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Bovine Tuberculosis in Badger (Meles meles) Populations in Southwest England: An Assessment of Past, Present and Possible Future Control Strategies Using Simulation Modelling

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Bovine tuberculosis in badger (*Meles meles*) populations in southwest England: an assessment of past, present and possible future control strategies using simulation modelling

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CONTENTS

SUMMARY

A spatial stochastic simulation model was used to compare the efficacy of different badger control policies and to determine the theoretical requirements for the control of endemic bovine tuberculosis in badger populations in southwest England. Culling-based strategies for controlling endemic disease were compared with strategies employing a yet-to-be-developed oral vaccine which would provide uninfected badgers with immunity to the infection. A comparative assessment was made of the efficacy of previous and proposed culling-based strategies employed by the Ministry of Agriculture, Fisheries and Food for the control of localized disease, and the potential for an oral vaccine-based strategy for the control of localized disease was examined.

For endemic bovine tuberculosis, to achieve a reasonable probability ($p > 0.70$) of successful control with a strategy involving a single culling operation, a very high proportion of the badger population ($> 90\%$) must be culled. Single vaccination would not be successful in combating endemic disease. However, strategies involving repeated annual vaccination would have a very high probability of eradicating endemic disease, even with a relatively low (40–50%) annual vaccination efficiency.

The most successful culling-based strategies for the control of localized disease were the gassing and clean ring strategies. Compared with no control at all, the interim strategy only offered benefits of a lower probability of disease spread and persistence in populations with low disease-free equilibrium group sizes or low initial prevalences of infection. In all other instances the benefits were negligible. The live test strategy will offer an improvement over the interim strategy, but will not be as effective as either the gassing or clean-ring strategies. In addition, it is likely to necessitate the culling of approximately four times as many badgers each year as the interim strategy, and the proportion of those killed that are infected will be approximately half that under the interim strategy. The efficacy of a strategy involving annually repeated oral vaccination of the badgers within a similar area to that covered by the live test depended on the efficiency of vaccination. A vaccination efficiency of 20–60% represented an overall

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improvement in efficacy over the interim strategy, being equivalent to the live test strategy. However, only vaccination efficiencies of 60–80 % or greater achieved similar results to the gassing strategy, and none were so successful as the clean-ring strategy.

Recommendations for future management are provided. Reactive strategies based on culling or vaccination will not solve the problem of bovine tuberculosis in badgers. Proactive strategies directed in those areas with a recent history of bovine tuberculosis in badgers should be considered as an alternative short-term control measure. The only strategy likely to eradicate bovine tuberculosis from badger populations in the long term is the use of repeated vaccination in proactive control operations in areas with a history of bovine tuberculosis in the badger population. Analyses should be conducted to evaluate whether the economic benefits of the live-test strategy are likely to outweigh its economic and ecological costs and whether continued research into the development of a vaccine for badgers is likely to offer any significant long-term economic benefits.

1. INTRODUCTION

It was discovered that badgers (*Meles meles*) were a potential wildlife reservoir for *Mycobacterium bovis* in the early 1970s; since then a high proportion of the cases of bovine tuberculosis in cattle in southwest England have been attributed to a badger origin (Zuckerman 1980). Since 1975, the Ministry of Agriculture, Fisheries and Food (MAFF) have undertaken badger control operations in those areas where badgers have been considered the most likely source of bovine tuberculosis in cattle.

Various control strategies have been employed by MAFF over this time period. From August 1975 until June 1982, badgers were gassed by pumping hydrogen cyanide powder into setts (Cheeseman *et al.* 1981; Little *et al.* 1982), the 'gassing' strategy. After each gassing operation there was a twelve-month monitoring period during which the gassed setts were checked for any signs of badger activity. If any activity was detected around a sett during this time, the sett was re-gassed (E. Krolick, personal communication).

From August 1982, cage trapping replaced gassing, which was then considered inhumane. Trapping was concentrated around the setts, and badger groups were removed in a centrifugal manner until a 'clean ring' of uninfected social groups had been removed. This is referred to as the 'clean ring' strategy throughout this paper. All trapped badgers were shot except lactating females, which were released. Once again, there was a monitoring period during which the controlled setts were checked for signs of badger activity and, if any occurred, further trapping was undertaken.

From April 1986, after the recommendations of the second government review on badgers and bovine tuberculosis (Dunnet *et al.* 1986), an 'interim' strategy was introduced: it remained in force until October 1994. In this strategy, trapping operations were confined to that part of a farm where cattle were believed to have contracted the disease from badgers, or to the whole farm if it was not possible to be more precise (MAFF 1987). All badgers caught, except lactating sows, were killed and tested for *M. bovis* infection *post mortem*.

In November 1994, a new strategy, initially announced in December 1993 (MAFF 1993*a*), was introduced. Under this strategy, control operations are conducted as before under the interim strategy in those areas of southwest England without a history of bovine tuberculosis in cattle. However, where there is a history of bovine tuberculosis in cattle thought to have originated from badgers, three alternative approaches are employed. In the first, no control operations are conducted where the farmer chooses not to have control operations on his farm, unless there is a subsequent outbreak of bovine tuberculosis in the cattle. In the second approach, the interim strategy is conducted as before. In the third approach, the original intention was that the interim strategy would be conducted on the farm where the tuberculosis outbreak occurred, but in addition, a blood test for diagnosing TB in live badgers – the 'live test' strategy (Nolan & Goodger 1993) – would be used on a sett-by-sett basis on the surrounding land (approximately 10 km² in total area), to see whether any badgers around the original farm are also infected with bovine tuberculosis. This third option has now been revised so that the live test rather than the interim strategy will be used on the farm where the tuberculosis outbreak occurred as well as in the surrounding area.

