F.F.	r.t.a., M.A.F.F.
date...	Apr.–July	—	—	—	autumn	—	—	—
sample size...	75	92	232	1259	745	757	1457	49
age class	percentage in each age class							
0–1	32	26	25	21	26	11	20	41
1–2	29	33	23	11	19	9	12	29
2–3/3+	39	41	51	8	18	14	13	12
3–4/4+	—	—	—	60	37	41	17	2
4–5/5+	—	—	—	—	—	25	15	6
5–6	—	—	—	—	—	—	5	6
6–7+	—	—	—	—	—	—	19	4
references...	Stubbe (1970); Stubbe (1973); S. Forgher and P. Morris (unpublished); R. Page (unpublished); H. Ahnland (unpublished); J. Gallagher (unpublished); Cheeseman *et al.* (1985).							

(*c*)

area...	Sweden	E.(M.)	Switz.
source...	T	n.d.	—
date...	—	—	—
sample size...	130	140	702
age class	percentage in each age class		
0–1	—	33	20
1–2	24	8	18
2–3	24	12	10
3–4	19	{22}	13
4–5	14		14
5–10	21	16	20
10–15		7	4
references...	H. Ahnland (unpublished); Graf & Wandeler (1982*b*); Hancox (1980*a*)		

† S, shooting; T, trapping; obs., direct observation; r.t.a., road traffic accidents; M.A.F.F., Ministry of Agriculture, Fisheries and Food; H, hunting; n.d., natural death; G.D.R., German Democratic Republic; E., England (SW southwest, M., Midlands); Switz., Switzerland.

consequent poor food availability) (Neal 1977). Dispersing juveniles attempting to join new social groups also tend to suffer a further period of high mortality as a result of starvation (if unsuccessful in their search for good habitat) and aggressive behaviour.

The adult death rate appears fairly low and is often approximately constant with age at around 25% per annum. It is argued that adult animals have no natural predators (other than man) (Neal 1977). Disease, food availability, territorial aggression and road accidents probably constitute the major causes of adult death (Gallagher & Nelson 1979; Hancox 1980). In

Table 6. Mortality rates for badgers

age class	source	sample size	percentage mortality (per age class)
prenatal:	pre- and postnatal litter sizes	—	18
cubs			
0–8 weeks	Pre- and postnatal litter sizes	134	19
0–12 weeks		60	26
		—	25
0–6 months	spring versus autumn sample	—	48–68
0–1 year	skulls	—	50
0–1 year	skulls	—	70
cubs–adults			
0–3 years	skulls	140	65
adults			
1–3 years	skulls	—	20–25
1+ years	mark–recapture	86	25
1+ years	spring versus autumn sample	—	25
3+ years	skulls	—	low
all ages	—	—	25

Data from H. Ahnlund (unpublished); Neal (1977); Neal & Harrison (1958); M.A.F.F. (1979); C. Cheeseman (unpublished); Hancox (1980*a*); H. Ahnlund and Lindahl (unpublished).

Gloucestershire, bovine tuberculosis was recorded as a major cause of mortality (Gallagher & Nelson 1979) although a more recent study suggests that the disease induced mortality rate from bovine tuberculosis is fairly low (Cheeseman *et al.* 1985). On the continent of Europe, several rabies epidemics have resulted in high badger mortality, but usually as a consequence of close contact with infected fox (*Vulpes vulpes*) populations. The fox appears largely responsible for the maintenance of the current rabies epidemic in Europe (Anderson *et al.* 1981). Deaths due to bite wounds suffered during intraspecific aggressive acts are a common cause of mortality in Britain and Sweden (Gallagher & Nelson 1979; H. Ahnlund and Lindahl, unpublished). In older animals, starvation may result from excessive teeth wear (Gallagher & Nelson 1979). The largest single cause of mortality in Britain at present, however, is road traffic accidents (Neal 1977).

Dispersion

At present little is known about the dispersal behaviour of young badgers and the distances travelled in the search for territories. In areas capable of supporting population growth, young animals may settle in the social group of their parents (Neal & Harrison 1958). When territories, in areas of good habitat, are occupied and strongly defended, young animals may have difficulty establishing within breeding groups and often have to live in outlying sets in suboptimal areas (C. Cheeseman, personal communication). Dispersal probably plays a role in the regulation of population density, but not a substantial one as in the case of the red fox, *Vulpes vulpes* (Macdonald 1980).

Density

The choice of habitat for the establishment of badger sets depends on various factors including local geological conditions (soil type and suitability for tunnel construction), food availability

and the degree of vegetational cover plus seclusion from areas of high human activity (Neal 1977). Given suitable geological conditions, the availability and biomass of earthworms (the main food item of *M. meles*) plays an important role in determining territory size and population abundance (Kruuk & Parish 1982). Hilly areas, with sandy soil, interspersed with permanent pasture land and deciduous woodland provide the most favourable habitats (Neal 1972). Such conditions are common in parts of south and southwest England. On the Cotswold escarpment, for example, the highest known badger densities are recorded at 20 adults per square kilometre. More generally a value of five to eight adults per square kilometre is typical of favourable habitats in Britain (table 7). On the continent, and in less suitable regions in Britain much lower densities are recorded (table 7). The association between territory size and overall population density in an area is strong: at high densities territories are on average small (reflecting high food availability and abundant sites for set construction).

TABLE 7. THE RANGE OF DENSITIES AND TERRITORY SIZES FOR BADGERS IN BRITAIN AND EUROPE

area	adult density km^{-2}	territory size km^2	reference number†
Czechoslovakia	under 0.1–0.6	—	1
Ukraine	0.5–1.8	—	2
France	under 1.0	—	3
Holland	1.0	—	4
France	1.6	2.5	5
Sweden	2.4–3.2	1.6–2.4	6
East Germany	2.0–4.0	—	7
Scotland	1.5–2.7	1.5–2.0	8
	1.1–3.2	1.2–3.1	
	5.7–6.2	1.3–2.0	
Cornwall	4.7	0.75	9
Avon	4.9	0.79	9
Durham	5.8	—	10
Oxford	5.2–8.4	0.5–1.5	11
Staffordshire	6.2	1.04	12
Britain	10.6	—	13
Gloucestershire	19.4	0.25	9
	19.7	0.22	9
national badger survey categories	set density (per 100 km^2)	—	14
very common	over 50		
common	31–50		
frequent	16–30		
infrequent	6–15		
scarce	1–5		

† References: 1, Pelickan & Vackar (1978); 2, Abellencev (1966); 3, Rapaport (1979); 4, Wijngaarden & Peppel (1964); 5, Mouches (1982); 6, H. Ahnlund and Lindahl (unpublished); 7, Stubbe (1965); 8, Kruuk & Parish (1982); 9, Cheeseman *et al.* (1981); 10, Fargher & Morris (unpublished); 11, Fisher (1931); 12, J. Gallagher (unpublished); 13, Hancox (1980*a*); 14, Mammal Society Records.

Fluctuations in population abundance

To date there have been few detailed long term studies of badger populations in which quantitative data on abundance is available. What little evidence there is (mainly anecdotal) suggests that unperturbed populations remain fairly constant in size through time. In Wytham Woods, Oxfordshire, (M. K. Hancox, unpublished), little change in badger numbers was observed over a 20 year period (small peaks in abundance were recorded in 1953, 1959 and

1964). Observations on badger populations in Coombes Valley, Staffordshire, show that between 1947 and 1978 population size and number of sets occupied varied very little (Royal Society for the Protection of Birds, personal communication). In Sweden, postwar hunting returns suggests that badger numbers have also remained fairly constant (H. Ahnlund, personal communication). Similarly in a 22-year study in Czechoslovakia, Perlickan & Vackar (1978), recorded little change in set occupancy and population abundance within a small area of badger habitat.

Depression of population abundance is most commonly associated with years of severe drought which limit the supply of earthworms (Wytham Woods, Oxfordshire (J. Phillipson, personal communication); Coombes Valley, Staffordshire (R.S.P.B., personal communication); Tula Abatis forest, Russia (Likhacev 1956)) and disease (rabies epidemics in West Germany (Wackendorf & Schwierz 1980); undefined infection in Habel Wood, East Germany, (Stubbe 1965)). After perturbation (the cessation of the drought or the termination of the disease epidemic) badger populations in general exhibit a slow return to the levels of abundance recorded before depression. In both Wytham Woods (Oxfordshire) and Tula Abatis (Russia) the population recovered to their original levels after falling by 30–50% (figure 1). Recolonization after a severe reduction, however, may depend upon the status of the surrounding populations or the degree of isolation of the habitat from other areas of badger occupancy. Serious disruption of the social organization of badger groups, resulting from a drastic reduction in density, appears to reduce the rate of return to preperturbation levels of abundance. In two areas in the southwest of England (figure 2) where the badger populations were completely removed, the level of recolonization is still low after an interval of three to four years. In one area no cubs were known to be produced for three years despite the presence of adult females and males (C. Cheeseman, personal communication).

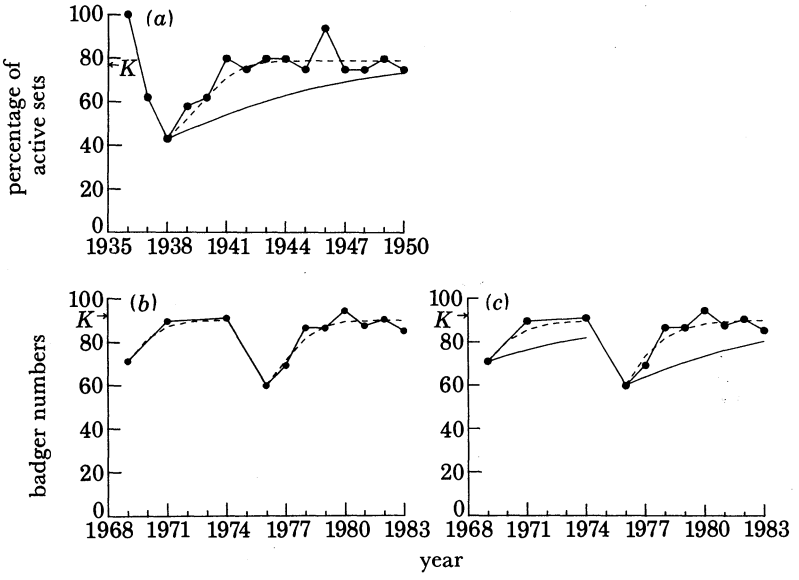

FIGURE 1. Recolonization studies following population depression. Recovery of the populations is shown by ●—●, with fitted models of population growth shown by solid and dashed lines. (*a*) Tula forest, Czechoslovakia: K approximately equal to 79% active sets. – – –, $r = 0.2$ per year, curvilinear density dependence, $c = 7$; ——, $r = 0.2$ per year, logistic density dependence. (*b*), (*c*) Wytham Woods, Oxfordshire K, 90 badgers in 1080 ha; that is, 8.5 km^{-2}. (*b*) – – – $r = 0.2$ per year, curvilinear density dependence, $c = 7$; (*c*) – – – $r = 0.8$ per year, logistic density dependence; ——, $r = 0.2$ per year, logistic density dependence.

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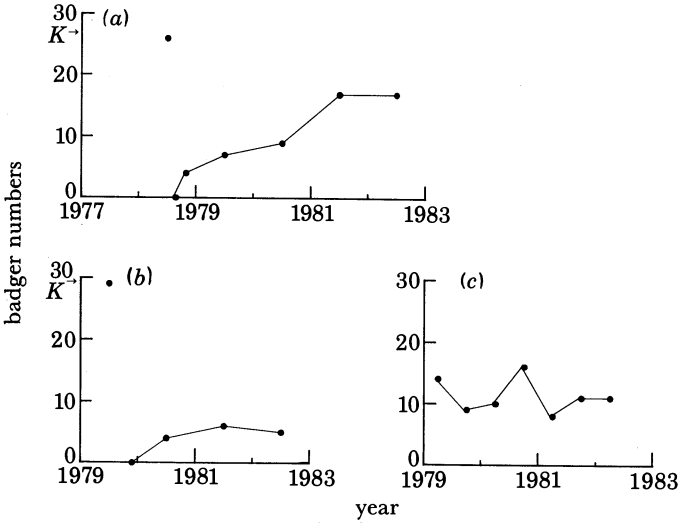

FIGURE 2. Recolonization studies following population removal. (*a*) Recolonization of a badger population in Gloucestershire. *K* before culling at 26 adult badgers in 132 ha, was 20 km^{-2}. Slow recolonization of populations in: (*b*) Gloucestershire (*K* before culling at 29 adult badgers in 124 ha, was 19 km^{-2}); (*c*) Dorset study group (*K* before culling unknown, but much more than 30 badgers in the area).

An interesting feature of these unpublished experiments is the observation that the home ranges of the recolonizing badgers were much larger than normal for the area and not clearly delineated as is usually the case (M.A.F.F. 1981). It appears likely that a degree of stability in social organization and group structure is necessary for successful breeding.

Population regulation and the intrinsic growth rate, r

The dynamical properties of mammalian populations are to a large extent determined by two factors; namely, the nature and form of regulatory constraints on growth, and the magnitude of the population's natural intrinsic growth rate, *r* (May 1981).

(*a*) *Density dependence*

The principal density-dependent check on growth appears to act on fecundity as opposed to adult or juvenile mortality. This is in contrast to certain other mammals, such as the red fox (*V. vulpes*), where density-dependent mortality associated with juvenile dispersal behaviour is of great importance (Anderson *et al.* 1981). The belief that effective fecundity is influenced by density centres on differences in pregnancy rates and cub production. Almost all mature females in medium to high density populations in Britain are mated and possess blastocysts in the summer months (see table 3). At a postimplantation stage, however, there is a reduction in the proportion pregnant, with 50% or less harbouring foetuses. Pregnancy rates also appear much lower when based on the proportion of lactating females than would be the case if the calculation centred on the presence or absence of blastocysts. In Sweden, where densities are typically much lower, there is no sign of this sequential reduction in effective fertility as the animals move via the seasonal reproductive cycle (see table 2). Some of this observed loss in British populations, however, might be accounted for by high early cub mortality.

Evidence to this effect has been reported from the more density populated areas of central as opposed to northern Sweden (H. Ahnlund, unpublished). In general, however, early cub

mortality (which may or may not be density related) can be subsumed into a net measure of 'effective' reproduction (the word effective is meant to imply the production of juvenile and adult animals).

The mechanisms by which fecundity or early cub mortality are influenced by density are not well understood. Neal (1977) argues that food availability in the autumn, and stress on females induced by frequent contact in high density populations, are important. Social status may also play a role. Kruuk (1978) suggests that only one litter is typically produced per social group, despite the presence of more than one mature female. The social groups containing dominant males or females appear to occupy the most favourable set sites in a territory and this factor is thought to be of great advantage to breeding success (Neal 1977). Stubbe (1970, 1973) suggests that respiratory diseases are significant in early cub mortality and as such this factor will be linked to overall group density. In general, however, hard quantitative evidence on any association between fecundity and mortality, and population density is not available at present. Manipulative experimental work under field conditions is required to obtain such information.

The qualitative evidence on pregnancy rates and early cub mortality plus the quantitative evidence on the temporal stability of natural populations argues that density-dependent processes exert a strong influence on population behaviour. In connection with this assumption, we also need to know the precise functional relationship between population growth rate (or fecundity and mortality) and animal abundance. The well known logistic model of population growth, for example, is based on the assumption that the relationship is linear (Pearl & Reed 1920). For medium to large mammalian species, however, theoretical and empirical evidence suggest that density-dependent effects enter nonlinearly, such that constraints on growth only begin to play a regulatory role at high densities. At low to medium densities net growth is thought to be directly proportional to abundance (Caughley 1977; Fowler 1981; Fowler & Smith 1981). This type of response is typical of species with low reproductive rates, long life spans and of populations dominantly limited by resources as opposed to climatic factors (mammalian K strategists: see Southwood (1981), Pianka (1970, 1972)). The badger clearly possesses these life history characteristics and as such its intrinsic growth rate is likely to exhibit the 'large mammal' type functional response to density (Fowler 1981).

(*b*) *The intrinsic population growth rate, r*

In the absence of resource limitation the intrinsic potential of a species for population growth is difficult to quantify solely on the basis of field data. Estimates of *per capita* fecundity and mortality obtained from natural populations reflect, to some degree, the prevailing conditions of abundance and resource availability.

With this caution in mind a crude estimate of the intrinsic growth rate of the badger, r, can be calculated from published data on productivity, adult survival and the response of populations to perturbation (induced by either disease, man's activities or drought). The *per capita* birth rate, γ, can be estimated from information on the productivity per female (the average number of cubs produced per year) and the sex ratio of the population. Productivity itself is determined by the pregnancy rate and the average litter size in each age class. The most detailed information available on age-specific productivity comes from Sweden (H. Ahnlund, unpublished). The total productivity per female per year as determined from data on blastocysts was 1.64, while the equivalent figure calculated from information on placental

scars was 1.34. M.A.F.F. scientists have produced similar information for England (R. Page, unpublished) based on blastocysts and the proportion of cubs within different populations and this yields an average productivity figure of 1.67 per female per year (table 8). Given these estimates, the *per capita* birth rates for the Swedish habitat are 0.5–0.6 per year (based on placental scars and blastocysts, respectively). For the English habitats we calculate on the basis of blastocysts a figure of 0.6 per year. Note the remarkable similarities between these estimates based on data from different European countries. In the context of the estimation of the intrinsic growth rate r, the figures based on blastocyst counts are the relevant ones. As discussed earlier, density dependence factors may influence the likelihood that a female badger proceeds from the blastocyst stage of the reproductive cycle through to cub production.

TABLE 8. PRODUCTIVITY OF BADGERS IN SOUTHWEST ENGLAND

age class	percentage age structure			litter size	percentage pregnancy rate		productivity
0–1	21	25	30	0–2.0	0–20		0–0.12
1–2	11	23	25	2.04	37		0.18–0.19
2–3+	68	31	45	2.65	95		1.13–1.71
reference	28	10	(table 4)	28	28	total	**1.31–2.02**
						mean	1.67

Data from R. Page (M.A.F.F., unpublished) and S. Fargher and P. Morris (unpublished).