The live test produces a relatively small proportion of false positives (90 % specificity) but a large proportion of false negatives (41 % sensitivity overall). The sensitivity is also affected by the infective status of the badger. For infectious animals, the sensitivity is 62.0 % ($n = 71$), whereas for infected-but-not-infectious animals it is 36.6 % ($n = 374$) (R. Clifton-Hadley, personal communication). Thus it fails to detect infection in just under 40 % of all infectious animals and just under 65 % of all infected-but-not-infectious ones. As the test cannot therefore be used to detect infection in individual animals, as was the original intention, it is used to identify which setts contain badgers that react positively to the test. All animals caught in the first week are tested for bovine tuberculosis using the live test. If one infected animal is found, trapping continues at that sett for a further three weeks, with the aim of catching at least 90 % of all the badgers in positive setts. All badgers trapped at positive setts, except lactating sows, are killed. This strategy is designed to measure the effectiveness of eliminating badgers from infected setts around farms with infected cattle compared with taking no action on the surrounding land (MAFF 1993*a*).

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In addition to this new strategy, MAFF has also embarked on a research programme aimed at developing a vaccine for badgers (MAFF 1993*a*). One of the primary advantages of a vaccination campaign over one based on culling is the lack of disruption to the host population (White & Harris 1995). One vaccine for badgers, based on killed *Mycobacterium vaccae*, is currently undergoing field trials in Ireland (McCarthy 1993). However, although killed *M. vaccae* has proved very effective for the prevention and treatment of tuberculosis in humans, there is as yet little direct evidence of its efficacy in badgers (Stanford *et al.* 1993). Although no vaccine has yet been demonstrated to protect badgers against infection with *M. bovis*, and although the technical problems of developing such a vaccine that could be delivered orally through a bait are considerable, they are unlikely to be insoluble (D. Newell, personal communication). There is a consensus that the problem of *M. bovis* infection in badgers can not be solved without such a vaccine (Morris & Pfeiffer 1991; Stanford *et al.* 1993). However, it is unlikely that a fully tested and proven vaccine for badgers will be available for use in England until well into the next century.

Up to the end of 1993, MAFF had undertaken 1096 badger control operations, all but 42 of which were in southwest England (MAFF 1994). Notwithstanding a handful of local successes (e.g. Little *et al.* 1982), there has been no significant decline in bovine tuberculosis in cattle in the southwest since badger control began (MAFF 1993*b*), and bovine tuberculosis in cattle is still largely confined to the same limited areas (White *et al.* 1993). However, until the new policy announcement, there has been no attempt to monitor the relative success of the different control strategies employed since 1975. In the absence of any field data on which to base such an assessment, we use here a spatial stochastic simulation model (White & Harris 1995) to examine the theoretical requirements for control of the disease in badgers in an endemic situation. We also make the first quantified comparison of the efficacy of past, present and proposed future badger control strategies, and consider the likelihood of a vaccine-based strategy successfully reducing the level of infection in the badger population. So far, the relation between the prevalence of bovine tuberculosis in badgers and in cattle is unknown (White *et al.* 1993), and so any effects of disease control in badgers on the number of infected cattle herds are impossible to quantify. Therefore we have, of necessity, considered only the potential benefits of control on disease in the badger population.

2. METHODS

(*a*) *The model*

(i) *Structure*

The model used in these analyses is described in full by White & Harris (1995), and so only a brief resumé is given here. The terms used here are as defined by White & Harris (1995). The model is a spatial stochastic simulation that operates on a grid cell framework. The main grid contains 100 square cells arranged in a 10×10 grid. Each grid cell represents a territory that may contain a single group of badgers. The main grid is surrounded by a further 44 boundary cells which serve as a source of immigrants and emigrants to and from the grid. The model operates on a quarterly (three-monthly) timescale. The disease-free equilibrium badger population density is determined at the social group level, and the dynamics of group size are controlled through relations between group size, fecundity and density-dependent cub mortality. The transmission of *M. bovis* within and between groups occurs at predetermined probabilities, and dispersal may also act to transmit infection between groups.

(ii) *Badger group size*

White & Harris (1995) quantified the effects of group size on the potential rate of spread of bovine tuberculosis. They showed that there was a threshold disease-free equilibrium group size below which bovine tuberculosis was unlikely to spread and could not become endemic. Under normal circumstances of movement and intergroup contact, this was around six adults and yearlings, which is also the number in an average social group in Britain (Cresswell *et al.* 1990). If the rate of intergroup infectious contacts was artificially increased, the disease could sometimes spread in populations with a disease-free equilibrium group size of only four. However, even so it would not persist endemically. Therefore, disease-free equilibrium group sizes of six, eight and ten adults and yearlings are considered in this paper.