Per capita mortality rates, b, may be estimated from three different types of information. First, there is the direct method of mark and recapture, where individual animals within a population are tagged and the habitat is trapped frequently over many years in an attempt to monitor the life span of individual animals. This method is very labour intensive and subject to many sources of error such as the dispersal of animals from the study area and trap avoidance behaviour. More reliable estimates can be obtained from a second method, as described by Caughley (1967), which assumes that the population has a stable age distribution, and uses observations on the ratio of juveniles to adults in a population immediately following the breeding season. The method produces an estimate of mean life expectancy from birth, e_0, where

$$e_0 = (2n-j)/j. \tag{1}$$

Here n denotes sample size and j records the number of animals aged up to one year immediately after the breeding season. Estimates based on (1) are present in table 9. The average value of between two and three years (the *per capita* mortality rate, b, is simply the reciprocal of e_0) may be an over-estimate in the case of *M. meles* since cubs spend a significant period underground in the set following birth when they are hidden from observation. During this period significant mortality is thought to occur. The third method probably provides the most reliable estimates and is based on age distributions within the populations. Under the assumptions that the distribution is stable and that each age class is equally likely to be sampled, the data represents a survival curve. Three such patterns are portrayed in figure 3 and from this information it is possible, by standard methods (see Pollard 1973), to estimate age-specific mortality rates and life expectancy from birth. Interestingly, after the first year of life, the mortality rate appears to be approximately constant and independent of age. Fitting exponential decay curves to the data recorded in figure 3 yields estimates of life expectancy between 2.2 and 2.9 years (*per capita*

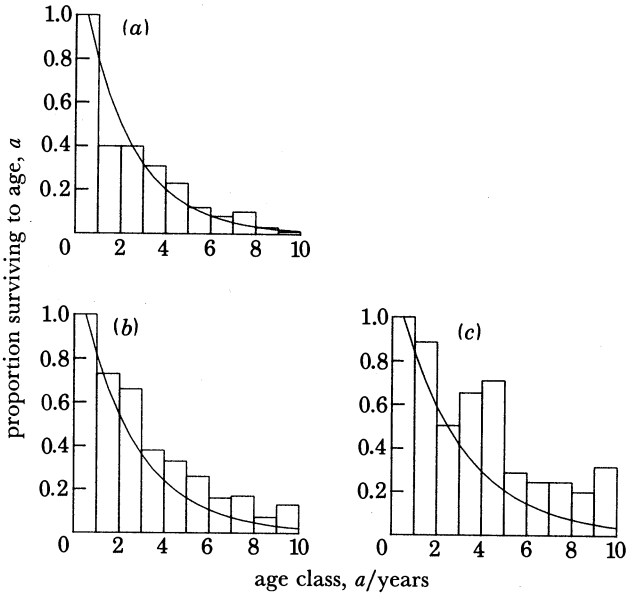

FIGURE 3. Survivorship curves for three badger populations. The *per capita* death rate, b, is estimated in each case by fitting an exponential decay curve. (*a*) Sweden (H. Ahnlund and Lindahl (unpublished)), $b = 0.46$ per year; (*b*) Sweden (H. Ahnlund (unpublished)), $b = 0.41$ per year; (*c*) Switzerland (Graf & Wandeler 1982), $b = 0.35$ per year.

TABLE 9. THE LIFE EXPECTANCY AT BIRTH FOR BADGERS (BASED ON THE ESTIMATION METHOD OF CAUGHLEY 1967)

area	sex	n	j	life expectancy years	*per capita* mortality rate per year	date of sample
SW England	♀	57	39	2.67	0.37	July–Oct.
	♂	40	23	1.85	0.54	July–Oct.
	combined	**97**	**35**	2.27	0.44	July–Oct.
SW England	♀	192	36	2.93	0.34	July–Oct.
	total	**289**	**91**	2.68	0.37	July–Oct.
E. Germany	combined	**75**	**24**	2.63	0.38	Apr.–July

Data from S. Fargher and P. Morris (unpublished); R. Page (unpublished) and Stubbe (1973).

mortality rates of 0.46–0.35 per year). Encouragingly, the second and third techniques produce similar estimates of e_0.

Combining the estimates of fecundity (γ) and mortality (b) we arrive at an estimate of the intrinsic growth rate ($r = \gamma - b$). For the southwest of England we obtain a value of 0.24 per year ($\gamma = 0.61$ per year, $b = 0.37$ per year) while for Sweden we arrive at an average value of 0.15 per year ($\gamma = 0.5$–0.6 per year, $b = 0.35$–0.46 per year) and a maximum value of 0.25 per year. If average values of γ and b from all sources (independent of country or habitat type) are used we obtain a value of 0.2 per year.

For comparison, the intrinsic growth rate of the red fox in Europe (*Vulpes vulpes*) is approximately 0.5 per year (Anderson *et al.* 1981). With respect to the life histories of the two species, the badger has a lower birth rate, longer life expectancy, lives in smaller well-defined

territories at higher densities and exhibits limited dispersal behaviour. Foxes, in contrast, mature earlier and disperse, on average, further.

Fenchel (1973) has elegantly demonstrated that the relationship of the intrinsic population growth rate r, and of metabolic rate per unit mass, to average organism size is approximately linear on a log–log scale. The relationships fall into three groups; unicellular organisms, poikilotherms and homeotherms. Each major evolutionary step slightly increases both the metabolic rate and r for a given size (Southwood 1981). For mammalian species an approximately linear relationship exists, as depicted in figure 4. Given an average adult badger mass of 11 kg, the value of $r = 0.2$ per year is consistent with the linear trend projected by larger and smaller species for which r values are available (that is, deer and mice). This provides corroborative evidence for the estimates derived in this study.

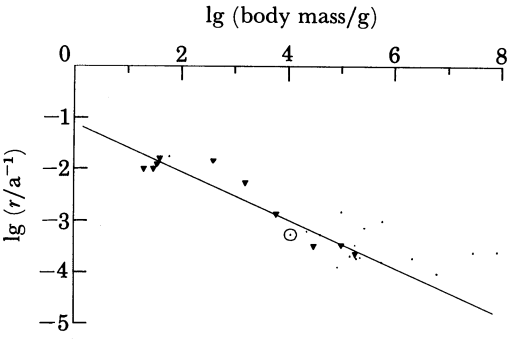

FIGURE 4. The relation between body mass and intrinsic growth rate for mammals. ○, badger; ▼, points where r determined on either laboratory populations or expanding natural populations; these points used to fit the straight line, correlation coefficient, −0.944 (mammal species range from small rodents to large ungulates); ●, points where r derived from life tables (mammal species range from small rodents to large ungulates).

2. Population dynamics and mathematical models

Simple models

The most widely used single species model, which captures the essential features of population growth in a finite environment where resources (either space, food or other factors) are limited, is the logistic equation:

$$\mathrm{d}N/\mathrm{d}t = rN(1-N/K). \tag{2}$$

Here N denotes population size, K is the habitat carrying capacity and r is the intrinsic growth rate. Embodied in this model is the assumption that the effective *per capita* growth is linearly dependent on population density ($f(N) = r(1-N/K)$): the growth rate is positive if $N < K$ and negative if $N > K$. The characteristic return time, T_{R}, of this system, a parameter that gives an order-of-magnitude estimate of the time the population takes to return to equilibrium following a disturbance (May 1973), is simply

$$T_{\mathrm{R}} = 1/r. \tag{3}$$

In other words the smaller the value of r the longer the period required to recover from a perturbation. Given the assumption that the logistic model is a crude mimic of the dynamics of badger populations ($r = 0.2$ per year), (3) would suggest a return time of roughly five years.

For comparison, a similar calculation for the red fox (*V. vulpes*) would give a return time of only two years.

As discussed earlier, in the section on population regulation, for many medium to large sized mammalian species the assumption of a linear dependency between the net growth rate, $\hat{r}(N)$, and density N is not appropriate. As discussed by Fowler (1981), a nonlinear function is required such that density-dependent constraints begin to operate as density N approaches the carrying capacity of the habitat, K. An empirical description of such a trend is given by the following function for the effective *per capita* growth rate:

$$\hat{r}(N) = r[1-(N/K)^c], \tag{4}$$

where c is a constant that reflects the severity of density-dependent constraints and r and K as defined for (2). The equation for population growth through time is therefore

$$\mathrm{d}N/\mathrm{d}t = r[1-(N/K)^c]\,N \tag{5}$$

with equilibrium K.

Equation (5) is globally stable to perturbations for all values of r, c and K, and the characteristic return time T_R is again approximately equal to $1/r$. Note that in the limit $c = 1$, the logistic equation (2) is recovered. Graphs depicting the functional dependencies of $\hat{r}(N)$, and $\mathrm{d}N/\mathrm{d}t$, on density N are presented in figure 5. As the value of c increases, the nonlinear effects of density-dependent constraints act closer and closer to the equilibrium density of the population, K. The discrete time analogue of (5) may, or may not, be stable depending on the values of r and c. Local stability analysis reveals that the difference equation exhibits a non-oscillatory return to the equilibrium K if $0 < rc < 1$, and involves damped oscillations if $1 < rc < 2$. The system is unstable (limit cycles moving to chaos) if $rc > 2$. For badger populations with an r value of 0.2 per year and $c \approx 7$ (see following paragraphs and figure 1) the system is predicted to be locally stable.

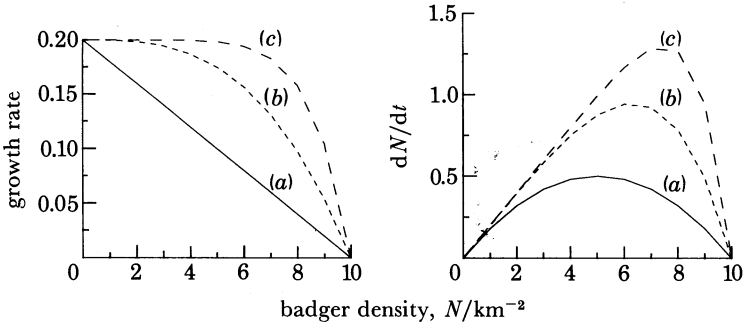

FIGURE 5. Logistic and curvilinear density-dependent functions. (*a*) Logistic form, where $\hat{r}(N) = r-\gamma N$; (*b*) 'mild' curvilinear form, where $\hat{r}(N) = \gamma - dN^c$, for $c = 3$; (*c*) 'severe' curvilinear form, where $\hat{r}(N) = \gamma - dN^c$, for $c = 7$.

As mentioned previously, precise quantitative data on the functional relationship of $\hat{r}(N)$ with population density, N, is not available at present. A crude guide to the value of the parameter c, however, can be obtained from field data recording badger population growth back to the habitat carrying capacity following a disturbance. Two suitable studies are available for analysis. The Wytham Wood (Oxfordshire) badger census (J. Phillipson, personal communication) reveals two occasions where density declined rapidly over a short period of time; one

due to the removal of badgers in 1969, and one following the drought in 1976 (it is likely that the drought of 1983 will have had a similar effect but data is not available at present). The second study was that of Likhacev (1956) in the Tula Abatis forest in Czechoslovakia and Russia. The percentage of active sets was used as an index of badger density before, during and after a decrease in abundance due to a drought and increased hunting pressure in 1938–1940.

Figure 1 records the fit of the 'mammal type' density-dependent model (5), and the logistic model (2) to the two data sets. For an r value of 0.2 per year, the mammal type density-dependent model provides a much more precise description of observed trends than the logistic equation (on the basis of the logistic model an r value of between 0.6 and 0.8 per year would be necessary to mimic the observations). A value of $c = 7.0$ provides a good fit of (5) to both sets of observations.

The simple differential equation model (5) can be defined to encorporate maturation delays in breeding. For example, if N is the density of mature adult badgers, and T is the time delay to first breeding (in the case of badgers T may be one or two years), (5) can be adapted as follows:

$$\mathrm{d}N(t)/\mathrm{d}t = N(t-T)\,[\gamma - \mathrm{d}N(t-T)^c] - bN(t). \tag{6}$$

Here the density-dependent fecundity term is $\hat{a}(N(t-T)) = \gamma - \mathrm{d}N(t-T)^c$ and $(\gamma - b) = r$; the equilibrium state is $K = (r/d)^{1/c}$. By using standard techniques of local stability analysis (see Nisbett & Gurney 1982) it can be shown that with parameter value $r = 0.2$ per year, $c = 7$ and $T = 2$ years the system exhibits divergent oscillations leading to a limit cycle. The period of these cycles is approximately $4T$, in other words eight years given a two-year maturation delay. However, for $T = 1$ year the system is locally stable. This analysis, in conjunction with the earlier biological discussion of maturation delays, suggest that badger populations border on the region of stability. They may, or may not, show oscillatory behaviour depending on the precise values of r, c and T in a given habitat (if they do occur an eight year period is to be expected). Numerical studies of (6), however, suggest that the amplitude of the oscillations will be small in relation to the magnitude of the equilibrium density K. In other words, in natural populations they may not be detectable as a consequence of sampling inaccuracies.

Age structured models

The simple model (5) discussed in the preceeding section provides a crude description of observed changes in density following a disturbance. Most importantly, it produces a rough guide to the characteristic return time, T_R, of badger populations. The model, however, has two major shortcomings. First, it fails to take account of the age structure of the badger communities and only crudely approximates the maturation delays to first breeding. Second, it treats both birth and mortality as continuous functions in time. This latter problem is difficult to handle in a simple model since birth is discrete (seasonal breeding) and mortality is continuous through time. Ideally, difference equations should be used to handle the discrete processes and differential equations for the continuous rates.

We can modify our model (5) to take account of these complications in two ways. First, we consider an age-structured difference equation model. The demography of a mammalian population with a well-defined age structure and seasonal breeding habits can be described in terms of a modified Leslie matrix model. We define $N_i(t)$, for $i = 0, 1, \ldots, k-1$, as the number

of animals of age i years in the population in year t and $N_k(t)$ as the number of reproductively mature animals of age k years and over in year t. We shall assume that the sex ratio of the population is unity. The population in year $t+1$ can then be related to the population in year t by:

$$\begin{bmatrix} N_0(t+1) \\ N_1(t+1) \\ \vdots \\ N_{k-1}(t+1) \\ N_k(t+1) \end{bmatrix} = \begin{bmatrix} 0 & 0 & \dots & 0 & 0 & R(N_k(t)) \\ (1-\mu_0) & 0 & \dots & 0 & 0 & 0 \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ 0 & 0 & \dots & (1-\mu_{k-2}) & 0 & 0 \\ 0 & 0 & \dots & 0 & (1-\mu_{k-1}) & (1-\mu_k) \end{bmatrix} \begin{bmatrix} N_0(t) \\ N_1(t) \\ \vdots \\ N_{k-1}(t) \\ N_k(t) \end{bmatrix}. \quad (7)$$

Here μ_i, for $i = 0, 1, \dots, k-1$, is the annual mortality rate of individuals of age i years, μ_k is the mortality rate of those mature animals aged k years and over and $R(N_k(t))$ is the 'mammal-type' density-dependent fecundity function. This system of equations can be collapsed to a single equation:

$$N_k(t+1) = (1-\mu_k)\, N_k(t) + (1-\mu_{k-1})\,(1-\mu_{k-2}) \dots (1-\mu_0)\, R(N_k(t-k))\, N_k(t-k). \quad (8)$$

For badgers in Europe, as discussed earlier, the majority of males and females mature in the second and third years of life. In the southwest of England some may mature in their first year, while in Sweden it may take at least three years to mature. Taking a value of $k = 2$ years, (8) may be simplified to give

$$N_2(t+1) = (1-\mu_2) N_2(t) + (1-\mu_0)(1-\mu_1)\, R(N_2(t-2))\, N_2(t-2). \quad (9)$$

Note that the number of cubs at time $t+1$, $N_0(t+1)$ is

$$N_0(t+1) = R(N_2(t)) N_2(t) \quad (10)$$

with $R(N)$ defined as

$$R(N_2(t)) = [\gamma - \mathrm{d}(N_2(t))^c]. \quad (11)$$

The equilibrium density of adult badgers, N^*, is

$$N^* = \left[\frac{\gamma(1-\mu_1)\,(1-\mu_0) - \mu_2}{\mathrm{d}(1-\mu_1)\,(1-\mu_0)}\right]^{1/c}. \quad (12)$$

The local stability properties of (9) are outlined in appendix 1. Equations of this general type may exhibit a complex range of possible dynamical behaviours, depending on the values of the parameters. The patterns range from monotonic growth to a stable equilibrium, damped oscillation to a stable point, limit cycles and chaotic trajectories through time. For a fuller discussion of the properties of nonlinear difference equations see Allen (1963), May (1974), Clark (1976), May & Oster (1976), Beddington (1978), Nisbett & Gurney (1982) and Fisher & Goh (1984).

The parameter values relevant to the dynamics of the badger in Europe are summarized in table 10. Analytical (see appendix 1) and numerical studies reveal some interesting patterns. With an adult density of $N^* = 10$ per square kilometre, a maturation delay of two years and the density-dependent parameter (the c of (11)) set at 7.0, the model exhibits oscillatory behaviour (cycles in adult and cub numbers) with a period of approximately eight years (figure 6*a*). Note the similarity of this result to that obtained from the time delayed differential

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TABLE 10. POPULATION PARAMETERS USED IN THE AGE-STRUCTURED MODELS

birth rate: γ finite rate = 1.82 per year, instantaneous rate = 0.6 per year
death rate
- μ difference model:
  - adult, μ_2, 25% per year
  - juvenile, μ_1, 35% per year
  - cubs, μ_0, 65% per year
- differential model
  - age class (3–10), 25% per year
  - age class 2, 35% per year
  - age class 1, 65% per year

adult carrying capacity: $K = 10$ km^{-2}
density-dependent parameters for nonlinear function
- (i) mild: $c = 3$
- (ii) severe: $c = 7$

equation model (6). With a maturation delay of one year (badgers breeding in their second year of life), and a slightly higher rate of mortality in the first year of life, the system oscillates but damps to a stable equilibrium. The period of the damped oscillations is approximately six years (figure 6*b*). In both cases the amplitude of the fluctuations in cub density are greater than those in adult numbers. Moreover, the cycles are asynchronous, with peaks in adult abundance following peaks in cub production (figure 6). The oscillations arise as a consequence of the maturation time delay and the nonlinear 'mammal type' density-dependence function. The system tends to overshoot the carrying capacity K and then immediately exhibit a reduction in numbers as a consequence of severe reductions in fecundity at high adult densities.

The analyses (both numerical and analytical) of the model suggests that badger populations may, or may not, exhibit oscillatory behaviour depending on the length of the maturation delay. In good habitats within Britain with most females breeding in their second year of life the populations are likely to be stable. In poorer habitats in Britain and in more northerly regions of Europe, where the maturation delay is longer, the populations may tend to oscillate in abundance with periods of six to eight years. Stochastic effects may perpetuate the damped oscillation of the short maturation delay populations but fluctuations in adult abundance will be of low amplitude and hence difficult to detect in practice (given the sampling errors inherent in population census techniques). The more marked oscillation in cub production may be detectable.

As noted earlier in this section, difference equation models have the advantage of accurately reflecting the discrete nature of seasonal breeding, but the concomitant disadvantage of only providing a crude treatment of mortality (which in reality is a continuous process). We can make some progress with these problems by using an age-structured continuous time framework (a partial differential equation) modified to take account of maturation delays and discrete seasonal breeding.