(iii) *Area occupied by each grid cell*

The mean density of main setts, even in the most favourable landscape types, does not exceed 0.7 per km^2, and so large areas of contiguous badger groups must be relatively uncommon. However, it is likely that badgers are living in relatively high density contiguous populations in those areas where badgers are believed to serve as a reservoir for bovine tuberculosis. Mean badger territory size in the four control areas described by Cheeseman *et al.* (1981) was 0.49 km^2, compared with a mean badger territory size in southwest England of about 0.7 km^2 (White & Harris 1995). At these densities, the total area covered by the grid of 100 territories in the model would be between 49 and 70 km^2. It is unlikely that contiguous badger territories, in which badgers are living at such high densities throughout, are found over significantly larger areas in southwest England. Therefore, this was deemed to be an adequate size for the simulation area. It was also not considered necessary to allow for any differences in territory size within the model due to the relatively small variations likely to be encountered in those areas where bovine tuberculosis is most frequent in badgers.

(iv) *Inter- and intragroup infection probabilities*

White & Harris (1995) showed that a variety of combinations of inter- and intragroup infection proba-

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bilities could replicate the observed patterns of spread and prevalence of bovine tuberculosis, although the values most likely to result in an endemic infection were an intragroup infection probability of 0.1 or smaller combined with an intergroup probability of 0.01 or smaller. The effects of variations in the combinations of these parameters on disease spread and persistence were quantified by White & Harris (1995); the highest mean prevalences at apparent equilibrium and the most rapid spread from a point source were obtained with a combination of an intragroup infection probability of 0.05 and an intergroup infection probability of 0.01. This combination was, therefore, used in this paper as the standard to compare the efficacy of different control measures. However, because these values are likely to show some variations both within and between populations, some care should be taken in interpreting the quantitative results presented here for other than comparative purposes: the results are based on idealized theoretical situations rather than real ones in specific locations.

(*b*) *Control of endemic disease by culling*

(i) *Prevalence of infection in the badger population in an endemic disease situation*

Of the badgers killed during control operations in southwest Britain between 1985 and 1991, 15.4 % were positive for *M. bovis* compared with 5.7 % in a sample of badgers killed on the roads in the same region over the same time period (MAFF 1993*b*). In 1992, the average prevalence in badgers killed as part of MAFF control operations was 20 %, compared with 10 % of dead badgers submitted to MAFF by the public in the same region (MAFF 1993*a*). The reasons for the differences in the two samples are discussed by White & Harris (1995).

The model used in these analyses showed that overall prevalence levels with the disease at apparent equilibrium should range between 11–22 %, the exact level depending on the disease-free equilibrium group size and the combination of inter- and intragroup infection probabilities used (White & Harris 1995). After an initial homogeneous seeding of infection throughout the population in the form of one infected male per group, apparent equilibria were reached after approximately 60 years, in those simulations where the disease persisted in the badger population. It is presumed that badgers first acquired bovine tuberculosis from cattle early this century when the disease was widespread. Thus, these equilibria were considered to be representative of the situations where the disease remains endemic in southwest England. After this time in the simulations, the infection would also have acquired the typical spatially heterogeneous pattern found in real badger populations. They were, therefore, used as the basis for simulations examining the requirements for the control of endemic disease.

(ii) *Efficiency of control*

Badger control took the form of single culling operations conducted simultaneously over all groups, and was initiated in the first quarter of year 60. The subsequent pattern of disease in the badger population after the onset of control was monitored for 40 years. The efficiencies of culling used in the simulations ranged from 50–95 %. For a particular culling efficiency, each individual badger was independently subjected to the specific equivalent probability of being culled. Thus for a 50 % culling efficiency, each badger was culled independently with a probability of 0.5.

(*c*) *Comparison of past and present strategies for the control of localized disease*

(i) *Local variations in the prevalence of bovine tuberculosis in badgers*

There is considerable local variation in the prevalence of bovine tuberculosis in badgers in the southwest. For example, Cheeseman *et al.* (1981) examined four areas of southwest Britain after outbreaks of bovine tuberculosis in cattle, and found that the prevalence of *M. bovis* in badgers ranged from 6.9 % to 34.5 %. In one recent control operation in Somerset over 65 % of badgers were infected (MAFF 1993*a*). However, the disease may not be endemic in badgers in all of those areas subjected to badger control operations. To compare the efficacy of different control strategies, it was decided therefore to examine artificially regulated initial infections rather than those arising from an endemic situation. This also had the advantage of ensuring reliable comparisons in terms of the level and pattern of initial infections.

(ii) *Size of the control area and the number of social groups subjected to control*

Under the gassing strategy, the mean area covered by each control operation was 7 km^2 (J. Wilesmith, personal communication). Under the clean ring strategy, the area subjected to badger control was, in theory at least, unlimited. However, in practice the mean area over which these control operations took place was also 7 km^2 (J. Wilesmith, personal communication). The control area ranged from 1–25 km^2, this latter figure being the exception rather than the rule (E. Krolick, personal communication). However, in exceptional circumstances, these figures could be much higher. For example, at Thornbury, an area of 100 km^2 was cleared of badgers by gassing between 1975 and 1981 (MAFF 1981), which represented around 90 social groups (M. Hutchings, personal communication). Under the interim strategy, the mean area of land covered by each control operation was less than 1 km^2 (R. Clifton-Hadley, personal communication).

For the analyses, each gassing operation was taken to encompass nine groups of badgers. The exact number of groups controlled under the clean ring strategy would vary according to whether any new infected animals were found in the outermost ring of groups trapped. Thus, for the clean ring strategy the following procedure was adopted in this analysis.