Single species age-structured population models in continuous time have received some attention in the ecological literature (see Nisbett & Gurney 1982) although rather less than their discrete time counterparts (see Leslie 1945; Pielou 1969). If $N(a, t)$ denotes the number of badgers of age a at time t then the partial differential equation model has the general form

$$\frac{\partial N(a, t)}{\partial t} + \frac{\partial N(a, t)}{\partial a} = -N(a, t)\, b(a), \tag{13}$$

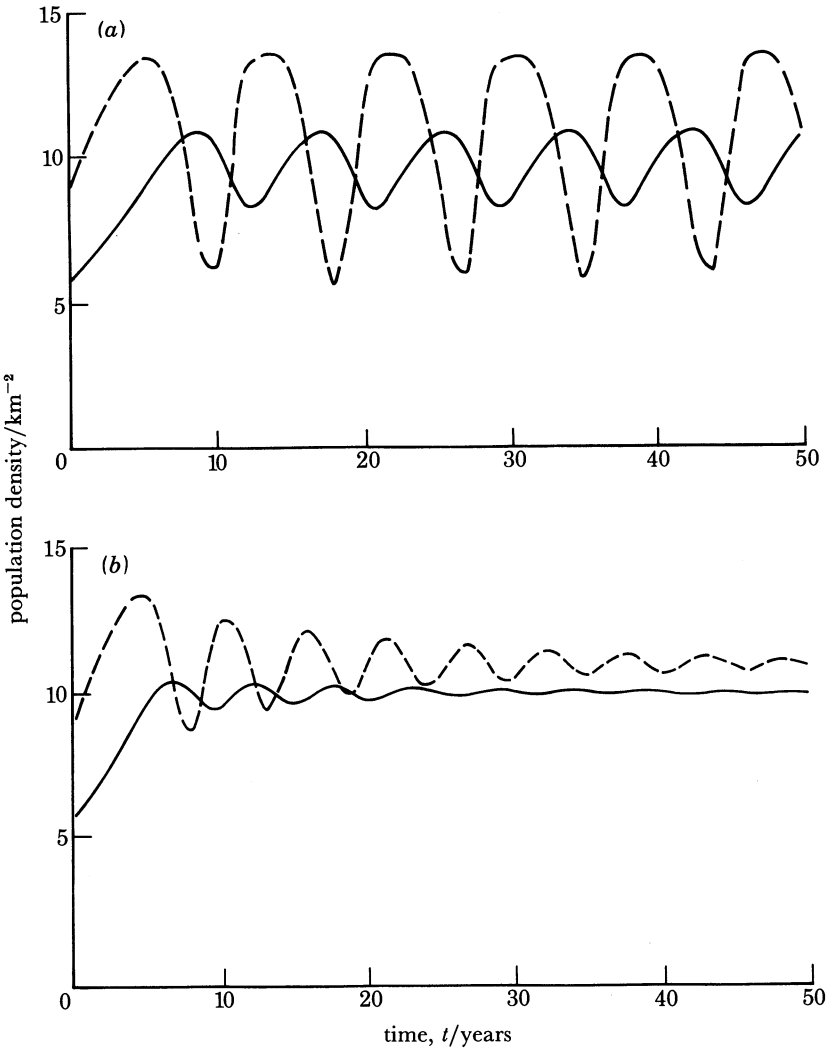

FIGURE 6. Age-structured difference equation models of badger population growth ((8) in main text). (*a*) A two-year maturation delay ($N^* = 10$, $k = 2$, $c = 7.0$, $\gamma = 1.82$, $\mu_0 = 0.65$, $\mu_1 = 0.35$, $\mu_2 = 0.25$). ——, adult density; – – –, cub density. Initial conditions; $N_0 = N_1 = N_2 = 5$. (*b*) Similar to (*a*) but with a one-year maturation delay ($k = 1$, other parameter values identical to (*a*) except $\mu_0 = 0.77$, $\mu_1 = 0.25$). Initial conditions: $N_0 = N_1 = 5$.

where $b(a)$ denotes the age specific mortality rates. Equation (13) has the formal solution

$$N(a, t) = N(a-t, 0) \exp\left[-\int_0^t b(a-t+x)\,\mathrm{d}x\right] \quad \text{if} \quad t \leqslant a \tag{14}$$

and

$$N(a, t) = N(0, t-a) \exp\left[-\int_0^a b(x)\,\mathrm{d}x\right] \quad \text{if} \quad t > a. \tag{15}$$

The first of these solutions (14) represents the 'dying of' of the population present at $t = 0$ (the initial condition). The second represents the mortality in subsequent cohorts of size $N(0, t-a)$ at time $t-a$ (the boundary condition). We therefore need to specify the boundary condition

(number of births at time $(t-a)$) in the light of our previous discussion on badger fecundity and its dependency on density. First note that the total population size $\bar{N}(t)$ is simply

$$\bar{N}(t) = \int_0^\infty N(a, t)\, \mathrm{d}a. \tag{16}$$

The renewal (boundary) condition may be expressed as follows:

$$N(0, t) = \int_0^\infty \gamma(a, \bar{N})\ N(a, t)\, \mathrm{d}a, \tag{17}$$

where $\gamma(a, \bar{N})$ denotes the age-specific, density-dependent birth rate. It can be shown that the steady-state of the model, $\bar{N}^*$ (that is, the equilibrium total density of badgers) must satisfy

$$\int_0^\infty \gamma(a, \bar{N}^*) \exp\left[-\int_0^a b(x)\, \mathrm{d}x\right] \mathrm{d}a = 1. \tag{18}$$

In other words, at equilibrium (if it exists) the average number of offspring born to a single individual (assuming at 1:1 sex ratio) must equal unity throughout the life span of that animal. Since males do not reproduce this implies that each female (on the basis of a 1:1 ratio) must produce on average exactly two individuals.

Further analytical treatment of this model is difficult given our need to include a maturation delay (that is, age classes (in units of one year) 1 and 2 do not reproduce) and the nonlinear nature of the 'mammal type' density-dependent function. We therefore used numerical methods to solve (15) and (16), given parameter values relevant to the population biology of the badger (see table 10). In these calculations we encorporated the further refinement of discrete breeding (that is, the fecundity term $\gamma(a, \bar{N}(t))$ is a function of both density, age and time of the year). Numerical studies of the model were carried out using the following parameter values: ten yearly age classes ($b(a) \to \infty$ for $a > 10$ years) with mortality rates of 65% for cubs ($0 \leqslant a < 1$), 35% for juveniles ($1 \leqslant a < 2$) and 25% for the remaining age classes ($2 \leqslant a \leqslant 10$). With cub production between mid January and mid March (with an overall yearly *per capita* birth rate of $\gamma = 0.6$ per year) and juveniles maturing in their second year of life, the system exhibits weakly damped oscillatory behaviour with an average period for the slowly damping oscillation of approximately six to eight years. The system can be tipped into limit cycle behaviour by alterations in the maturation delay and the values of the fecundity and mortality terms. In general, however, the relevant parameters suggest oscillatory behaviour since weak damping will probably be forced into permanent oscillations by stochastic effects (see Bartlett 1960; Nisbett & Gurney, 1982). Also note the similarity in behaviour with that predicted by the difference equation model and that of the time-delayed non-age-structured model (6). In particular, adult numbers show very small amplitude changes, while cub numbers oscillate widely from year to year. As in the case of the difference equation model, such behaviour arises as a consequence of the nonlinear density-dependent term and the maturation delay.

The suggestion of oscillatory behaviour emerging from model analysis is of some interest. In natural populations, the small scale fluctuation in adult numbers would, in practical terms, be difficult to detect. Cub density, however, oscillates more widely and hence it should in theory be detectable in natural populations provided the model assumptions and parameter estimates

accurately reflect reality. Unfortunately, to our knowledge, no long-term information on cub numbers is available at present to test this prediction. Future survey work could profitably focus on this aspect of badger population biology.

3. Epidemiology of bovine tuberculosis in badger populations

Information on the epidemiology of bovine tuberculosis in natural badger populations, and on the course of infection in individual animals, is limited at present. This section summarizes the available data with the dual objectives of identifying gaps in our knowledge and providing (where possible) parameter estimates for studies of transmission dynamics. In this latter context our interest focuses on the factors responsible for disease persistence and stability within the host population.

Transmission

New infections may arise when susceptible badgers come in contact with *Mycobacterium bovis* bacilli, either directly from an infectious animal, or indirectly from the environment. Transmission is most frequent via the respiratory route as a consequence of the inhalation of bacilli, although the infection can also be passed from animal to animal by biting. In a survey in Gloucestershire, for example, 14% of the observed cases of disease in badgers were thought to have arisen from bite wounds (Gallagher & Nelson 1979). Animals with severe lung lesions have high concentrations of bacilli in saliva and sputum and are therefore highly likely to transmit infection by biting uninfected badgers. The territorial and social behaviour of badgers, however, reduces the frequency of aggressive interactions. *M. meles* is highly territorial, but does not tend to stray frequently into surrounding territories and in general avoids direct contact with neighbouring animals at the boundaries. The chance of inhaling or ingesting live bacilli from the environment may appear low at first sight but the available evidence suggests that various factors conspire to make this the most common route of transmission. Diseased animals tend to contaminate their environment heavily with bacilli passed via faeces, urine, sputum and supurating bite wounds. Badgers spend a great deal of their active periods foraging on pasture for earthworms and also frequently visit locations on territory boundaries which are used as latrine pits for defaecation (these serve as territory markers) (Kruuk 1978). Such areas may therefore become heavily contaminated by bacilli. In addition, badgers urinate very frequently as a consequence of the high water content of their principal food item, the earthworm. Animals with kidney lesions can pass up to 300000 bacilli per millilitre of urine (M.A.F.F. 1979). The majority of bacilli may be killed quickly by exposure to direct sunlight, some may survive for periods of several weeks depending on the prevailing climatic conditions of their microhabitat. In general, warm, dark, moist locations appear optimal for bacterial survival (Maddock 1933; Williams *et al.* 1930; M.A.F.F. 1979) (see table 11). Within a contaminated habitat, the risk of becoming infected is solely determined by the statistical probability of inhaling or ingesting bacilli, but the risk of subsequent disease is highly variable and depends on many host factors.

The chance of contact with bacilli, the survival of bacilli, and the chance of aerial inhalation are all greatly increased as a consequence of the communal life badgers live underground and the conditions found within the sets. As social animals, relying on scent for identification,

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TABLE 11. THE VIABILITY OF *M. BOVIS* BACILLI IN BADGER PRODUCTS

badger product	viability: winter: *M. bovis* found in large numbers after	viability: winter: *M. bovis* recovered after	viability: summer: *M. bovis* recovered after
urine	1 week	1 month	3 days
sputum	1 month	2.5 months	1 week
faeces	—	1 month	2 weeks

Source: M.A.F.F. 1979.

badgers frequently nuzzle and examine each other (Neal 1977) facilitating the spread of disease within a social group.

Disease transmission between social groups can occur as a result of dispersal, territorial aggression or unusual behaviour induced by tuberculosis infection. Immature badgers, particularly the males, may disperse at the end of their first year of life and attempt to join new social groups. In the breeding season, adult males sometimes move into adjoining territories seeking sows in oestrus (Cheeseman & Mallinson 1979). Aggressive behaviour, particularly by males, increases during the breeding season, as part of territorial defence (Neal 1977). All of these behavioural factors enhance the likelihood of disease transmission between groups. In addition, severely diseased animals may exhibit abnormal behaviour. They are thought to wander further, and more frequently, through neighbouring territories (Cheeseman & Mallinson 1979).

The course of infection in individual animals

At present there is no effective method of accurately detecting *M. bovis* infection in live animals. Nevertheless, culture and examination of faeces, urine and sputum (Little *et al.* 1982) can assist by detecting infected badgers currently secreting bacilli. Culture techniques are labour intensive and subject to error. As such, information on the incubation and infectious periods of *M. bovis* in badgers is of poor quality.

Little *et al.* (1982) describe laboratory experiments on a small sample of artificially and naturally infected badgers (tables 11 and 12). Cheeseman *et al.* (1985) have monitored disease

TABLE 12. OBSERVATIONS ON EXPERIMENTALLY INFECTED BADGERS

days to faecal excretion of *M. bovis* following inoculation or exposure to infected badgers	days surviving after inoculation or post exposure	days surviving following first positive faeces swab
	experimentally infected badgers	
158	367 (k)	209
n.i.	402 (k)	n.k.
97	132 (d)	35
	badgers in contact with above	
95	170 (d)	75
4 badgers: n.i.	284–305 (k)	n.k.

Method: eight healthy badgers placed in three groups; one member of each group inoculated with *M. bovis*. all eight badgers yielded *M. bovis*. (Little *et al.* 1982.)

n.i., No isolation; n.k., not known; (d) died; (k) killed (both at end of experiment).

progression in natural populations by regular trapping and clinical examination. These authors also collected samples of faeces, urine, sputum and exudate from bite wounds for culture and examination. On the basis of the laboratory work, the best indicator of the duration of incubation is the time between first exposure to infection and the first recording of bacilli excretion. The range is from 95 days to 158 days (three to five months), although excretion of bacilli is at first intermittent (Little *et al.* 1982). This latter factor suggests caution in placing too much reliance on this method of estimation.

The duration of infectiousness appears to be life-long following first exposure although the rate of excretion of infective stages may vary greatly throughout the course of infection. As such the infectivity of disease animals varies in time and may be determined by extrinsic factors such as food availability (that is, nutritional status and general health of the host). Cough, extensive pulmonary or kidney lesions, and large numbers of bacilli in urine, sputum and faeces (entry into the digestive tract results from swallowing excretions from the respiratory system) distinguish those animals with tuberculosis who are most infectious. The effective duration of infectiousness, however, is approximately determined by the average life span of infected animals (excluding the incubation period). Recovery and the acquisition of immunity to reinfection do not appear to occur although the infection may become latent (inactive) in the sense that bacilli cease to be excreted in detectable quantities for certain periods of time. Life expectancy of infected animals appears highly variable with some badgers dying quickly in laboratory studies (after 35–75 days) while others survive for much longer (see tables 12 and 13). In the laboratory one badger survived for 3.5 years and five survived for between one and two years while excreting *M. bovis* bacilli (Little *et al.* 1982). In the field survey by Cheeseman *et al.* (1985), three animals were infectious for between one and two years.

TABLE 13. OBSERVATIONS ON NATURALLY INFECTED BADGERS

survival time from isolation of *M. bovis* to death		died (d) or killed (k) at end of experiment
days	years	
167	0.5	k (ill with tuberculosis)
372	1.0	k (ill with tuberculosis)
372	1.0	k (good condition at end)
973	2.7	k (ill with tuberculosis)
1305	3.6	k (good condition at end)
165	0.5	d (due to tuberculosis)
335	0.9	d (due to tuberculosis)
364	1.0	d (due to tuberculosis)
837	2.3	d (due to tuberculosis)
mean	543	1.5 (minimum value, as some badgers killed)

No information from four badgers, two of which died from tuberculosis.
Method: 13 badgers taken from an area of high tuberculosis prevalence and assumed to be naturally infected. *M. bovis* isolated from all badgers (Little *et al.* 1982).

The progression of the disease is again highly variable and in part seems to depend on the route of transmission. Infections induced by bite wound tend to pass rapidly into an acute fulminating infection with severe lesions (Gallagher & Nelson 1979). Infections caused by inhalation progress at a more variable pace and any lung lesion so caused tends to be

of a chronic nature (Gallagher & Nelson 1979). In *post mortem* surveys of diseased animals between 50% and 70% showed gross lesions (Gallagher & Nelson 1979; unpublished data from Gloucestershire Veterinary Investigation Centre). The remainder were considered to be inactive (latent) infections with minor lesins in lymph nodes, or no lesions at all (Gallagher & Nelson 1979).

The significance of bovine tuberculosis as a cause of badger mortality is unclear at present although general knowledge of *Mycobacterium* infections in other mammals (including man) would suggest that it is likely to be important. Gallagher & Nelson (1979) reported that in one sample of animals in Gloucestershire the infection was responsible for 40% of all 'natural' deaths. More recently, however, Cheeseman *et al.* (1985) report that in a population of 65 animals only four died of infection over a period of 2–2.5 years (table 14). A summary of the available information on incubation and disease induced mortality is presented in table 15.

TABLE 14. PROGRESSION OF INFECTION IN A DISEASED BADGER POPULATION

approximate age in years when: first caught excreting tuberculosis	approximate age in years when: last caught excreting tuberculosis	estimate of max. time in years excreting tuberculosis	comment
6.3+	8.1+	1.9	died tuberculosis; initial s.m.l. lesion regressed
1.6	3.3	1.7	died tuberculosis; initial s.m.l. lesion regressed
1.3	2.5	1.2	died tuberculosis
4.0	4.3	0.3	—
7.0	7.3	0.3	s.m.l. abscess reduced and subsequently negative, tuberculosis when caught
1.3	—	(0.5)	s.m.l. abscess reduced and subsequently negative tuberculosis when caught
1.8	—	—	died tuberculosis

Four badgers (aged 0.5, 1.3, 1.3, 2.0 years) only caught once with tuberculosis.

References: Cheeseman *et al.* (1985); C. Cheeseman (personal communication).
s.m.l., Submandibular lymph node.

TABLE 15. DISEASE PROGRESSION IN BADGERS

experimentally infected badgers ($n = 3$)	time to faecal excretion of *M. bovis* following inoculation or exposure to infection. 0.3–0.4 years
and badgers placed in contact with above sample ($n = 5$)	time from first isolation of *M. bovis* to death. 0.1–0.6 years
naturally infected badgers (in laboratory) ($n = 13$)	time from first isolation of *M. bovis* to death 0.5–3.6 years mean: 1.5 years
a diseased badger population (in field) ($n = 11$)	estimate of max. time when *M. bovis* isolated from badgers 0.3–1.9 years

Data from Cheeseman *et al.* (1985) and Little *et al.* (1982).

TABLE 16. PREVALENCE OF BOVINE TUBERCULOSIS INFECTION IN BADGERS

area	sample type	year	sample size	percentage prevalence
SW England	M.A.F.F.			
	badgers	1971–82	6318	13.2
	faeces	1971–82	8853	2.9
	M.A.F.F.	1982	1005	10.4
		1981	571	13.3
		1980	731	10.3
		1979	654	9.6
		1977 to Dec. 78	872	11.8
		Sept. 76 to Aug. 77	551	13.4
		July 71 to Aug. 76	1934	17.4
	public	1976–82	2438	3.8
Gloucestershire, Avon and Wiltshire	M.A.F.F. and public	1971	20	20.0
		1972	94	22.3
		1973	175	20.0
		1974	253	19.8
		1975	337	17.5
		1976	443	19.0
		1977	491	12.6
		1978	551	10.8
		1979	549	8.4
		1980	575	9.2
		1981	579	11.0
		1982	809	8.3
Gloucestershire	M.A.F.F.	1971–3	78	21
	r.t.a.		70	16
	n.d.		17	53
	total		**165**	**22**
	M.A.F.F.	1973–6	676	21.6
	r.t.a.		460	14.0
	n.d.		70	42.8
	total		**1206**	**19.3**
	trapping	1973–5	151	43.0 (range 18–58)
West Penwith	M.A.F.F.	June and July 73	49	4.1
Cornwall	trapping	Nov. 78	29	34.5
Avon	trapping	Jun. 79	40	20.0
Gloucestershire	trapping	Aug. 78	29	6.9
		Nov. 79	38	31.6
		1981–2	5 out of 30 social groups contained positive faeces samples. In a subsample of the population 4 out of 9 social groups contained positive badgers	
E and W Sussex	M.A.F.F	1978–80	114	7.0
	public	1982	70	1.4
Surrey	public	1976	6	33.0
		1979	14	7.1
Hereford & Worcester	public	1978	23	4.3
Staffordshire	trapping	1981–2	45	17.8
	public	1978	9	11.1
Shropshire	public	1982	17	5.9
Cumbria	public	1982	13	7.7
Dyfed	M.A.F.F.	1980	23	8.7
		1982	66	1.5
N Ireland	M.A.F.F.	—	50	36.0

Data from Zuckerman (1980); Cheeseman *et al.* (1981, 1985) and unpublished M.A.F.F. sources. See M.A.F.F. reports from 1971 to 1983.