Initially, trapping was carried out on one group. If any infected badgers were caught, trapping was extended to the immediately adjacent surrounding four groups in the subsequent quarter (a total of five groups). If any infected animals were caught from these groups, trapping was extended to a total of nine

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groups in the next quarter. If more infected animals were caught, trapping was further extended in a centrifugal manner to a total of 13, 21 and 25 groups in subsequent quarters. Under the interim strategy, one social group would normally be trapped initially. However, prolonged trapping in a restricted area is likely to catch badgers from an additional one or exceptionally two adjacent groups as the badgers from the first group are removed (C. Cheeseman and E. Krolick, personal communications). Thus for the analysis this strategy was conducted over two groups. At the time of these analyses, the live test had yet to be introduced. However, the intention was that the live test would be applied over an area of up to about 10 km² around the farm with the initial infection in the cattle. Thus to compare the efficacy of the live test with the other strategies, it was conducted over nine social groups, as for the gassing strategy.

(iii) *Extent and prevalence of initial infection*

To compare past and future control strategies, it was necessary to determine the extent of the initial infection. This was estimated as follows. The area covered by a single control operation under the clean ring strategy was 7 km², or about ten social groups at a mean territory size of 0.7 km². However, under this strategy trapping continued until a ring of uninfected groups was found, and so the average number of infected badger groups associated with an outbreak of bovine tuberculosis in cattle was taken as five. To account for local variations in the prevalence levels observed in real populations, initial prevalences ranging from 10 % to 60 % over these five groups were considered. In real environments, these prevalences will arise locally against a background of endemic disease. However, because of the spatially heterogenous and dynamic nature of endemic disease, it was decided (for reasons of simplicity and reliability of comparisons) to ignore this background infection for these analyses. Thus in these simulations, the initial infections occurred against a disease-free background in the rest of the grid.

(iv) *Duration and timing of control*

There are no published data on the duration of badger control operations which could assist in the evaluation of past and present control strategies, and during the gassing strategy some areas were re-gassed if a significant number of badgers remained after the first control operation. Indeed, in a few exceptional cases, gassing may have been conducted on the same area of land over a period of up to six years after an outbreak of bovine tuberculosis in cattle (Clifton-Hadley 1994). However, gassing was generally much less prolonged. As the model of White & Harris (1995) operates on a quarterly timescale, it was assumed for the purposes of the present analysis that gassing was an instantaneous event within this timescale. The clean ring strategy was of more variable duration. Twenty-five groups represented the greatest extent of trapping operations under this strategy in this analysis, and this would be reached after 18 months from the onset of control. This was therefore used as the maximum duration of trapping operations under this strategy. Although some trapping operations during the interim strategy extended for several months, most lasted between five and six weeks (E. Krolick, personal communication). Trapping in the interim strategy was therefore considered as instantaneous for this analysis. Trapping under the live-test strategy proceeds for up to a total of four weeks at a particular sett, if infected badgers are detected among those trapped during the first week. Thus, trapping under this strategy was also considered as instantaneous for the analysis.

In the standard comparisons, control was initiated in the third quarter (seven months after the initial infection was set up). Although many control operations, especially in recent years, may not take place until more than 12 months after the initial infection in cattle is believed to have occurred, this approximates to the average delay of eight months (Cheeseman *et al.* 1981).

(v) *Efficiency of badger control under the different strategies*

There are few published data on the numbers of badgers killed during the early MAFF control operations. However, figures for the years 1987–1990 inclusive are available (MAFF 1989, 1990, 1991, 1993*b*). Over this four-year period, 2929 badgers were killed in 297 separate control operations: a mean of 9.86 badgers killed per operation. This period covered the interim strategy when the mean number of social groups encompassed by the control operations was around two. Because of the restricted area of land subjected to trapping under the interim strategy, trapping efficiency will be unequally divided between these two groups. It could be up to 80–90 % for the main group concerned (C. Cheeseman & E. Krolick, personal communications), but will certainly be much lower for the second group. For the purposes of this analysis, trapping efficiency was assumed to be the constant over the two groups. The mean size of a badger social group in the southwest is six adults and yearlings, plus cubs. Thus the total number of badgers (excluding cubs) subject to trapping would be around 12 adults and yearlings (approximately 14 individuals including cubs), and the maximum efficiency of control during the interim strategy would have been around 70 %. This was the value used for the analysis. Using figures for the number of control operations authorized each year under the clean ring strategy (MAFF 1983, 1984, 1985, 1986) and unpublished figures on the numbers of badgers killed over this period (R. Clifton-Hadley, personal communication), the mean trapping efficiency for the clean ring strategy was estimated to be around 80 %. It is likely that this higher efficiency was achieved because trapping was not confined to the piece of land where the cattle were thought to have contracted the disease, and possibly also due to a period of monitoring after the cessation of trapping.

There are no data to assist in an evaluation of the efficiency of the gassing strategy on a sett-by-sett basis, although it is likely to have been as high as, if not higher than the clean ring strategy, because an even longer monitoring period was employed after control.