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The prevalence of infection

Table 16 summarizes the available data on prevalence of infection in badger populations. For the whole of the southwest of England, the mean prevalence for 1971–81 is 14% (samples primarily consist of badgers killed in control programmes and in investigations of badger disease). This average figure, however, disguises some temporal and spatial heterogeneity (Cheeseman *et al.* 1981; Gallagher & Nelson 1979).

The first infected badgers in the U.K. were discovered in 1971. There is therefore too little information, as yet, to detect any long term oscillatory trends. The identification of oscillatory fluctuations in prevalence has obvious implications in the interpretation of the impact of control measures. Analysis of the data is made complicated by the increased effort by M.A.F.F. scientists over the period 1971 to 1984 in sampling badger populations. In addition, badger control programmes instigated in 1976 further complicate the interpretation of natural trends in endemic infection within badger populations. It is clear from table 16, however, that differences occur from year to year in prevalence within defined regions. Most importantly a trend is discernable in the amalgamated data for Gloucestershire, Avon and Wiltshire where prevalence appears to have declined from an average level of around 20% in 1971 to 1976 to around 10% in the period 1977–82 (figure 7). It seems highly probable that this is a direct consequence of control measures which reduced overall badger density by the removal of infected social groups of animals

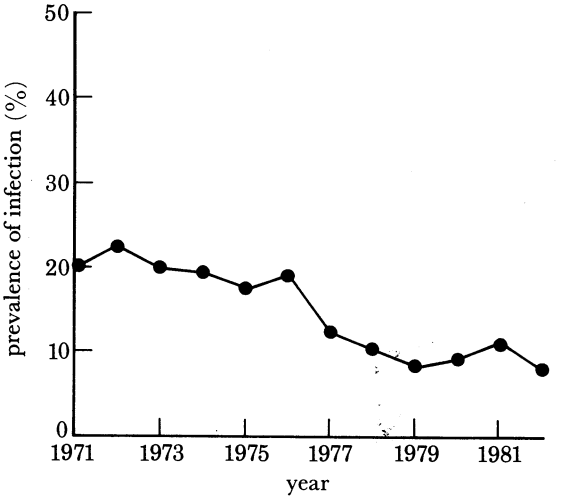

FIGURE 7. Changes in the prevalence of bovine tuberculosis in badger populations in Gloucestershire, Avon and Wiltshire from 1971 to 1982 (see table 16).

Data on the incidence of bovine tuberculosis in cattle herds in the southwest of England from 1960 onwards, suggests a possible oscillation of between eight and twelve years (J. Wilesmith, personal communication) (figure 8). This may well reflect similar natural fluctuations in prevalence within the reservoir host (the badger) or cyclic changes in badger abundance. The data is insufficient, however, for statistical analysis (time series methods) to ascertain the significance of the apparent cyclic fluctuations.

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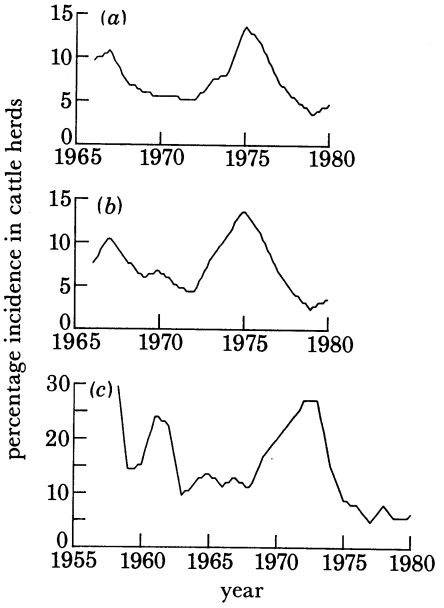

FIGURE 8. The incidence of bovine tuberculosis in cattle herds in southwest England (J. Wilesmith (personal communication)). The percentage of herds with reactors (visible lesion and non-visible lesion) in (*a*) all control areas in southwest England; (*b*) Gloucestershire; (*c*) West Penwith.

Prevalence by age and sex

Age prevalence data is of great value in the determination of the net force or rate of transmission within host communities (see Anderson 1982). Unfortunately, the problems surrounding the accurate determination of adult badger age hinder the acquisition of such information as does the absence of a reliable diagnostic test for infection in live animals. Table 17 summarizes the available data. It is important to note, however, that a comparison between tables 5 and 17 suggests that samples collected for prevalence determination are biased towards older animals (the young animals are under-represented in the samples with respect to their proportional contribution to overall population size).

With this caution in mind, the two sets of data show an overall trend for increased prevalence with age, although a plateau is attained fairly rapidly after birth. The greatest *per capita* rate of acquisition of infection therefore occurs in the young age classes (up to two years) (table 17).

TABLE 17. BADGER AGE–DISEASE PREVALENCE IN GLOUCESTERSHIRE

	1973–5		1977–9		
age class (years)	sample size	percentage infected	sample size	percentage infected	age class (years)
0–1	81	13.6	284	7.8	0–1
1–2	69	24.6	168	6.5	1–2
2–3	109	17.4	191	8.9	2–3
3–4	309	16.5	254	10.2	3–4
aged	188	27.1	215	9.8	4–5
			68 } 559	8.8 } 12.7	5–6+
			276	15.9	aged
total	**756**	**19.8**	**1456**	**9.2**	total

Unpublished data from S. Gallagher and M.A.F.F.

Young cubs can be infected at an early age; advanced lung lesions have been found in cubs aged four to five months (Zuckerman 1981). The high prevalence of infection in cubs reported in many surveys suggests that new births in diseased social groups of animals have a very high risk of acquiring the infection (Cheeseman *et al.* 1981). This observation hints at a form of 'pseudo' vertical transmission with passage of infection from parent to new born offspring.

Differences in prevalence between the sexes are not marked although a study by Gallagher & Nelson (1979) reports a consistent trend from slightly higher levels of prevalence in males (average 22%) when compared with females (17%). This may reflect the wider ranging movement and activity of males in the habitat.

Threshold density for infection persistence

Bovine tuberculosis in badgers is most commonly found in the southwest of England where average badger densities are generally higher than other regions of Britain. This observation, in a qualitative sense, hints at a relationship between host density and prevalence (Zuckerman 1981): an association that is well established for many other infectious diseases of plants, animals and man. Specifically, for most disease agents, there exists a critical host density necessary for the persistence of the pathogen within the community (see Anderson & May 1979; May & Anderson 1979). Its precise level is determined by many factors, which include the duration of infectiousness, the length of the incubation period, the magnitude of natural and disease-induced mortality, the mode and rate of transmission between hosts and climatic factors. For many infections, the notion of this critical density provides a focus for quantitative work on the design of control measures (see Anderson *et al.* (1981) in the context of fox rabies and Anderson & May (1982) in the context of childhood bacterial and viral infections of man).

In the case of bovine tuberculosis, the *quantitative* evidence for the existence of such a phenomena, and indeed for an association between density and prevalence, is at present very limited. On the one hand work by Gallagher & Nelson (1979) in Gloucestershire suggests a relationship between density and prevalence, while on the other hand, studies by Cheeseman and coworkers in Gloucestershire, Avon, Cornwall and Staffordshire show no consistent trends (Cheeseman *et al.* 1981; C. Cheeseman, unpublished information). As illustrated in figure 9, data from five surveys provides insufficient evidence to reach a conclusion.

Circumstantial evidence for the existence of a threshold density is provided by a comparison of the incidence of infection in cattle herds in the southwest and badger set density (Wilesmith 1983). In Cornwall, Gloucestershire and Avon, where the set density averaged over 30–50 sets per 100 square kilometres, there was a significant increase in the number of cattle herd infections (termed 'breakdowns') thought to have arisen from contact with badgers as opposed to the purchase and introduction of infected animals from other areas or countries (figure 10). This may reflect a threshold badger density for disease transmission between badger and cattle populations, and possibly for the maintenance of endemic infection in badger communities. Based on Neal's assumption of an average of five badgers per set (Neal 1977), 30–50 sets per 100 square kilometres is approximately equivalent to 1.5–2.5 badgers per square kilometre. These figures, however, should be interpreted with caution since an association between set density and the frequency of herd breakdowns may well reflect a combination of other factors. These include cattle density, habitat type (for example, the proximity of badger territories to grazing pasture) and farm management practices which clearly vary in different geographical locations in Britain. These issues will be discussed further in the concluding section of this paper.

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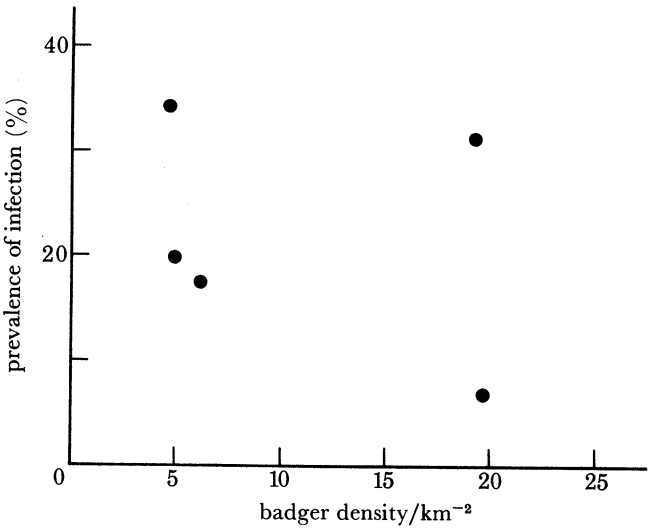

FIGURE 9. A plot of disease prevalence (percentage) as a function of badger density (per square kilometre) for five study sites (Cheeseman *et al.* 1981; C. Cheeseman, unpublished).

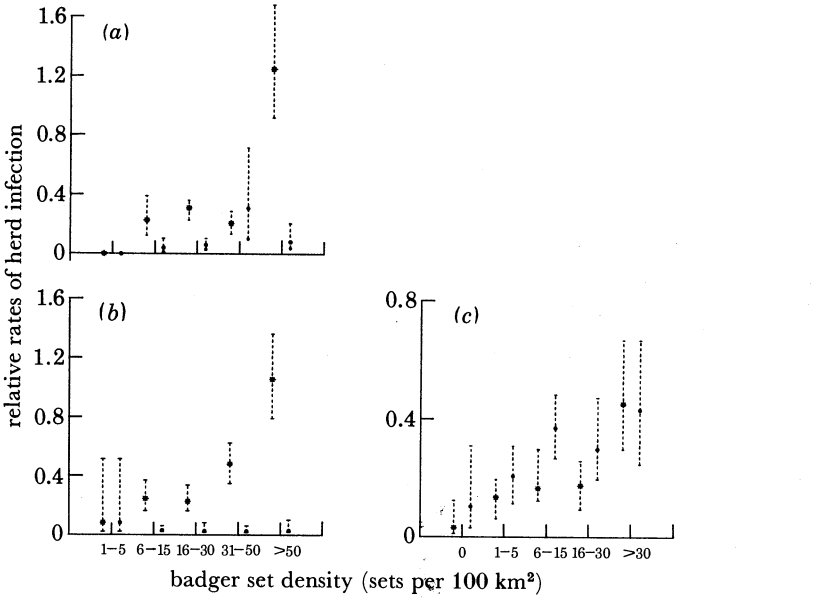

FIGURE 10. Herd infection in relation to badger set density, taken from Wilesmith (1983). The relative rates of herd infection in (*a*) Cornwall; (*b*) Gloucestershire and Avon; (*c*) England and Wales, excluding the southwest; by badger set density (number of sets in an area 10 km × 10 km). *, Herds with an unknown source of infection and herds associated with infected badgers; ●, herds infected by the purchase of animals (including Irish animals). Vertical bars indicate 95% confidence limits. Herd infection is defined as the number of infected herds in 1972–78 divided by the number of herds in a random sample of (*a*) 700, (*b*) 600 and (*c*) 500.

4. TRANSMISSION DYNAMICS AND MATHEMATICAL MODELS

It is clear from the preceding section that our understanding of the epidemiology of *M. bovis* infection is limited, both in context of endemic maintenance within badger populations and of transmission between cattle herds and communities of reservoir hosts. In this section we use simple mathematical models of disease transmission in an attempt to improve on this understanding. The aims of model construction and analysis centre on the identification of gaps

in the available data, on generating hypotheses that are testable against field data and on gaining some insight into the likely impact of control measures.

Model structure

Past work on the dynamics of bacterial infections has, to a large extent, been concerned with diseases of man. Reviews of the relevant literature are provided by Bailey (1975) and Cvjetanovic *et al.* (1978). Deterministic models of the dynamics and control of tuberculosis in human communities have been developed by Waaler *et al.* (1962), Mahler & Piot (1966*a*, *b*), Brøgger (1967), Revelle *et al.* (1967), Lyn & Revelle (1968), Waaler (1968*a*, *b*), Waaler & Piot (1969, 1970), Revelle & Male (1970) and Feldstein *et al.* (1973).

The generic feature of these studies is the use of a compartmental multistate framework in which the human population is divided into a series of infection states. Most commonly these divisions denote susceptible (uninfected) individuals, infected individuals who are latent (the infection is 'inactive') and infected individuals who have active infections and are infectious to other people (excreting bacilli). The net rate of infection transmission is generally assumed to be proportional to the density of susceptible hosts multiplied by the density of infectious individuals (the so-called mass action principle: see Bailey (1975) and Anderson (1982)). A further feature of these models is the assumption that active cases may both recover to become inactive and relapse to again become infectious.

Basic model

Models of this general structure have been used in the study of animal infections, such as rabies in fox populations, where disease transmission is embedded in a framework that takes account of dynamic changes in host abundance (Anderson & May 1979; Anderson 1979; Anderson *et al.* 1981). We begin by considering a very simple model in which the badger population is divided into three classes denoting susceptible animals ($X(t)$), infected but not yet infectious animals ($H(t)$) and infectious animals ($Y(t)$) (figure 11). As badgers are not

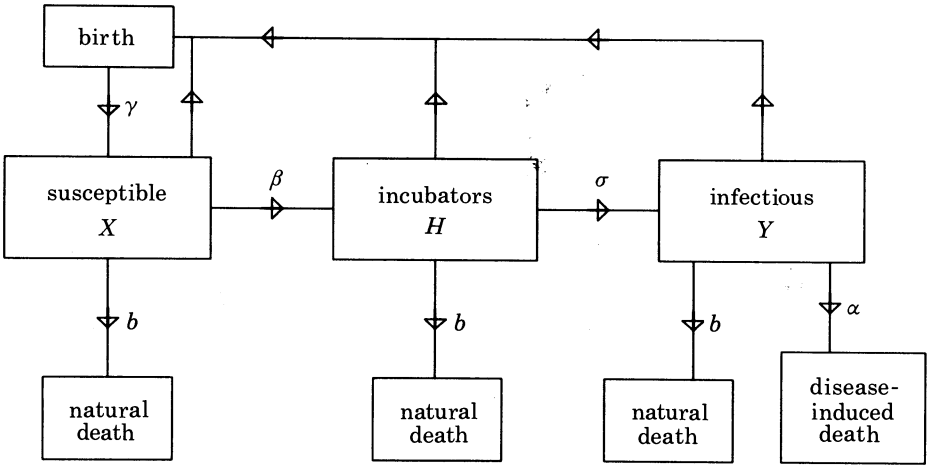

FIGURE 11. Diagrammatic flow chart of the badger–bovine tuberculosis system, with susceptible (X), infected (H) and infectious (Y) hosts. The flow of individual hosts between compartments is controlled by the following rate parameters; γ is the *per capita* birth rate; b is the natural death rate; β is the disease transmission coefficient; σ is the rate at which incubating individuals become infectious ($1/\sigma$ is the incubation period); α is the disease-induced death rate ($1/(\alpha+b)$ is the life expectancy of diseased badgers).

thought to acquire immunity to reinfection (M.A.F.F. 1979) we exclude an immune state. The total density of badgers at time t, $N(t)$ is therefore the sum of X, H and Y.

In the absence of tubercular infection we assume that the dynamics of the disease-free population are captured (approximately) by (5), in which it is assumed that density-dependent checks on growth act on the birth rate. To start with we ignore age structure, partly for the sake of simplicity and partly as a consequence of the ability of (5) to mimic crudely the growth of badger populations following disturbances (figure 1). When the disease is present, and using the mass-action assumption for the net transmission of infection (βXY where β is a transmission coefficient), we arrive at a system of three nonlinear differential equations for the rates of change of X, H and Y with respect to time:

$$\mathrm{d}X/\mathrm{d}t = \hat{r}(N)N - bX - \beta XY, \tag{19}$$

$$\mathrm{d}H/\mathrm{d}t = \beta XY - (b+\sigma)H, \tag{20}$$

$$\mathrm{d}Y/\mathrm{d}t = \sigma H - (b+\alpha)Y. \tag{21}$$

It is here assumed that infected but latent animals become infectious at a rate σ (average latent period, $1/\sigma$) and that infectious animals do not 'recover' to a latent inactive state. Infectious animals are assumed to have an increased mortality rate (α) over that suffered by susceptible and latent badgers; all mature animals are taken to contribute to the reproductive effort of the population. The term $\hat{r}(N)$ defines the *per capita* density-dependent fecundity function. A summary of the various populations and parameter defined in (19) to (21) is presented as a flow chart in figure 11. We refer to these equations as the basic model and in latter sections modifications are made to this framework to take account of additional biological processes and assumptions.

Investigation of the local stability properties of the model, by analytical methods, is made difficult by the nonlinear fecundity term. Two important properties emerge, however, by means of direct inspection of the structure of (20) and (21). Following the introduction of a single infectious animal into a population of K susceptibles, the pathogen will only perpetuate provided $\mathrm{d}Y/\mathrm{d}t > 0$. For this to occur the density of susceptibles must exceed a critical value, K_{T}, where

$$K_{\mathrm{T}} = [(b+\alpha)(b+\sigma)]/\sigma\beta. \tag{22}$$

The notion of a critical density of susceptible hosts is linked to a further concept of great value in epidemiological study. The basic reproductive rate of infection, R_0, is defined as the average number of secondary cases generated by one primary case of infection in a susceptible population of density X. For the basic model R_0 is defined as

$$R_0 = (\beta X\sigma)/[(b+\alpha)(b+\sigma)] \tag{23}$$

(see Anderson & May 1981; Anderson *et al.* 1981; Anderson 1982; Dietz 1976). The value of R_0 must equal or exceed unity if the pathogen is to persist. Equation (23) simply states that the number of secondary cases is equal to the rate at which cases arises in a population of X susceptibles, multiplied by both the proportion that survive to become infectious ($\sigma/(\sigma+b)$) and the life expectancy of an infectious animal ($1/(b+\alpha)$). Note that the condition for K_{T} (22) arises directly from the constraint that $R_0 \geqslant 1$ for pathogen persistence. At equilibrium the effective reproductive rate $\bar{R}$ is equal to unity.