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Table 1. *Summary of past and present badger control strategies employed by the Ministry of Agriculture, Fisheries and Food in areas where incidents of bovine tuberculosis in cattle have occurred and badgers have been considered the most likely source of infection. See text for explanation.*

strategy	dates	details	monitoring period	estimated efficiency per group	release of lactating females
gassing	1975–1982	hydrogen cyanide powder pumped into sett and entrances blocked Sett re-gassed if any signs of activity during monitoring period average area subjected to control 7 km²	12 months	90%	no
clean ring	1982–1986	badger groups removed in centrifugal manner by trapping around main and outlying setts until a 'ring' of uninfected social groups had been removed further trapping conducted if any signs of renewed badger activity average area subjected to control 7 km² (range 1–25 km²)	6 months	80%	yes
interim	1986–1994	badgers controlled by trapping on that part of the farm where the TB outbreak in cattle started, or the whole farm if it was not possible to be more precise average area subjected to control < 1 km²	none	70%	yes
live test	1994–	badgers trapped and a blood test used to identify infected badgers on a sett-by-sett basis on the farm where the TB outbreak occurred and over an area of approximately 10 km² surrounding it trapping initially takes place for one week; if an infected animal is found, trapping continues at that sett for another three weeks	none	70/80%	yes

The efficiency is known to have been 100% in certain experimental areas where badgers were eliminated and prevented from recolonizing (Little *et al.* 1982; MAFF 1993*a*). However, it is more realistic to assume a mean efficiency of no more than 90% for most areas, as the large volume and complex architecture of badger setts (Roper 1992) combined with the fact that some badgers may be using subsidiary or outlying setts which could remain undiscovered, will undoubtedly result in some loss of efficiency.

No data are yet available on the likely efficiency of trapping operations under the live test strategy. Under the interim strategy the percentage of the total number of badgers trapped which are caught in the first week ranges between 35% and 65% (C. Cheeseman, unpublished data). This averages 40% for January to September inclusive, whereas between October and December it is between 60% and 65%. However, under the live test strategy, trapping in the first week will be concentrated around the main sett rather than confined to the piece of land where the cattle were believed to have contracted the infection, so the efficiency of trapping in this period would be expected to be higher than under the interim strategy. Therefore in these analyses a trapping efficiency of 70% was used for the first week of control operations under the live test strategy. After this initial week, it was assumed that the total trapping efficiency will be similar to that obtained under the clean ring strategy, and a value of 80% per group therefore was used for any subsequent trapping. A summary of the main features of the different control strategies employed to date is given in table 1.

(*d*) *Effects of timing of control on the efficacy of control measures under the interim strategy*

Although the average time between an outbreak of bovine tuberculosis and the onset of badger control operations is eight months, many control operations are initiated well after this period. Of all control operations conducted by MAFF in 1992 and 1993, 12% failed to catch any badgers and a further 28% failed to catch any badgers with bovine tuberculosis (H. Kay, unpublished data). We therefore investigated whether delays to the onset of control operations might affect the probability of successful disease control and the probability of trapping infected badgers. The initial conditions of prevalence used were as described above. However, as well as control commencing seven months after the initial infection, the interim strategy was also initiated 13, 19, 25, 31 and 37 months after the initial infection.

(*e*) *Vaccination as a potential strategy for the control of endemic and localized disease*

(i) *Efficiency of an oral vaccine*

When assessing the likely efficacy of an oral vaccine, it is first necessary to obtain some estimate of the percentage of animals likely to be reached by the

vaccine. Some indication of this comes from work on bait uptake by foxes. In a review of studies in Europe and North America, Trewhella *et al.* (1991) found that in most cases baits were reaching 60–80% of foxes. No comparable studies have been published on badgers, although relatively high efficiencies might be expected in high-density areas where baits could be placed around the main setts. In field trials in Ireland, McCarthy (1993) reported a very high rate of bait uptake, although the proportion of animals taking baits was unknown. With any vaccination strategy, a further complicating factor is the proportion of animals that actually develop immunity after bait uptake. Because this will not be known until vaccine development is completed, for these preliminary analyses efficiency is taken to be a combined measure of bait uptake and the acquisition of immunity.

(ii) *Control of endemic disease*

To investigate the potential of vaccination to control endemic bovine tuberculosis infection in badgers, an endemic disease situation was set up as described above, using simulations where an initially homogeneous infection had persisted throughout a 60-year period, and where an apparent equilibrium had developed. Vaccination was initiated in the first quarter of year 60 throughout the grid, and both single and annually repeated vaccination campaigns were investigated.

(iii) *Control of localized disease*

To investigate the potential of vaccination to control localized disease outbreaks, the initial conditions were the same as for the comparison of the past and present culling-based strategies. Thus, five groups were initially infected with prevalences ranging from 10–60%. Vaccination took place over nine social groups, as for the gassing and live test strategies, and both single and annually repeated vaccination strategies were considered.

Table 2. *Frequency distribution of the percentage of simulations in which successful control of endemic disease was achieved within specific time periods after single culling operations of different efficiencies in badger populations with different disease-free equilibrium group sizes*

(Results are based on 50 simulations.)

disease-free equilibrium group size	culling efficiency (%)	time taken for successful control of endemic disease/years				
		< 5	5–9	10–14	15–19	⩾ 20
6	50	0	0	0	0	0
	60	0	0	0	0	0
	70	0	0	0	2	0
	80	0	6	8	6	8
	90	14	22	16	6	16
	95	44	36	12	4	2
8	50	0	0	0	0	0
	60	0	0	0	0	0
	70	0	0	0	0	0
	80	0	0	6	2	0
	90	6	28	22	8	10
	95	22	44	12	14	4
10	50	0	0	0	0	0
	60	0	0	0	0	0
	70	0	0	2	0	0
	80	0	4	8	4	2
	90	8	28	24	10	4
	95	30	40	14	4	0

3. RESULTS

(*a*) *Control of endemic disease by culling*

Figure 1 shows the relation between the proportion of badgers killed during a single culling operation and