Three properties of the model are of particular interest. First, if the infection is able to persist

($X \geqslant K_T$ or $R_0 \geqslant 1$), the disease suppresses badger density below that which would have been obtained in its absence (the disease-free habitat carrying capacity, K). We define the degree of depression d, as $d = 1 - N^*/K$, where N^* is equilibrium badger density in the presence of tuberculosis (figure 12). The value of d increases as the habitat carrying capacity rises. Second, the prevalence of infection at equilibrium, y^* (where $y^* = Y^*/N^*$) also rises as the carrying capacity K increases. The model predicts that in areas of high badger density before the introduction of infection, the disease will be more prevalent once established than in areas of poor badger habitat (figure 12). Third, for parameter values relevant to the badger–bovine tuberculosis interaction (for a summary see table 18) numerical studies reveal that the system of equations is stable to small perturbations and exhibits damped oscillations to the equilibrium state (figure 13). The average period of the oscillation is between 10 and 15 years depending on the precise values of the parameters (in particular K and the severity of density dependence, c).

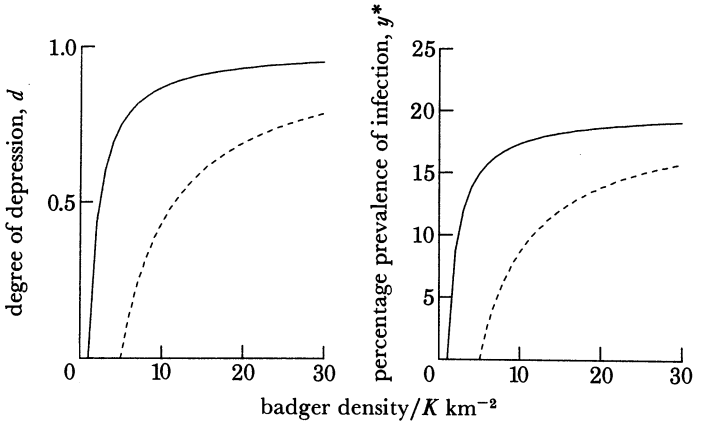

FIGURE 12. The degree of disease-induced badger population depression, d, and the equilibrium prevalence of infection, y^*, for the basic disease model (as defined by equations (19)–(21)). For model parameters, see table 17. ——, $K_T = 1$ km^{-2}; – – –, $K_T = 5$ km^{-2}.

TABLE 18. PARAMETER VALUES USED IN THE DISEASE MODELS

σ, the *per capita* rate of transfer from the incubating to infectious state (where $1/\sigma$ is the incubation period = 3 months)	= 4 per year
α, the *per capita* disease induced mortality rate	= 1 per year
K_T, the threshold density for persistence of the disease	= 1 and 5 km^2
β, the disease transmission coefficient (varies according to K_T)	= 0.308 km^2 per year, if $K_T = 5$ km^{-2} = 1.54 km^2 per year, if $K_T = 1$ km^{-2}
unless otherwise stated, the disease-free carrying capacity is set at	$K = 10$ km^{-2}
per capita birth rate = 0.6 per year, *per capita* natural death rate = 0.4 per year, giving intrinsic growth rate	$r = 0.2$ year^{-1}

The relevance of these predictions to natural populations is difficult to assess in the absence of detailed longitudinal data of disease prevalence and badger abundance. The predicted degree of depression of badger density appears high given the current belief that infection has little impact on host abundance. The predicted levels of prevalence, however, are in general accord with observed values (see figure 12 and table 16), for a range of assumptions concerning the

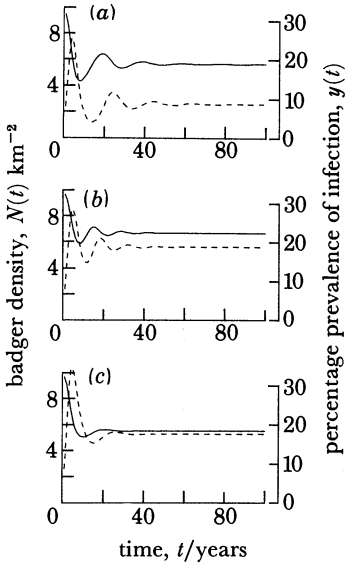

FIGURE 13. Numerical solution of the basic disease model (equations (19)–(21)) for two values of the threshold density, K_T. For model parameters, see table 18. ——, badger density $N(t)$ at time t; – – –, prevalence of infection $y(t)$. (*a*) $c = 1$, $K_T = 5$ km^{-2}; (*b*) $c = 7$, $K_T = 5$ km^{-2}; (*c*) $c = 7$, $K_T = 3.7$ km^{-2}.

disease-free carrying capacity of the badger habitat. No oscillatory trends in disease prevalence have been recorded, but data is sparse at present.

In our assessment of the relevance of model behaviour, we are severely hampered by the limitations of the data base. Note, that the parameter values recorded in table 16 are at best crude estimates (σ and α), and at worst, simple guesses (the value of K_T). Altering the values of the incubation period ($1/\sigma$) and the disease induced mortality rate (α), for example, has a significant effect on the predicted levels of badger abundance and disease prevalence (figure 14). In light of these problems we attempt therefore to gain some qualitative understanding of the robustness of model predictions to a variety of assumptions concerning disease transmission and persistence.

Vertical transmission

Within infected social groups, the close contact between mother and cubs places new born animals at a high risk of infection during early life (Cheeseman *et al.* 1981). This route of transmission may be termed pseudo-vertical since there is no evidence to suggest that cubs are born with the infection (see flow chart in figure 15). We assume that the interval between birth and the acquisition of infection is of very short duration. The basic model can be altered to take account of this modification, where

$$\mathrm{d}X/\mathrm{d}t = f(N)\,[X+H+(1-p)\,Y]-bX-\beta XY, \tag{24}$$

$$\mathrm{d}H/\mathrm{d}t = f(N)\,Yp+\beta XY-(b+\sigma)\,H, \tag{25}$$

$$\mathrm{d}Y/\mathrm{d}t = \sigma H-(b+\alpha)\,Y, \tag{26}$$

where p denotes the proportion of cubs produced by infectious mothers that acquire the infection soon after birth. The net effective of pseudo-vertical transmission is to lower the critical

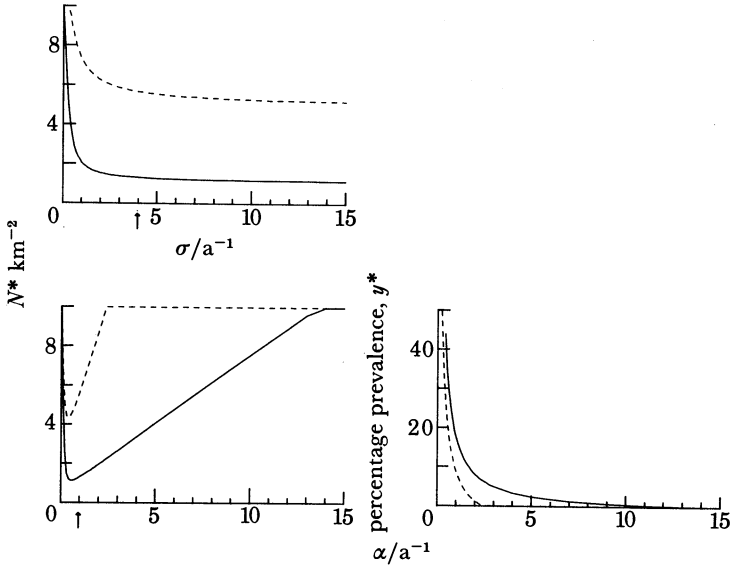

FIGURE 14. The effect of altering α and σ on the equilibrium badger population density, N^* and the disease prevalence, y^*, for two values of the threshold density K_T (basic disease model, equations (19)–(21)). For model parameters, see table 17. ——, $K_T = 1$ km^{-2}, $c = 1$; – – –, $K_T = 5$ km^{-2}, $c = 1$. Arrow marks ($1/\sigma = 3$ months) on top graph, and ($1/\alpha = 1$ year) on bottom graph.

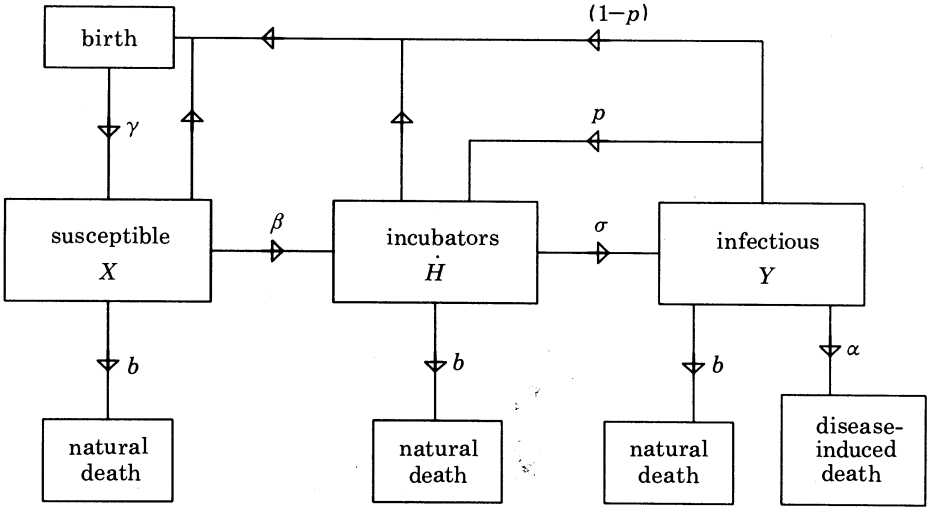

FIGURE 15. Diagrammatic flow chart of the badger–bovine tuberculosis system, with pseudo-vertical transmission of the disease from parents to newborn offspring. p, proportion of offspring produced by infectious parents (Y), which pass directly into the incubating class (H).

threshold density of susceptibles, K_T, for disease persistence. For example in the case of a logistic form for the density-dependent function, $f(N)$ (for example, $c = 1$), K_T is defined as

$$K_T = [(b+\sigma)(b+\alpha) - \gamma p\sigma]/[(\beta - \gamma p)\sigma]. \quad (27)$$

Note that if p is high in relation to other parameter values, then $K_T < 0$ which implies that the disease will persist irrespective of the prevailing density of susceptible animals (provided $R_0 \geqslant 1$). Numerical studies reveal that as a consequence of a high component of vertical

transmission (p close to unity in value) the period of the damped oscillations increases as does the damping time to equilibrium.

Reservoir of infection on the pasture

Badgers may contract infection from contaminated pasture as well as by direct contact with infectious animals. The rate of horizontal transmission via the former route will be proportional to the density of live bacilli on the pasture multiplied by the density of susceptible animals foraging in the contaminated area. If we define $W(t)$ as the density of tubercule bacilli in the habitat at time t, then the basic model may be modified as follows (see figure 16):

$$dX/dt = f(N)N - bX - \beta_2 XW - \beta_1 XY, \tag{28}$$

$$dH/dt = \beta_2 XW + \beta_1 XY - (b+\sigma)H, \tag{29}$$

$$dY/dt = \sigma H - (b+\alpha)Y, \tag{30}$$

$$dW/dt = \lambda Y - \mu W - \beta_2 NW. \tag{31}$$

Here β_1 and β_2 are transmission coefficients which represent, respectively, infection by badger–badger contact and infection by badger–bacilli contact. The parameter λ denotes the

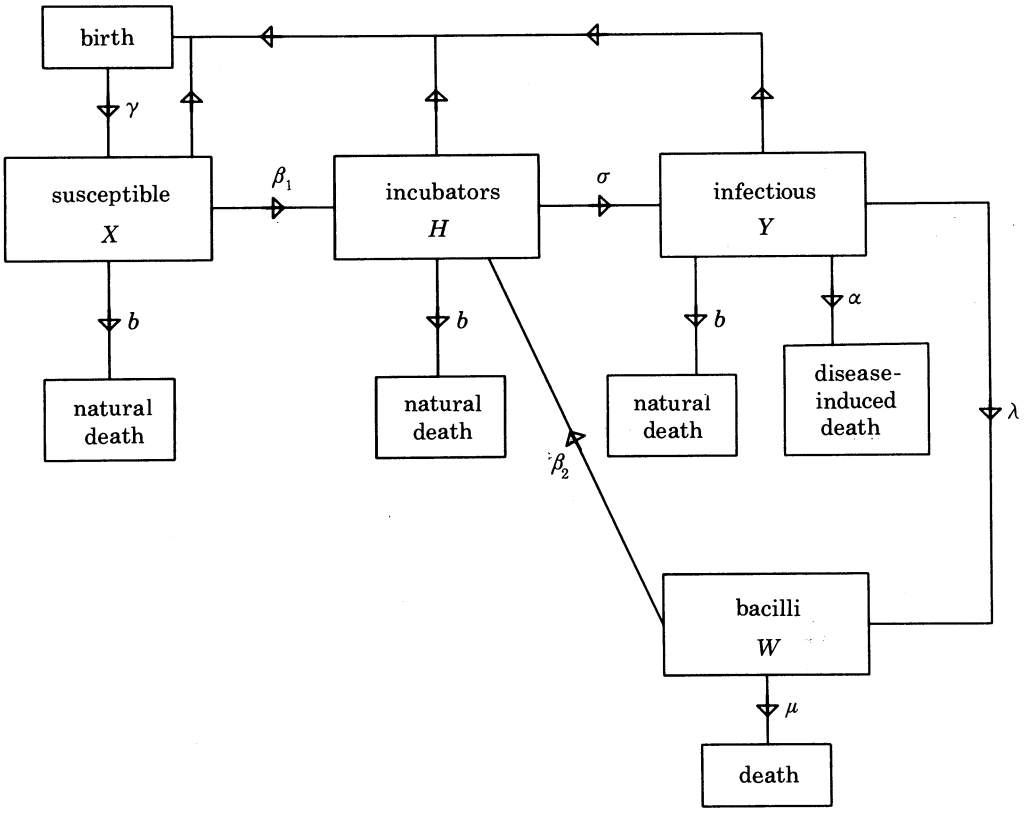

FIGURE 16. Diagrammatic flow chart of the badger–bovine tuberculosis system, with disease transmission both directly between badgers, and from contaminated pasture. W, Density of tubercle bacilli in the environment; λ, *per capita* rate of bacilli production by an infectious badger; β_1, disease transmission coefficient for infection via badger to badger contact; β_2 disease transmission coefficient for infection derived from contaminated pasture.

average *per capita* rate of excretion of bacilli by infectious animals and $1/\mu$ denotes the typical life expectancy of the bacilli on the pasture.

In general, the survival attributes of bacilli are poor, particularly during the summer months (see table 11). The characteristic time scales, on which dynamical changes occur, are therefore very different for the various equations of the model ((28) to (31)). Changes in $W(t)$ occur rapidly (a time scale of a few weeks) while changes in $H(t)$ and $Y(t)$ occur more slowly (a matter of months to years). As such, the density of bacilli will essentially be instantaneously adjusted to the equilibrium state (W^*) in relation to the slower time-scale changes in X, H and Y. We may therefore reduce the dimensionality of the model by setting $\mathrm{d}W/\mathrm{d}t = 0$ and substituting for W^* in (28) and (29). The net effect of such modifications on the dynamical properties of the basic model are negligible. Differences will only result if the bacilli have much longer life expectancies on pasture (or in badger sets or woodland habitat) than is suggested by the available data to date (see table 11). Long-lived invective stages tend to enhance the oscillatory behaviour of models of disease dynamics (see Anderson & May 1981).

Carriers

Many bacterial infections, such as tuberculosis, are characterized by the occurrence of apparently healthy individuals (with no overt symptoms of disease) who are actively excreting infective stages and are thus able to transmit the disease to susceptible hosts (Cvjetanovic *et al.* 1978). The highly variable infectious period of bovine tuberculosis in badgers (see tables 12 and 13) and the presence of non-visible lesions in infected but apparently non-infectious and otherwise healthy animals (often 30–60% of the infected population), suggest that the carrier state may be an important feature of transmission dynamics (figure 17). In general, carriers act to enhance the ability of bacterial infections to persist endemically within low density host populations (see Bailey 1975; Cvjetanovic *et al.* 1978).

We modify the basic model to encorporate two classes of infectious animals, Y_1 and Y_2, where Y_1 denotes infected animals with overt signs of disease (with a disease induced mortality rate α_1) and Y_2 denotes carriers (with a disease induced mortality rate α_2).

$$\mathrm{d}X/\mathrm{d}t = f(N)\,N - bX - X(\beta_1 Y_1 + \beta_2 Y_2), \tag{32}$$

$$\mathrm{d}H/\mathrm{d}t = X(\beta_1 Y_1 + \beta_2 Y_2) - (b+\sigma)\,H, \tag{33}$$

$$\mathrm{d}Y_1/\mathrm{d}t = f\sigma H - (b+\alpha_1)\,Y_1, \tag{34}$$

$$\mathrm{d}Y_2/\mathrm{d}t = (1-f)\sigma H - (b+\alpha_2)\,Y_2. \tag{35}$$

Here $(1-f)$ denotes the fraction of the incubators who become carriers of the disease. It is assumed that in general $\alpha_2 \ll \alpha_1$ since the carriers show no signs of overt disease. The basic reproductive rate of infection, R_0, is

$$R_0 = \frac{\beta_1 X f\sigma}{(b+\alpha_1)\,(b+\sigma)} + \frac{\beta_2 X(1-f)\sigma}{(b+\alpha_2)\,(b+\sigma)} \tag{36}$$

and the critical density for disease maintenance, K_T, is

$$K_T = X/R_0. \tag{37}$$

Note that the presence of carriers (given that $\alpha_2 < \alpha_1$) tends to increase the value of R_0 and reduce the value of K_T when compared with the model in which all infected animals show overt

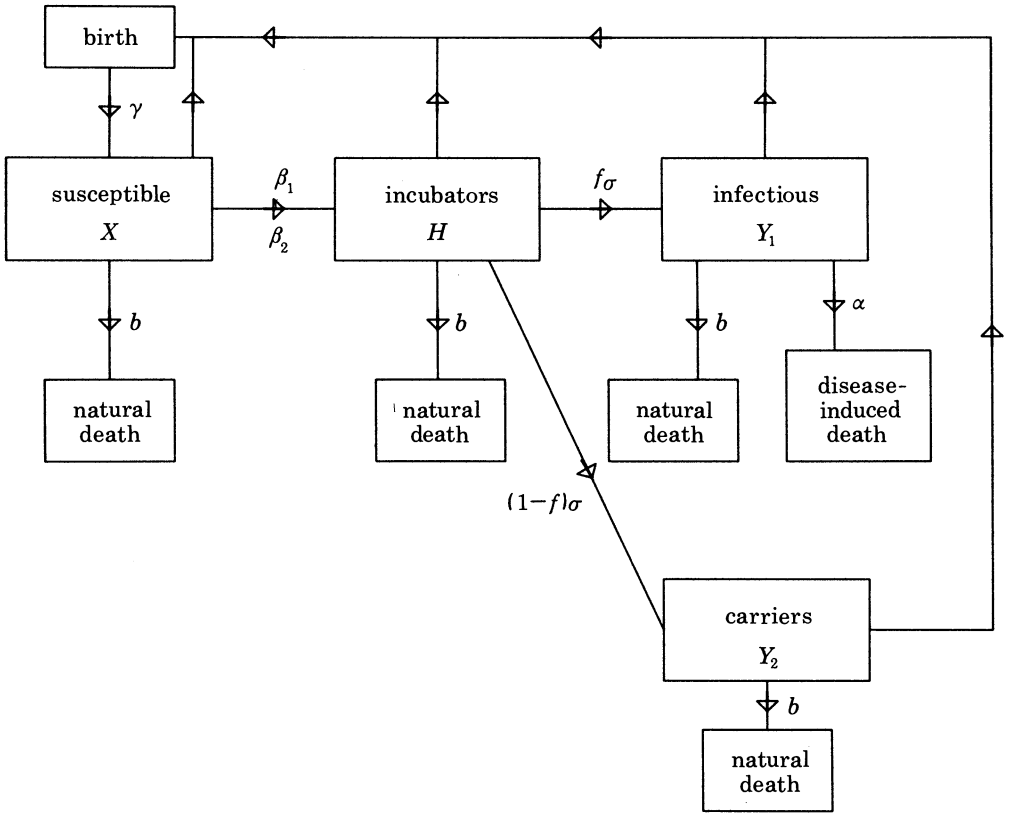

FIGURE 17. Diagrammatic flow chart of the badger–bovine tuberculosis system, including a carrier class of infectious badgers (Y_2). f, Fraction of incubators which become infectious individuals (Y_1), with a high transmission coefficient β_1. $(1-f)$ is the fraction of incubators that become carriers (Y_2), with a lower transmission coefficient β_2.

signs of disease (that is, all suffer a disease induced mortality rate of α_1). At equilibrium the ratio of carriers to non-carriers is

$$Y_2^*/Y_1^* = [(1-f)\,(b+\alpha_1)]/[f(b+\alpha_2)]. \tag{38}$$

A crude estimate of the proportion of carriers in natural populations may be derived from a comparison of the numbers of infected animals with visible and non-visible lesions at autopsy (Gallagher & Nelson 1979). Between 30 and 60% of infected badgers examined *post mortem* in the southwest of England had non-visible lesions. These observations are somewhat biased by the presence of infected but incubating animals, since it was not known what proportion of the animals examined were infectious (excreting bacilli) before death.

Numerical studies reveal that the presence of carriers acts to reduce the impact of the infection on badger density (since $\alpha_2 < \alpha_1$), reduce the tendency for oscillatory behaviour, enhance disease persistence in low density populations and increase the overall prevalence of infection. It can be seen from (38) that small values of f increase the proportion of carriers and the overall prevalence of infection.

Inactive cases

As noted earlier in this paper, infected badgers sometimes cease to release detectable numbers of bacilli (in urine, faeces or sputum) and therefore the infection becomes essentially inactive

with respect to transmission. Evidence from field studies suggests that inactive cases may relapse to the active state and again become infectious to uninfected animals. Intermittent excretion of bacilli by infected animals is often observed in field studies; this may be either a consequence of inadequacies in the methods for detection of bacilli or a result of active cases becoming inactive and vice versa. A simple modification to the basic model, along similar lines to that made for carriers of infection, can mimic this process. If we define δ as the *per capita* rate at which infectious animals become inactive, Λ as the *per capita* rate at which they relapse to the infectious state, and α_1 and α_2 as the disease-induced mortality rates of infectious and 'inactive' animals (where $\alpha_1 > \alpha_2$) then the basic reproductive rate of infection, R_0, becomes

$$R_0 = \frac{\beta X \sigma (\delta + b + \alpha_2)}{(b+\sigma)\,[(b+\alpha_1)\,(\delta+b+\alpha_2)+\Lambda(b+\alpha_2)]}. \tag{39}$$

The critical density of susceptibles for disease persistence, K_T, is as defined in (37).

The presence of inactive cases acts to reduce the value of R_0, increase the critical density for disease persistence and increase the equilibrium prevalence of infection (since $\alpha_2 < \alpha_1$).

Seasonality in transmission and host reproduction

The birth rate of the badger is clearly seasonal, with cub production being limited to the months of January, February and March. It has been suggested that disease incidence is also seasonal although quantitative information is unavailable at present. Certain factors may, however, conspire to induce a seasonal element in transmission. Male aggressive behaviour, for example, is greatest in the spring months when territories are actively defended during the breeding season (Kruuk 1978). Similarly, the movement of males into neighbouring territories is greatest during the periods of search for females in oestrus (Cheeseman & Mallinson 1979). Juvenile dispersal is also seasonal, occurring mainly in the spring months. Finally, the survival of bacilli on pasture is greatest in the winter and spring months when climatic conditions favour persistence in the surface soil (see table 11 and M.A.F.F. 1979).

In the absence of precise quantitative data on seasonal processes it is difficult to assess their impact on disease prevalence and badger abundance. Numerical studies reveal two points of general interest. First, seasonality in birth (the inclusion of a time-dependent fecundity term) acts to induce seasonal fluctuation in total abundance but has little effect on the long-term damped oscillatory behaviour. This is a direct result of the relative time scales of the two oscillatory processes; seasonal changes act on a short time scale with respect to the longer term oscillations (10–15 years) induced by the dynamical interaction between host and pathogen populations. Seasonal changes in transmission have similar effects but they may tend to lengthen the damping period of the longer term oscillations (depending on their amplitude and phase).

Stochastic models

So far our discussions have centred on deterministic models which take no account of chance elements in the timing of birth, death and infection events. Monte Carlo methods can be used to examine the impact of demographic stochasticity (see May 1973; Bartlett 1960; Pielou 1969) on temporal changes in badger abundance and disease prevalence. Such processes will be of great importance in determining events within small populations of animals and in small areas of badger habitat.

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Numerical studies of a basic stochastic model (a direct equivalent of the basic deterministic model), were carried out using Monte Carlo techniques (see Pielou 1969). A large number of simulations were performed with the habitat size fixed at 100 km². The initial badger population size was varied, in different simulation runs, by altering the carrying capacity, K, of the habitat. Values of 1, 3, 5, 10 and 20 km^{-2} were used, giving absolute population sizes in an area of 100 km² of 100, 300, 500, 1000 and 2000 animals, respectively. Twenty simulation runs (with a different seeding of random numbers) were performed for each density. The results are displayed in figure 18 as frequency distributions of the time to the extinction of the disease within the habitat. A summary graph is also depicted in figure 18, showing the probability of extinction of the infection as a function of habitat carrying capacity K within 30 years, 50 years and 100 years after introduction. Owing to the apparently low pathogeneity of bovine tuberculosis in badgers (when compared with infections such as rabies), the disease is able to persist for reasonable periods of time (without introductions from surrounding habitats) within moderately sized populations (that is, 300–500 badgers in 100 km²). The predicted patterns, however, are fairly sensitive to changes in the value of the transmission coefficient, β (see (19) to (21)). A very crude guide to its value in natural populations is obtained by taking the critical density for disease persistence (in a deterministic sense, as defined by K_T of the basic model) to be around one badger per square kilometre.

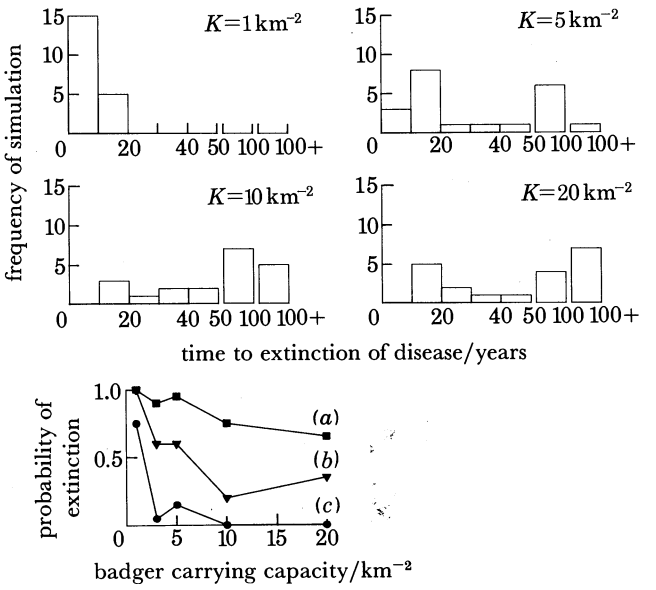

FIGURE 18. The stochastic model of bovine tuberculosis in badgers. Histogram of the frequency of stochastic fade out of the disease, and graph of the probability of disease extinction within (*a*) 100, (*b*) 30 and (*c*) 10 years of introduction into a population, for varying values of the carrying capacity. The simulation is for an area of 100 km². K_T (threshold density) = 1 km^{-2}. Standard population and disease dynamics parameters were used (see table 18). For each value of K_T 20 simulations were done with the same parameter values each time. Only the random numbers used in the Monte Carlo simulations varied in each of the 20 simulations.

The continual introduction of infectious animals from surrounding habitats (to simulate dispersal and migration, from and between social groups) results in much reduced probabilities of extinction. For example, as depicted in figure 19, the introduction of one infectious animal per year results in a marked change in the frequency distribution of time to extinction.

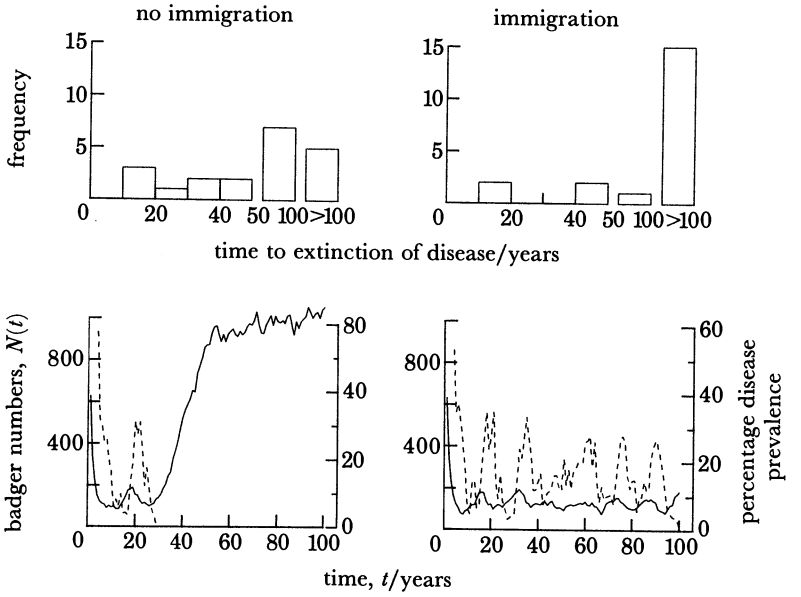

FIGURE 19. The affect of immigration on disease persistence in the stochastic model. The simulation is over an area of 100 km^2. K_T (threshold density) = 1 km^{-2}. Immigration rate of one infectious badger per year. Standard population and disease dynamics parameters were used (see table 18). Note how with immigration, the probability of disease extinction (top graphs) is less, as is graphically illustrated for one simulation of the model (bottom graphs) without (left-hand graph) and with (right hand graph) immigration.

The conclusion to emerge from these simulation studies is that bovine tuberculosis is likely to be able to persist in quite small populations of badgers, given a degree of dispersal and migration from and between social groups. This result is in marked contrast to similar studies of rabies transmission within fox populations (F. McAllister, personal communication) where the probability of extinction is extremely high as a consequence of disease pathogenicity and a low prevalence ('standing crop') of infectious (rabid) animals.

One further result to emerge from the simulation studies concerns the oscillatory fluctuations in badger abundance and disease prevalence. Stochasticity in demographic events tends to perpetuate oscillatory changes under circumstances in which the equivalent deterministic model predicts damped behaviour (Bartlett 1960). As displayed in figure 20, for bovine tuberculosis in badger populations the basic stochastic model generates quasi-oscillatory behaviour where the disease appears in a series of successive outbreaks. The magnitudes of these outbreaks do not tend to damp as the time period from introduction increases. The mean cycle length (estimated by time series analysis (see Box & Jenkins 1976)) of 20 simulation runs, each over 100 years, was approximately 15 years (this is similar to the period of the damped oscillations produced by the basic deterministic model with identical parameter values).

Age-structured models

As noted in the section on the dynamics of disease-free badger populations, age structure is an important determinant of population behaviour. A fully age-structured counterpart of the basic deterministic model is easily developed. It consists of a system of nonlinear partial

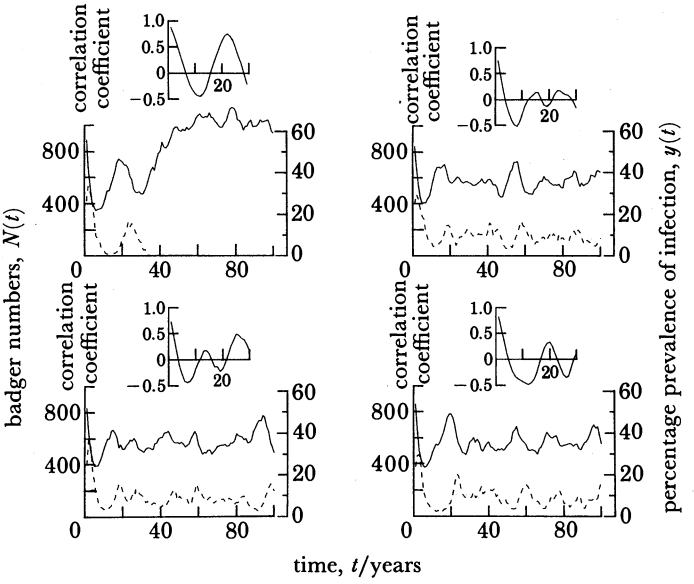

FIGURE 20. Simulations of the stochastic model showing the effect of chance events on model behaviour. The simulations are for an area of 100 km², with a badger-carrying capacity of 10 km^{-2}. K_T (threshold density) = 5 km^{-2}. Standard population and disease dynamics parameters were used (see table 18). ——, badger numbers; – – –, percentage disease prevalence. In all four simulations the same parameter values were used, but the Monte Carlo simulations used a different set of random numbers for each simulation run. The inset graphs for each run depict the serial correlation coefficients (time series analysis) for various yearly intervals. The vertical axes denote the correlation coefficient and the horizontal axes denote the time intervals in years.

differential equations denoting changes in the densities of susceptibles, incubators and infected both with respect to time (t) and age (a) ($X(a, t)$, $H(a, t)$ and $Y(a, t)$, respectively):

$$\frac{\partial X(a,t)}{\partial t}+\frac{\partial X(a,t)}{\partial a}=-\beta\left[\int_0^\infty Y(a,t)\,\mathrm{d}a+b(a)\right]X(a,t), \tag{40}$$

$$\frac{\partial H(a,t)}{\partial t}+\frac{\partial H(a,t)}{\partial a}=\beta\left[\int_0^\infty Y(a,t)\,\mathrm{d}a\right]X(a,t)-(b(a)+\sigma)H(a,t), \tag{41}$$

$$\frac{\partial Y(a,t)}{\partial t}+\frac{\partial Y(a,t)}{\partial a}=\sigma H(a,t)-(b(a)+\alpha)Y(a,t). \tag{42}$$

It is here assumed that the natural death rate of badgers, $b(a)$, is age-dependent and that β, α and σ are independent of age. The total population size, $\bar{N}(t)$, at time t is

$$\bar{N}(t)=\int_0^\infty [X(a,t)+H(a,t)+Y(a,t)]\,\mathrm{d}a \tag{43}$$

and the renewal (boundary) conditions may be expressed as

$$X(0,t)=\int_0^\infty \gamma(a,\bar{N})\,N(a,t)\,\mathrm{d}a \tag{44}$$

(where $\gamma(a, \bar{N})$ denotes the age-specific density-dependent birth rate) and

$$H(0,t)=Y(0,t)=0. \tag{45}$$

Analytical studies of (40) to (42) are made difficult by both the nonlinear transmission term and the boundary conditions (density-dependent fecundity). We use the model to examine only one aspect of transmission; namely, at equilibrium how does the prevalence of infection change with respect to age? In other words, we are interested in the age prevalence distribution within a badger population of constant size and stable age distribution (net births exactly balance net deaths). By setting the rates of change with respect to time equal to zero we obtain a set of linear differential equations,

$$dX/da = -(\lambda+b)\,X, \quad dH/da = \lambda X-(b+\sigma)\,H, \quad dY/da = \sigma H-(b+\alpha)\,Y, \quad (46)\text{–}(48)$$

where

$$\lambda = \beta\int_0^\infty Y(a)\,da = \beta\bar{Y} \tag{49}$$

and the death rate b is age-independent. The parameter λ is defined as the *per capita* 'force of infection' (the rate at which susceptible animals acquire the disease).

The solutions of (46) to (48) are

$$X(a) = X(0)e^{-(\lambda+b)\,a}, \tag{50}$$

$$H(a) = \frac{X(0)\lambda}{(\lambda-\sigma)}\left[e^{-(b+\sigma)a}-e^{-(\lambda+b)a}\right], \tag{51}$$

$$Y(a) = \frac{\sigma\lambda X(0)}{(\lambda-\sigma)}\left[\frac{e^{-(b+\sigma)a}}{(\alpha-\sigma)}-\frac{e^{-(\lambda+b)a}}{(\alpha-\lambda)}+\frac{e^{-(b+\alpha)a}}{(\alpha-\lambda)}-\frac{e^{-(b+\alpha)a}}{(\alpha-\sigma)}\right]. \tag{52}$$

The total numbers of susceptibles, incubators and infecteds are obtained by the respective integrals over all age classes (0 to ∞):

$$\bar{X} = X(0)/(\lambda+b), \quad \bar{H} = (\lambda X(0))/[(b+\sigma)\,(\lambda+b)], \quad \bar{Y} = (\sigma\lambda X(0))/[(b+\sigma)\,(\lambda+b)\,(b+\alpha)],$$

where total population size $\bar{N} = \bar{X}+\bar{H}+\bar{Y}$. The overall prevalence of infection in the population, $\bar{y}$, is

$$\bar{y} = (\bar{H}+\bar{Y})/\bar{N} = (b+\alpha+\sigma)/[[(b+\alpha)\,(b+\sigma)/\lambda]+(b+\alpha+\sigma)] \tag{53}$$

and the force of infection at equilibrium, λ, may be obtained by rearrangement of (53), where

$$\lambda = \frac{\bar{y}(b+\alpha)\,(b+\sigma)}{(1-\bar{y})\,(b+\alpha+\sigma)}. \tag{54}$$

The basic reproductive rate of infection (R_0), as defined in (23), is given by

$$R_0 = \left[1+\frac{(b+\alpha+\sigma)}{(b+\alpha)\,(b+\sigma)A}\right], \tag{55}$$

where A is the average waiting time from birth to infection (that is, the average age at infection, $1/\lambda$).