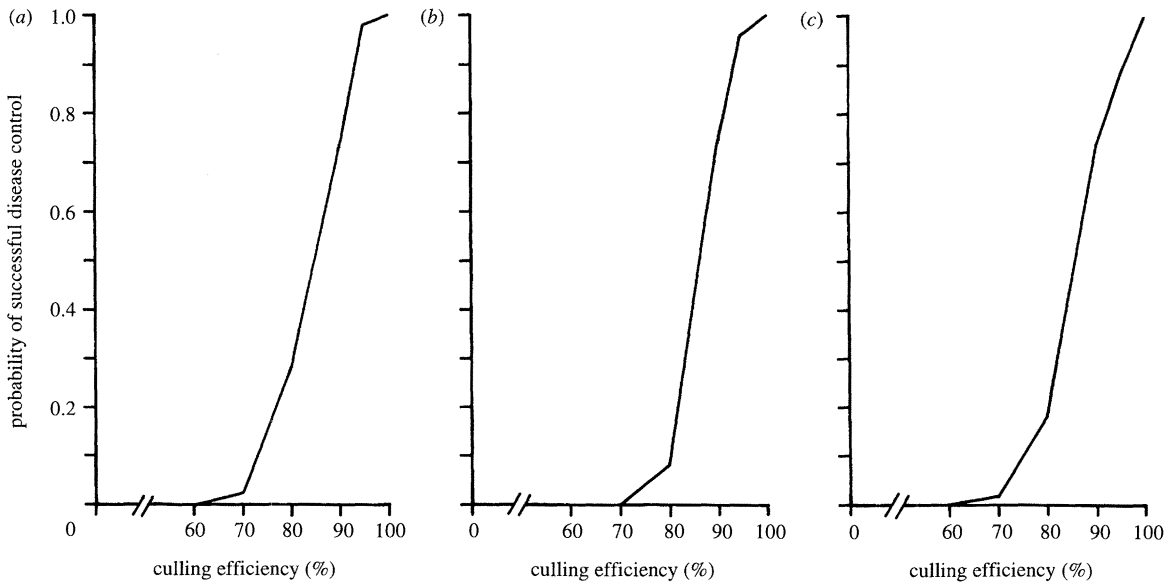

Figure 1. The relation between the proportion of badgers killed during a single culling operation and the probability of successful control of endemic bovine tuberculosis in badger populations with disease-free equilibrium group sizes of (*a*) six, (*b*) eight and (*c*) ten adults and yearlings. Results are based on 50 simulations.

Table 3. *The mean time (years) taken to control endemic disease if successful after single culling operations of different efficiencies*

(Results are based on 50 simulations. Means are shown ± standard error.)

disease-free equilibrium group size	culling efficiency (%)					
	50	60	70	80	90	95
6	–	–	15.0 ± 0.0	15.8 ± 2.2	12.4 ± 1.5	6.3 ± 0.7
8	–	–	–	13.8 ± 1.5	12.3 ± 1.4	8.7 ± 0.8
10	–	–	14.0 ± 0.0	14.8 ± 3.2	10.6 ± 0.9	6.9 ± 0.6

the probability of successful control of endemic bovine tuberculosis in a badger population, disregarding any possible external sources of reinfection such as infectious cattle or other wildlife. The frequency distribution the time taken to control the endemic disease is shown in table 2. The mean time taken to control the disease if successful is shown in table 3.

The probability of successfully controlling the disease for a given level of culling was generally smaller for larger group sizes. A single cull of approximately 90% of the population was required to ensure a reasonable probability ($p > 0.70$) of successfully controlling the disease. The mean time to eradicate the disease if control was successful fell markedly with increased culling efficiency; it ranged between 13.8 ± 1.5 ($n = 4$) and 15.8 ± 2.2 years ($n = 28$) for a cull of 80%, compared with between 6.3 ± 0.7 ($n = 49$) and 8.7 ± 0.8 years ($n = 48$) for a cull of 90%. The mean

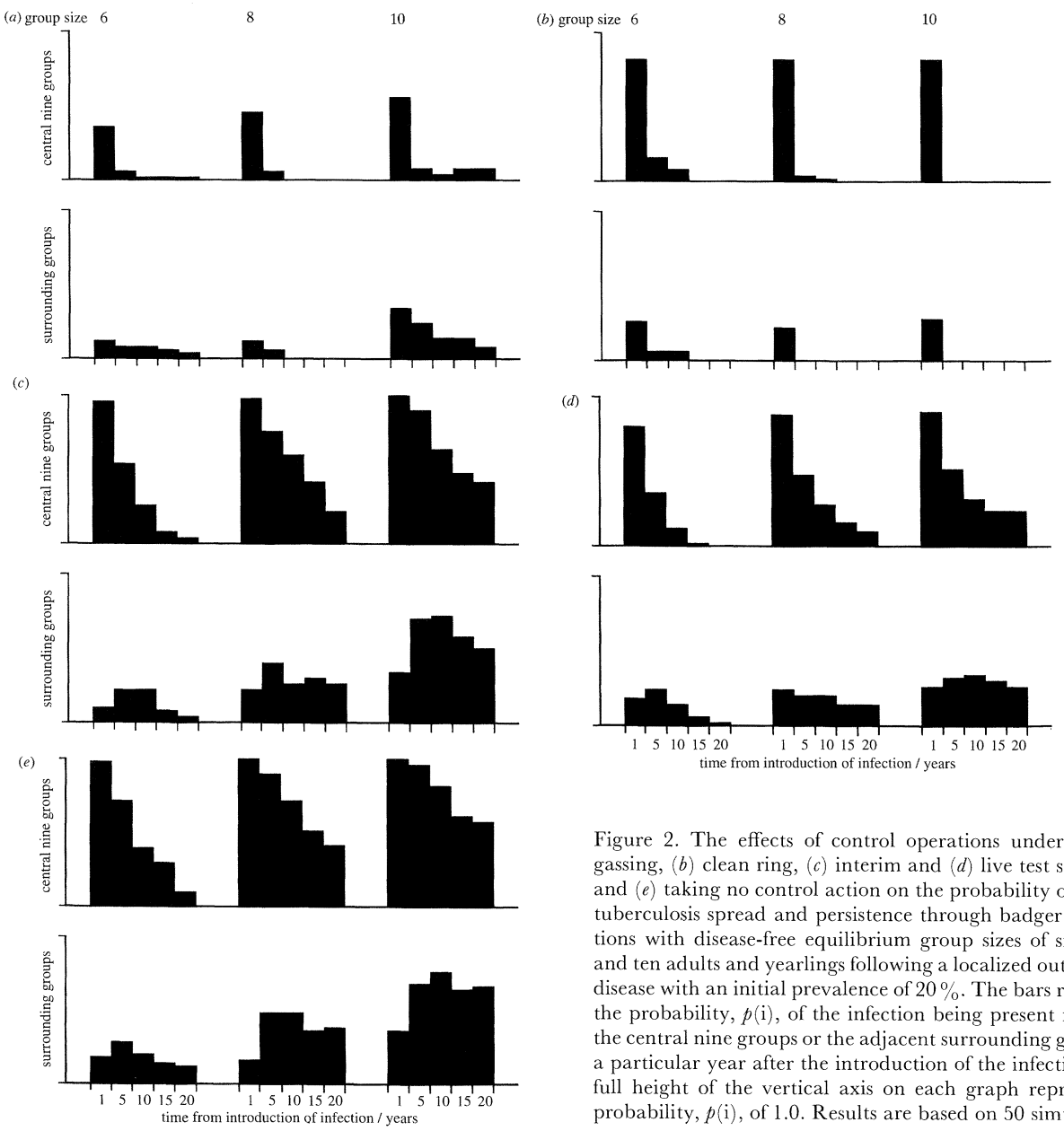

Figure 2. The effects of control operations under the (*a*) gassing, (*b*) clean ring, (*c*) interim and (*d*) live test strategies and (*e*) taking no control action on the probability of bovine tuberculosis spread and persistence through badger populations with disease-free equilibrium group sizes of six, eight and ten adults and yearlings following a localized outbreak of disease with an initial prevalence of 20%. The bars represent the probability, $p(\mathrm{i})$, of the infection being present in either the central nine groups or the adjacent surrounding groups in a particular year after the introduction of the infection. The full height of the vertical axis on each graph represents a probability, $p(\mathrm{i})$, of 1.0. Results are based on 50 simulations.

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Table 4*a*. *Probabilities of localized disease persisting to year five after control operations under the different culling-based strategies and with no control*

(Results are based on 50 simulations.)

control strategy	disease-free equilibrium group size	initial prevalence (%)					
		10	20	30	40	50	60
in the central nine social groups							
gassing	6	0.00	0.06	0.04	0.10	0.08	0.04
	8	0.04	0.06	0.16	0.10	0.12	0.28
	10	0.08	0.08	0.20	0.14	0.30	0.24
clean ring	6	0.26	0.16	0.10	0.04	0.04	0.02
	8	0.30	0.04	0.12	0.04	0.04	0.02
	10	0.30	0.00	0.08	0.02	0.02	0.00
interim	6	0.38	0.54	0.70	0.76	0.78	0.94
	8	0.42	0.76	0.88	0.98	0.94	0.92
	10	0.74	0.90	0.96	0.98	0.98	1.00
live test	6	0.16	0.36	0.34	0.46	0.44	0.40
	8	0.38	0.48	0.34	0.46	0.36	0.44
	10	0.56	0.52	0.42	0.70	0.64	0.62
no control	6	0.48	0.72	0.86	0.88	0.90	0.94
	8	0.62	0.90	0.88	0.94	0.98	0.96
	10	0.72	0.96	0.98	1.00	0.96	1.00
outside the central nine social groups							
gassing	6	0.00	0.08	0.08	0.14	0.08	0.06
	8	0.04	0.06	0.18	0.14	0.30	0.26
	10	0.12	0.24	0.20	0.26	0.22	0.28
clean ring	6	0.18	0.06	0.06	0.08	0.02	0.04
	8	0.14	0.00	0.10	0.02	0.02	0.00
	10	0.10	0.00	0.06	0.02	0.02	0.02
interim	6	0.08	0.22	0.34	0.36	0.48	0.48
	8	0.26	0.40	0.50	0.64	0.68	0.66
	10	0.32	0.70	0.72	0.74	0.76	0.84
live test	6	0.12	0.24	0.20	0.26	0.18	0.20
	8	0.20	0.20	0.32	0.34	0.28	0.38
	10	0.32	0.32	0.38	0.54	0.48	0.42
no control	6	0.20	0.28	0.36	0.42	0.38	0.50
	8	0.36	0.48	0.54	0.64	0.70	0.66
	10	0.30	0.68	0.74	0.94	0.86	0.82