This equilibrium age-structured model is very basic and contains many simplifying assumptions. With these cautions in mind we now proceed to compare its predictions with the available data (this, as discussed earlier, is recorded in table 17). Given estimates of $\bar{y}$ and using the values of σ, α and b recorded in table 18 we estimate the values of λ to be 0.278 per year

for Gloucestershire in 1973–5 and 0.118 per year for the same area in 1977–9. By means of (50) to (52) we calculate the age-dependent prevalence of infection ($y(a)$) and these predictions are compared with the observed trends in figure 21. In spite of its simplicity, the model provides a reasonable overall description of the observed age prevalence graphs. The plateau in prevalence, as the animal's age, is a consequence of disease induced mortality. It acts to suppress the prevalence by the more rapid removal of infected animals when compared with susceptibles.

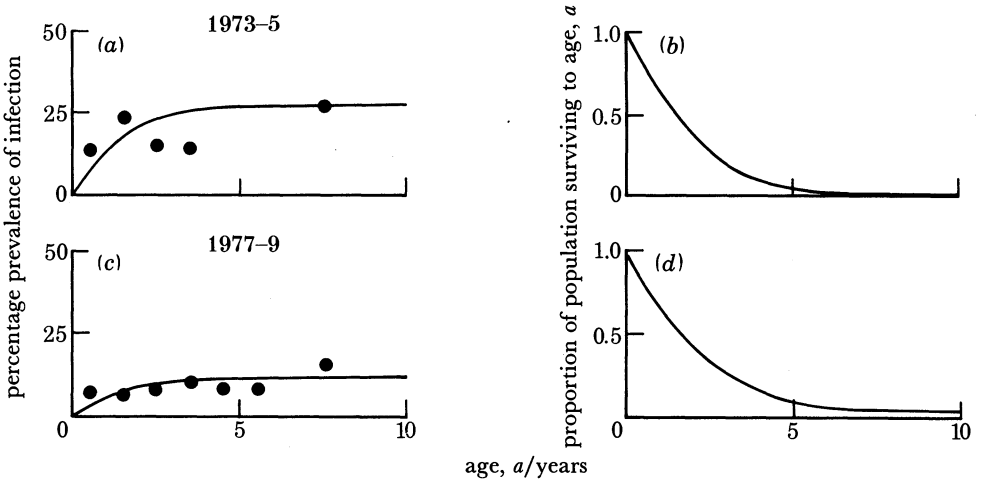

FIGURE 21. Changes in the prevalence of infection with badger age: observed trends and model predictions. The solid circles are observed values in (a) and (c) for the periods 1973–5 and 1977–9, respectively. The lines are the predictions of (50) to (52) in the main text. Parameter values: (a) $\lambda = 0.278$ per year; $\sigma = 4$ per year; $b = 0.4$ per year; $\alpha = 1.0$ per year; (c) the same as (a) but with $\lambda = 0.118$ per year. Graphs (b) and (d) denote the age-dependent survival curves of the badger populations (model predictions only).

The model's predictions, however, consistently underestimate the prevalence of infection in the young age classes of the population. We suspect (although we have no direct evidence) that the observed trend is a consequence of high transmission from parents to new born offspring in badger sets (pseudo-vertical transmission). An alternative explanation is that the value of the disease induced mortality rate, α, documented in table 18, is too low (and hence our estimate of λ is too small). Higher values of α (and hence λ) would mimic observed patterns more closely. We cautiously reject the latter explanation given current evidence of the comparatively good survival of infected badgers in natural populations (C. Cheeseman, personal communication). It is important to note, however, that such beliefs are based on limited data at present: little is known about the impact of bovine tuberculosis on the survival of cubs and juvenile badgers.

Summary of model predictions

Two major deficiencies restrict our analysis of disease dynamics. First, and most importantly, our qualitative understanding of the biology of the interaction between the badger and the bacterial infection is very limited (for example, the importance of pseudo-vertical transmission, the frequency of inactive infections, the pathology of the infection, etc.). Second, our quantitative knowledge of the values of the parameters that control disease transmission is equally sparse (for example, latent periods, disease-induced mortality rates, the force of infection, etc.). In these circumstances model construction and analysis has, in part, to be based

on a degree of guess work as opposed to certain knowledge. Our analyses of the different models reveal the following points.

(i) Deterministic models suggest that the interaction is stable and that oscillatory fluctuations will rapidly damp to the steady state (figure 13). Demographic stochasticity may pump otherwise damped oscillations into quasicycles with an average period of 10–15 years between peaks in disease prevalence and badger density (figure 20). The average amplitude of these cycles, however, is comparatively small and hence they may not be detectable in longitudinal sample data. Seasonality in transmission and cub production will generate seasonal fluctuations in badger density and disease prevalence but are unlikely to enhance the persistence of longer-term oscillations.

(ii) Models suggest that bovine tuberculosis acts to depress significantly badger density below disease-free levels, although the precise degree of depression depends critically on the magnitude of the rate of disease-induced mortality, α (figure 14). Very high and very low values of α result in little depression; intermediate values result in maximum depression (Anderson 1979). Our estimates of the parameters of the interaction (see table 18) suggest a higher degree of depression than is currently believed to be the case (on the basis of field observations).

(iii) The predicted levels of disease prevalence broadly match those observed. Pseudo-vertical transmission, the presence of 'carriers' and inactive cases all act to increase the standing crop of infected animals. Horizontal age prevalence data is adequately mimicked by simple age-structured models. Estimates of the force infection based on such models suggest that disease control programmes in the Gloucestershire area have reduced the net force of transmission between 1973 and 1979 by a factor of approximately 0.5. The sharp rise in prevalence during early life is thought to be due to pseudo-vertical transmission while the plateau in prevalence among older age classes of badgers appears to be a consequence of disease-induced mortality (figure 21).

(iv) The persistence of bovine tuberculosis in badger populations is enhanced by the probable involvement of pseudo-vertical transmission and the presence of carriers of infection. Both factors act to suppress the critical density of susceptible animals necessary for the maintenance of the infection within the host population. On this basis it is suggested that the infection will be able to persist in low density badger populations. The likelihood of disease extinction in such populations is reduced by small amounts of dispersal from and migration between social groups (figure 19). Model analysis suggests that the prevalence of infection will be related to badger density and, as such, to the incidence of infection in cattle herds (figure 10). A variety of other factors, however, will play an important role in the rate of transmission between badger populations and cattle herds (for example, cattle herd density, farming practices, habitat type, etc.).

5. Discussion and conclusions

Current knowledge of the ecology of the badger is limited, but probably sufficient to enable us to understand the principal processes controlling population abundance. This mammalian species has a low intrinsic growth ($r \approx 0.2$ per year), a not insignificant maturation delay to first breeding (with respect to life expectancy), produces relatively small litters of cubs, experiences high mortality in the first year of life but low thereafter, and exhibits limited migration and dispersal behaviour. Population abundance is largely determined by habitat

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type, and long term stability appears to be a consequence of density-dependent constraints on fecundity. Cyclic changes in density, with an average period of six to eight years, may arise in areas of moderate to poor habitat as a consequence of the constraints on fecundity and a lengthened delay to maturation. Oscillatory fluctuations will be most marked with respect to cub density, as opposed to adult numbers. Population growth, following periods of drought, suggests that the regulatory constraints on fecundity only come in to play as density approaches the carrying capacity of the habitat. There is some evidence from removal experiments of a lower threshold in density, below which successful breeding is inhibited as a consequence of social and behavioural factors. A graphical summary of these various population processes, as they influence the relation between net population growth and population density, is presented in figure 22.

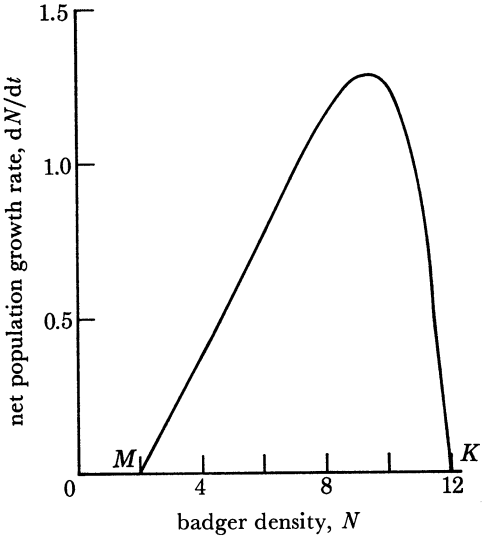

FIGURE 22. Diagrammatic representation of the relationship of the net badger population growth rate (dN/dt) as a function of population density, N. The density K denotes the carrying capacity of the habitat while the density M, denotes a lower threshold density below which social and behavioural factors prohibit successful breeding.

Past and current programmes for the control of bovine tuberculosis in England in areas of frequent herd infection have centred on the reduction of badger density by the removal of entire social groups of animals where one or more individuals were thought to be infected. The close proximity of cattle pasture to good badger habitat is invariably a characteristic of such areas. Trapping is currently the primary means of badger control although gassing of sets has been used in the past. Our analysis of the dynamics of badger populations provides some guidelines concerning the likely impact of such measures. On the one hand, the low intrinsic growth rate of the badger (with a characteristic return time of roughly five years), its limited powers of dispersal and the suggestion of a critical group size for successful cub production (see figure 22), argue that intensive trapping once every three or four years will effectively suppress badger density to low levels. Much greater effort is necessary to produce similar results for a species such as the red fox (*Vulpes vulpes*) which has an intrinsic growth rate more than twice the magnitude of that of the badger and greater dispersal and colonization abilities. On the other hand, however, the nature of the regulatory constraints on growth (mammal type density-

dependence) imply that moderate or limited trapping effort (small or medium reduction in density) will move the population into areas of high potential growth (see figure 22). In such areas of density suppression, the rate of return of population abundance to its precontrol level is likely to be relatively rapid (one, two or three years depending on the degree of suppression). Combined with this factor is the contentious point that trapping must be continual (since population abundance is a renewable resource) unless badgers are exterminated from large areas and recolonization from surrounding regions prevented. This view is based on our understanding of the population dynamics of the badger and the pathogen. It differs somewhat from the implicit assumption in the current M.A.F.F. control policy that a single and intensive intervention (in the area of a recent cattle herd breakdown) to remove all infected groups of animals will suffice to eliminate infection from that area for long periods of time.

Our review and analysis of the epidemiology of bovine tuberculosis in badger populations has been severely restricted by the lack of detailed information. On current evidence, however, it appears likely that the disease is endemic in many badger populations throughout England. The association between set density and the incidence of infection in cattle herds (see figure 10) argues that badgers play an important role in disease transmission to cattle. The foraging activities of badgers on cattle pasture, their preferred food item (the earthworm), the passage of large numbers of bacilli in urine, faeces and sputum of infected animals, the long duration of infectiousness of diseased animals, the survival of bacilli at the soil surface and the high frequency of urination (as a direct consequence of their feeding habits) combine to support this view.

Our analyses of transmission dynamics further support this view, suggesting that the infection is able to persist in a stable manner within badger populations at moderate levels of prevalence (10–20%). Average prevalence (over all age classes) is likely to be positively correlated with badger density (figure 12) although the relationship is nonlinear. Factors such as the frequent transmission of the infection from parent to new born offspring, and the presence of carriers of infection (infected animals with few overt signs of disease), imply that the disease will be able to persist endemically in low density badger populations. As such, we suspect that the infection is widespread in Britain in areas of good, medium and perhaps poor badger habitat (more widespread than is suggested by current survey work which has tended to focus on areas of medium to high badger density). This observation, combined with the reported association between the incidence of infection in cattle, and badger set density, argue that other factors in addition to the presence of infected badger populations play a role in cattle herd breakdowns. For example, a high prevalence of infected badgers (implying high badger density), high cattle herd density in pastures intimately connected with good badger habitat and certain practices in farm management (for example, allowing badger access to cattle sheds, salt licks and water troughs) are all likely to be important.

Reduction of host density is predicted to reduce the prevalence of infection in badger populations, but in a nonlinear manner (figure 12). Most importantly, given components of horizontal and pseudo-vertical transmission, plus the involvement of carriers, we predict that very substantial reductions in badger abundance are necessary to induce marked changes in prevalence (within badgers). Some support for this prediction is provided in figure 21; the net force of infection was reduced by just over 50% in an area of intensive control activity (Gloucestershire) between 1973 and 1979 (see also figure 7). It should be noted, however, that this moderate reduction in prevalence in badger populations was associated with a reduction

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in the incidence of cattle herd infections in this area over the same period. Also note that our analyses suggest that this interpretation is unlikely to be confounded by marked cyclic changes in disease prevalence and host abundance arising from the dynamic interaction between host and pathogen populations. These observations tentatively suggest that it may not be necessary to eradicate the infection in badger populations to reduce greatly the frequency of herd breakdowns. Infected badgers, however, even at low densities, will always pose a long term risk in cattle farming regions where badger habitat is intimately associated with cattle pasture. This creates a dilemma. Should the aim of control be the eradication of bovine tuberculosis in badger populations or the elimination of herd breakdowns? The former objective requires intensive effort, sustained in perpetuity, to reduce badger densities to, and maintain them at, very low levels. The latter objective seems to require more moderate reductions in badger density (although control effort must still be maintained in perpetuity), but carries with it the associated long term risk (however small) of herd breakdowns as a simple consequence of the persistence of low levels of infection in the suppressed badger populations. The endemicity of bovine tuberculosis in many badger populations, the widespread occurrence of the reservoir host in Britain and the intense plus continual effort required to control badger densities, all combine to suggest that alternative (or additional) methods of control should be actively sought. Plausible options are few at present but include vaccination of cattle and the development of farm management practices to reduce badger to cattle transmission and pasture contamination. The latter option appears attractive at first sight but is beset by many practical problems in regions where badger habitats are so intimately intermeshed with intensively used cattle pastures (that is, regions of the southwest of England). One controversial option is actively to discourage cattle farming in areas with a history of cattle herd infection and moderate to high badger densities. This approach is clearly unattractive to the cattle farming industry but there may be a case for exploring the implications and consequences of placing the responsibility (and hence cost) of bovine tuberculosis control, and the maintenance of disease-free cattle herds, on the farmer as opposed to the government (M.A.F.F.) (with, at present, the taxpayer ultimately carrying the burden of the cost of control and the compensation paid to farmers where cattle have had to be destroyed to control disease spread). Problems could arise from such an approach, not least of which would be the need to relax current regulations concerning the culling of badger populations by non-Ministry personnel. With respect to the conservation of badger populations in Britain, the relaxation of current restrictions could well be disastrous unless licences (or some such arrangement) to kill badgers were issued only in areas with an established history of cattle herd infection.

Vaccination is also, at first sight, an appealing long term solution, given the current availability of vaccines that have been used with a degree of success to control tuberculosis in cattle herds within certain regions of Africa and India. Again, problems surround this approach. First, current vaccines have a variable efficiency and vaccination of an entire herd would not in general imply a 100% degree of herd immunity to infection. The more immediate problem, however, is that connected with the movement of cattle within Britain and their export to European countries. Current regulations require tuberculosis tests to verify the disease-free status of cattle before movement within Britain, or export to other European Economic Community (E.E.C.) countries. At present, there is no satisfactory method for discriminating between a positive tuberculin test result arising from effective immunization, and a similar result arising from active infection. The creation of cattle herd immunity to tuberculosis in regions

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of Britain with infected badger populations would therefore prohibit the sale and movement of immunized animals within Britain and their export to E.E.C. countries (under current E.E.C. regulations). There is perhaps a case for mounting pressure to change current regulations concerning tuberculin tests and cattle export and movement, in situations where documentary evidence of vaccination is presented. Who, however, should bear the cost of immunization: the farmer or the government?

Vaccination of badgers has also been suggested but this again would be difficult to put into practice. First, there is the need for continual maintenance of high levels of herd immunity given the introduction (by births) of new cohorts of susceptibles each year. More importantly, the likely significance of pseudo-vertical transmission in disease maintenance within badger populations, implies that many animals would acquire the infection before leaving the set and become available for vaccination (either by trapping and inoculation or by oral vaccination via bait consumption). In any case, such considerations are entirely speculative at present since it is not known whether or not the currently available vaccines will effectively immunize badgers against tuberculosis infection.

It should be clear from the preceding discussion why badger culling (in particular the removal of infected social groups of animals) is currently viewed as the only practical short term solution to disease control. The difficulties surrounding bovine tuberculosis eradication are frequently underestimated by those who are concerned with the conservation of badger populations in Britain. In the long term, however, the recurrent cost of intensive badger control argues for a reappraisal of the practicalities and costs of alternative methods. In such considerations a detailed analysis of the costs (the cost of current control methods plus the compensation paid to farmers where herds have been destroyed to control the spread of infection) and benefits (the frequency of herd breakdowns today compared with the frequency before control) would be of great assistance.

To conclude we turn to the question of future research needs. It is clear, from the preceding sections of this paper, that our current knowledge of the population biology of badgers and the transmission dynamics of bovine tuberculosis is somewhat limited. In general, our understanding of badger biology and ecology is better than that of disease biology and epidemiology. We see four major needs with respect to badgers. First, experimental studies of natural populations are required involving the removal of animals and the monitoring of subsequent population growth. Data of this type would provide confirmation or refutation of our assumption that badger fecundity is density-dependent. Our estimates were based on two natural population perturbations induced by climatic change (for example, drought) (figure 1). Experimental studies could be designed to investigate population growth in areas with different habitat carrying capacities, following various degrees of population suppression. The trapping control programme itself provides an opportunity to carry out such work, given sufficient investment of resources and effort pre- and post-removal. Such studies should focus on gross population abundance, the reproductive performance of individual animals and cub survival. The second need, which is linked to the first, concerns long-term monitoring of changes in adult badger and cub abundance in areas of good, medium and poor badger habitat. The third aspect relates to the dispersal and migration behaviour of animals. Little is known at present concerning the frequency of movement of animals between social groups, in either disturbed or undisturbed habitats. The fourth need is for an accurate method for ageing live and dead badgers.

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With respect to the epidemiology of bovine tuberculosis in badgers, the needs are many and varied. First, and most importantly, further studies of experimentally infected animals are required to improve our basic biological understanding of the interaction between host and pathogen. Such experiments should be designed to provide information on, for example, the latent period of infection, the life expectancy of infectious animals, the frequency and duration of excretion of infective bacilli, the carrier and inactive states of infection and the general pathology of the infection in badgers. Second, a serological test is required to detect infection in live badgers and avert the current necessity of firm diagnosis *post mortem*. Current immunological techniques, involving the production of polyclonal antibodies, are, in principle, able to yield a reliable and specific diagnostic test for experimental and survey research. The third area concerns the long term monitoring of longitudinal and horizontal changes in disease prevalence. Ideally, such studies should focus on areas with different average badger densities to enable some sort of quantitative picture to be constructed of the relation between prevalence and host density. The trapping control programme potentially provides a large source of material for such research (between 500 and 1000 badgers per year), provided data on badger density and disease prevalence are collected concomitantly. Horizontal data on age prevalence provides the means of estimating the net force of transmission within defined habitats, but its collection is of course dependent on the availability of an accurate technique for ageing badgers. Incidentally, the source of material from the trapping programme could also provide valuable information on the reproductive status of populations in relation to overall population abundance. Survey studies could also profitably focus on the prevalence of infection in areas of Britain not afflicted by cattle herd infection and on spatial heterogeneity in the distribution of infected social groups of animals. There is also a need for further research on disease transmission, both within and between social groups. The relative importance of horizontal transmission through host-to-host contact and host-infective stage contact (bacilli-free in the habitat of the badger), and pseudo-vertical transmission, to the overall endemic maintenance of infection is unclear at present. Behavioural studies on naturally infected populations could provide valuable information on not only transmission but also the survival of infected badgers and the age at which infection is typically acquired. Some research has been initiated in certain of the areas discussed above (see Cheeseman *et al.* 1981, 1985; Wilesmith 1983; Little 1982*a*, *b*) but, inevitably, much of the work is long term in character and hence our knowledge of these topics remains limited at present. Finally, we stress the preliminary nature of our own study. The summaries of collected data and associated analyses merely serve as a starting point for future research once more data becomes available.

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PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

References

Abelencev, V. I. 1966 Do ekologii ta gospodarskogo znacennja borsuka na Ukraini. *Ekol. Istor. Chrebet. Fauni Ukrai, Kiiv* pp. 73–79.

Ahnlund, H. 1976 Age determination in the European badger (*Meles meles* L.). *Z. Saug.* **41**, 119–125.

Ahnlund, H. 1980 Sexual maturity and breeding season of the badger, *Meles meles*, in Sweden. *J. Zool., Lond.* **190**, 77–95.

Allen, K. R. 1963 Analysis of stock-recruitment relations in Antartic fin whales. *Cons. int. Explor. Mer-Rapp. Proc. Verb.* **164**, 132–137.

Anderson, R. M. 1979 Parasite pathogenicity and the depression of host population equilibrium. *Nature, Lond.* **279**, 150–152.

Anderson, R. M. (ed.) 1982 *Population dynamics of infectious diseases: theory and applications*. London: Chapman and Hall.

Anderson, R. M., Jackson, H., May, R. M. & Smith, A. D. M. 1981 Population dynamics of fox rabies in Europe. *Nature, Lond.* **289**, 765–771.

Anderson, R. M. & May, R. M. 1979 Population biology of infectious diseases, I. *Nature, Lond.* **280**, 361–367.

Anderson, R. M. & May, R. M. 1981 The population dynamics of microparasites and their invertebrate hosts. *Phil. Trans. R. Soc. Lond.* B **291**, 451–524.

Anderson, R. M. & May, R. M. 1982 Directly transmitted infectious diseases: control by vaccination. *Science, Wash.* **215**, 1053–1060.

Anderson, R. M. & May, R. M. 1983 Vaccination against rubella and measles: quantitative investigations of different policies. *J. Hyg., Camb.* **90**, 259–325.

Bailey, N. T. J. 1975 *The mathematical theory of infectious diseases and its applications*. London: Griffin.

Bartlett, M. S. 1960 The critical community size for measles in the United States. *J. R. stat. Soc.* B **123**, 37–44.

Beddington, J. R. 1978 On the dynamics of Sei whales under exploitation. *Rep. Int. Whal. Commn* **28**, 169–172.

Box, G. E. P. & Jenkins, G. M. 1976 *Time series analysis: forecasting and control*. San Francisco: Holden-Day.

Brøgger, S. 1967 Systems analysis in tuberculosis control: a model. *Am. Rev. resp. Dis.* **95**, 421–434.

Canivenc, R. 1966 A study of progesterone in the European badger (*Meles meles*). *Symp. zool. Soc. Lond.* **15**, 15–26.

Caughley, G. 1967 Calculation of population mortality rate and life expectancy for thar and kangaroos from the ratios of juveniles to adults. *NZ. Jl Sci.* **10**, 578–584.

Caughley, G. 1977 *Analysis of vertebrate populations*. London: John Wiley.

Caughley, G. & Birch, L. C. 1971 Rate of increase. *J. Wldl. Manag.* **35**, no. 4.

Cheeseman, C. L., Jones, G. W., Gallagher, J. & Mallinson, P. J. 1981 The population structure, density and prevalence of tuberculosis (*Mycobacterium bovis*) in badgers (*Meles meles*) from four areas in South West England. *J. appl. Ecol.* **18**, 795–804.

Cheeseman, C. L., Little, T. W. A., Mallinson, P. J., Rees, W. A. & Wilesmith, J. W. 1985 Progression of bovine tuberculosis (*Mycobacterium bovis*) in a European badger (*Meles meles*) population in South West England; preliminary findings. *Acta. Zool. Fennica* (In the press.)

Cheeseman, C. L. & Mallinson, P. J. 1979 Radio tracking in the study of bovine tuberculosis in badgers. In *A handbook on biotelemetry and radio tracking* (ed. C. J. Almaner & D. W. MacDonald), pp. 646–656. Oxford: Pergamon Press.

Cheeseman, C. L., Mallinson, P. J., Page, R. J. C. & Wilesmith, J. W. 1985 Population ecology and prevalence of tuberculosis in badgers in an area of Staffordshire. *Mam. Rev.* (In the press.)

Clark, C. W. 1976 A delayed-recruitment model of population dynamics, with an application to baleen whale populations. *J. math. Biol.* **3**, 381–391.

Cvjetanovic, B., Grab, B. & Uemura, K. 1978 Dynamics of acute bacterial diseases. *Bull. Wld Hlth Org.* **56** (suppl. 1), 1–143.

Dietz, K. 1976 The incidence of infectious diseases under the influence of seasonal fluctuations. *Lect. Notes Biomath.* **11**, 1–15.

Feldstein, M. S., Piot, M. A. & Sundoreson, T. K. 1973 Resource allocation model for public health planning. *Bull. Wld Hlth. Org.* **48**, 3–108.

Fenchel, T. 1973 Intrinsic rate of natural increase: the relationship with body size. *Oecologia* **14**, 317–326.

Fischer, E. 1931 Early stages in the embryology of the badger. *Verh. Anat. Ges. Jena* **40**, 22–34.

Fisher, M. E. & Goh, B. S. 1984 Stability results for delayed-recruitment models in population dynamics. *J. math. Biol.* **19**, 147–156.

Fowler, C. W. 1981 Density dependence as related to life history strategy. *Ecology* **62**, 602–610.

Fowler, C. W. & Smith, T. D. (eds) 1981 *Dynamics of large mammal populations*. Brisbane: John Wiley.

Gallagher, J. 1979 Social and ecological affects on the pattern of tuberculosis in the badger. Ph.D. thesis, University of London.

Gallagher, J. & Nelson, H. 1979 Causes of ill health and natural death in badgers in Gloucestershire. *Vet. Rec.* **105**, 546–551.

Graf, M. & Wandeler, A. I. 1982*a* The reproductive cycle of male badgers (*Meles meles L.*) in Switzerland. *Rev. Suis. Zool.* **89**, 1005–1008.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Hancox, M. K. 1980*a* Studies in the ecology of the Eurasian badger (*Meles meles*). D.Phil. thesis, University of Oxford.
Hancox, M. K. 1980*b* Parasites and infectious diseases of the Eurasian badger: a review. *Mamml Rev.* **10**, no. 4, 151–162.
Kruuk, H. 1978 Spatial organisation and territorial behaviour of the European badger (*Meles meles*). *J. Zool., Lond.* **184**, 1–19.
Kruuk, H. & Parish, T. 1982 Factors affecting population density, group size and territory size of the European badger (*Meles meles L.*). *J. Zool., Lond.* **196**, 31–39.
Leslie, P. H. 1945 On the use of matrices in certain population mathematics. *Biometrika* **33**, 183–212.
Levin, S. A. & May, R. M. 1976 A note on difference–delay equations. *Theor. Pop. Biol.* **9**, 178–187.
Likhagen, G. N. 1956 Some ecological traits of the badger of the Tuba Abatis broadleaf forest. In *Studies on mammals in government preserves* (ed. Yurgenson), Moscow: Ministry of Agriculture of the U.S.S.R.
Little, T. W. A., Naylor, P. F. & Wilesmith, J. W. 1982 Laboratory studies of *Mycobacterium bovis* infection in badgers and cattle. *Vet. Rec.* **111**, 550–557.
Little, T. W. A., Swan, C., Thompson, H. V. & Wilesmith, J. W. 1982 *Mycobacterium bovis* infection in cattle, badgers and other mammals in an area of Dorset. *Vet. Rec.* **110**, 318–320.
Lynn, W. R. & Revelle, C. S. 1968 Workshop on model methodology for health planning, with particular reference to tuberculosis. *Am. Rev. Resp. Dis.* **98**, 687–691.
Macdonald, D. W. 1980 *Rabies and wildlife: a biologist's perspective.* Oxford: Oxford University Press.
Maddock, E. C. G. 1933 Studies on the survival time of the bovine tubercle bacilli in soil, soil and dung, in dung and on grass. *J. Hyg. Camb.* **33**, 103–117.
M.A.F.F. 1973 *Triennial report of the Pest Infestation Control Laboratories.* p. 178. London: H.M.S.O.
M.A.F.F. 1976 *Bovine tuberculosis in badgers.* Report by the Ministry of Agriculture, Fisheries and Food, London.
M.A.F.F. 1977 *Bovine tuberculosis, in badgers.* Report by the Ministry of Agriculture, Fisheries and Food, London.
M.A.F.F. 1978 *Annual report on research and technical work 1978.* Department of Agriculture for Northern Ireland, p. 170. Belfast: H.M.S.O.
M.A.F.F. 1979 *Bovine tuberculosis in badgers.* Third report by the Ministry of Agriculture, Fisheries and Food, London.
M.A.F.F. 1980 *Bovine tuberculosis in badgers.* Fourth report by the Ministry of Agriculture, Fisheries and Food, London.
M.A.F.F. 1981 *Bovine tuberculosis in badgers.* Fifth report by the Ministry of Agriculture, Fisheries and Food, London.
M.A.F.F. 1982 *Bovine tuberculosis in badgers.* Sixth report by the Ministry of Agriculture, Fisheries and Food, London.
M.A.F.F. 1983 *Bovine tuberculosis in badgers.* Seventh report by the Ministry of Agriculture, Fisheries and Food, London.
Mahler, H. T. & Piot, M. A. 1966*a* Essais d'application de la recherche operationnelle dans la lutte antituberculeuse. I. Formulation des problèmes, rassemblement des données, choix de modèles. *Bull. INSERM* **21**, 855–881.
Mahler, H. T. & Piot, M. A. 1966*b* Essais d'application de la recherche operationnelles dans la lutte anti-tuberculeuse. II. Programmation lineaire: problèmes conceptuels et d'application. *Bull. INSERM* **21**, 1021–1045.
May, R. M. 1973 *Stability and complexity in model ecosystems.* Princeton: Princeton University Press.
May, R. M. 1974 Biological populations with non-overlapping generations: stable points, stable cycles and chaos. *Science, Wash.* **186**, 645–647.
May, R. M. 1981 Models for single populations. In *Theoretical ecology* (ed. R. M. May), pp. 5–29. Oxford: Blackwells.
May, R. M. & Anderson, R. M. 1979 Population biology of infectious diseases. II. *Nature, Lond.* **280**, 455–461.
May, R. M. & Oster, G. F. 1976 Bifurcations and dynamic complexity in simple ecological models. *Am. Nat.* **110**, 573–599.
Mouches, A. 1982 Le blaireau European (*Meles meles*) biologie et eco-ethologie. *Bull. Men. O.N.C.* 21–28.
Muirhead, R. H., Gallagher, J. & Burn, K. J. 1974 Tuberculosis in wild badgers in Gloucestershire: epidemiology. *Vet. Rec.* **95**, 552–555.
Neal, E. G. 1972 The national badger survey. *Mamml Rev.* **2**, 55–64.
Neal, E. G. 1977 *Badgers.* Poole, Dorset: Blandford Press.
Neal, E. G. & Harrison, R. J. 1958 Reproduction in the Europena badger (*Meles meles*). *Trans. zool. Soc. Lond.* **29**, 67–131.
Nisbet, R. M. & Gurney, W. S. C. 1982 *Modelling fluctuating populations.* London: John Wiley.
Notini, G. 1948 Biologiska Undersokningar over Gravlingen (*Meles meles*). *Svenska Jagareforbundel Meddelande* **13**, 1–126.
Pearl, R. & Reed, L. J. 1920 On the rate of growth of the population of the United States since 1790 and its mathematical representation. *Proc. natn. Acad. Sci. U.S.A.* **6**, 275–288.
Pelickan, J. & Vackar, J. 1978 Densities and fluctuations in numbers of red fox, badgers and pine marten in the Bucin forest. *Fol. Zool.* **27**, 289–303.
Pianka, E. R. 1970 On *r*- and *K*- selection. *Am. Nat.* **104**, 592–597.
Pianka, E. R. 1972 *r*- and *K*- selection or *b* and *d* selection? *Am. Nat.* **106**, 581–588.
Pielou, E. C. 1969 *An introduction to mathematical ecology.* New York: Wiley-Interscience.
Pollard, J. H. 1973 *Mathematical models for the growth of human populations.* Cambridge: University Press.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Rapaport, P. 1979 Contribution à l'etude du chat sauvage et des mammifères carnivores en Lorraine. *Rapport Ingenieur E.N.S.A., Nancy.*
Revelle, C., Lynn, W. R. & Feldman, F. 1967 Mathematical models for the economic allocation of tuberculosis control activities in developing countries. *Am. Rev. resp. Dis.* **96**, 893–909.
Revelle, C. & Male, J. 1970 A mathematical model for determining case finding and treatment activities in tuberculosis control programs. *Am. Rev. resp. Dis.* **102**, 403–411.
R.S.P.C.A. 1979 *Badgers and bovine tuberculosis: the case for further investigation.* Special report. Causeway, Horsham, West Sussex: R.S.P.C.A.
Southwood, T. R. E. 1981 Bionomic strategies and population parameters. In *Theoretical ecology* (ed. R. M. May) 2nd edn, pp. 30–52. Oxford: Blackwells
Stirling, E. A. & Harper, R. J. 1969 The distribution and habits of badgers on the southern outskirts of Durham City. *Bull. Mamml Soc.* **32**, 5–6.
Stubbe, M. 1965 Zur Biologie der Raubtiere einer abgeschlossenen Waldgebietes. *Z. Jagd.* **11**, 73–102.
Stubbe, M. 1970 Population biology of the badger (*Meles meles*). *Transactions of the IXth International Congress of Game Biologists*, Moscow.
Stubbe, M. 1973 Schutz und Hege des Dachses. In *Buch der Hege* (ed. M. Stubbe), Bd 1, pp. 227–249. Berlin: VEB Deutscher Landwirtschafts verlag.
Waaler, H. T. 1968*a* Cost-benefit analysis of BCG-vaccination under various epidemiological situations. *Bull. int. Un. Tuber.* **41**, 42–52.
Waaler, H. T. 1968*b* A dynamic model for the epidemiology of tuberculosis. *Am. Rev. resp. Dis.* **98**, 591–600.
Waaler, H. T., Geser, A. & Anderson, S. 1962 The use of mathematical models in the study of the epidemiology of tuberculosis. *Am. Jl Publ. Hlth* **52**, 1002–1013.
Waaler, H. T. & Piot, M. A. 1969 The use of an epidemiological model for estimating the effectiveness of tuberculosis control measures: sensitivity of the effectiveness of tuberculosis control measures to the coverage of the population. *Bull. Wld Hlth Org.* **41**, 75–93.
Wachendörfer, G. & Schwierz, G. 1980 Epidemiology and control of wildlife rabies – analysis of potential causes for the vigorous reduction of the badger (*Meles meles*) population in Hesse, 1952 till 1957. *Deut. Tier. Woch.* **87**, 255–260.
Wandeler, A. I. & Graf, M. 1982 The reproductive cycle of female badgers (*Meles meles*) in Switzerland. *Rev. Suis. Zool.* **89**, 1009–1016.
Wijngaarden, A. van & Peppel, J. van de 1964 The badger (*Meles meles*) in the Netherlands. *Lutra* **6**, 1–60.
Wilesmith, J. W. 1983 Epidemiological features of bovine tuberculosis in cattle herds in Great Britain. *J. Hyg., Camb.* **90**, 159–176.
Williams, R., Stenhouse, K. & Hoy, W. A. 1930 The viability of *B. tuberculosis* (*Bovinis*) on pastureland, in stored faeces and liquid manure. *J. Hyg., Camb.* **30**, 413–419.
Zuckerman, O. M. 1980 *Badgers, cattle and tuberculosis.* London: H.M.S.O.
Zuckerman, O. M. 1981 The great badger debate. *Nature, Lond.* **289**, 628–630.

Appendix 1

Local stability of the age-structured difference equation model

(Equation (9) in the main text)

Equation (10) can be written as a delay-difference equation of the form

$$N_2(t+1) = SN_2(t) + (1-S)g\,(N_2(t-k)), \tag{A 1}$$

where $S = (1-\mu_2)$ and

$$g(N_2(t-k)) = \frac{[(1-\mu_{k-1})\,(1-\mu_{k-2})\ldots(1-\mu_0)\,R(N_2(t-k))\,N_2(t-k)]}{[1-\mu_2]}.$$

If we linearize (A 1) about the equilibrium, N^* (see (12) in the main text) then a sufficient condition for N^* to be asymptotically stable is that all roots of the characteristic equation

$$\lambda^{k+1} - S\lambda^k - (1-S)g'\,(N^*) = 0 \tag{A 2}$$

have modulus less than one. The roots of (A 2) satisfy $|\lambda| < 1$ if $\delta_k(S) < g'\,(N^*) < 1$ where the function $\delta_k(S)$ depends on the length of the time delay, k, and must always satisfy $\delta_k(S) \leqslant -1$

(Fisher & Goh 1984). For $k = 1$, we obtain a quadratic equation (A 2) and $\delta_1(S) = -1/(1-S)$. For parameter values relevant to the dynamics of badger populations (see table 10) the system is stable. For a maturation delay of two or three years ($k = 2$ or $k = 3$), however, numerical solution of (A 2) with the parameter values listed in table 10 and N^* set at 10.0 km^{-2} reveals that the model is locally unstable (see Levin & May (1976) and Clark (1976) for further discussion of the evaluation of (A 2) with $k > 1$).

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