Table 4*b*. *Probabilities of localized disease persisting to year 20 after control operations under the different culling-based strategies and with no control*

(Results are based on 50 simulations.)

control strategy	disease-free equilibrium group size	initial prevalence (%)					
		10	20	30	40	50	60
in the central nine social groups							
gassing	6	0.00	0.02	0.02	0.00	0.00	0.00
	8	0.00	0.00	0.02	0.02	0.02	0.08
	10	0.02	0.08	0.02	0.06	0.00	0.10
clean ring	6	0.00	0.00	0.00	0.02	0.00	0.00
	8	0.14	0.00	0.06	0.00	0.02	0.00
	10	0.12	0.00	0.04	0.02	0.00	0.00
interim	6	0.00	0.04	0.06	0.08	0.08	0.16
	8	0.16	0.22	0.38	0.28	0.44	0.36
	10	0.44	0.42	0.62	0.62	0.46	0.62
live test	6	0.02	0.00	0.04	0.00	0.06	0.02
	8	0.12	0.10	0.04	0.06	0.10	0.08
	10	0.22	0.24	0.12	0.20	0.16	0.16
no control	6	0.10	0.10	0.16	0.14	0.10	0.24
	8	0.26	0.42	0.28	0.40	0.32	0.42
	10	0.38	0.58	0.66	0.84	0.86	0.80
outside the central nine social groups							
gassing	6	0.00	0.04	0.02	0.02	0.04	0.00
	8	0.00	0.00	0.08	0.04	0.04	0.14
	10	0.04	0.08	0.10	0.16	0.06	0.20
clean ring	6	0.06	0.00	0.00	0.02	0.00	0.00
	8	0.12	0.00	0.04	0.02	0.00	0.00
	10	0.10	0.00	0.08	0.02	0.00	0.00
interim	6	0.04	0.04	0.04	0.06	0.12	0.12
	8	0.18	0.26	0.42	0.30	0.46	0.32
	10	0.42	0.50	0.64	0.68	0.70	0.70
live test	6	0.00	0.02	0.06	0.00	0.04	0.04
	8	0.20	0.14	0.10	0.14	0.18	0.14
	10	0.24	0.26	0.28	0.32	0.30	0.22
no control	6	0.10	0.12	0.22	0.20	0.10	0.30
	8	0.20	0.38	0.32	0.32	0.36	0.48
	10	0.46	0.66	0.74	0.80	0.80	0.76

population size at the end of the simulations was negatively correlated with the efficiency of the cull. However, the extent of these variations was relatively small throughout the range of equilibrium group sizes and culling efficiencies considered, being equivalent to between 60–85% depression of the population below the disease-free equilibrium size.

(*b*) *Comparison of past and present strategies for the control of localized disease*

Figure 2 shows the effects of the gassing, clean-ring, interim and live-test strategies in controlling a localized outbreak of bovine tuberculosis in the badger population with an initial prevalence of 20%, compared with taking no control action. The gassing, clean-ring and live test strategies all resulted in noticeable decreases in the probability of disease persistence over the no control option. However, the interim strategy generally had little effect when compared to the no control option, especially with regard to the long-term persistence of the disease and its spread to surrounding groups. The only situations in which the interim strategy had some effect in decreasing the probability of persistence and spread of the disease were at the lowest disease-free equilibrium group sizes and the lowest initial prevalence levels.

The probability of the disease persisting in the badger population was affected by the disease-free equilibrium group size rather than by the initial prevalence conditions. There was a tendency for the probability of persistence to increase with initial prevalence, but this was neither marked nor consistent. As there was no difference between the trends shown for different initial prevalence levels, detailed data for an initial prevalence of 20% only are presented in figure 2 as an example. Summary data for all initial prevalences considered are given in tables 4*a* and *b*. The mean numbers of badgers killed and the percentage of infected badgers among those killed under the different control strategies are shown in tables 5 and 6, respectively.

The most successful strategies for eliminating the disease, both within the nine central groups and in the surrounding ones, were the gassing and clean-ring strategies. For an initial prevalence level of 20%, the

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Table 5. *The mean numbers of badgers killed in control operations under different culling-based strategies*

(Results are based on 50 simulations. Means are shown ± standard error.)

control strategy	disease-free equilibrium group size	initial prevalence (%)					
		10	20	30	40	50	60
gassing	6	59.9 ± 0.5	59.8 ± 0.6	59.8 ± 0.5	59.3 ± 0.6	58.3 ± 0.5	57.9 ± 0.5
	8	79.0 ± 0.6	79.3 ± 0.7	78.2 ± 0.7	77.5 ± 0.7	77.5 ± 0.6	76.0 ± 0.7
	10	99.5 ± 0.7	99.8 ± 0.7	97.9 ± 0.8	97.1 ± 0.8	96.6 ± 0.7	96.0 ± 0.8
clean ring	6	101.7 ± 12.8	129.7 ± 11.8	162.4 ± 8.5	172.7 ± 602	175.9 ± 3.8	175.3 ± 5.2
	8	143.8 ± 16.7	229.9 ± 8.2	209.6 ± 11.8	228.1 ± 8.2	235.4 ± 4.8	235.5 ± 5.0
	10	197.5 ± 20.2	297.1 ± 6.1	277.4 ± 11.5	288.8 ± 6.0	290.3 ± 6.0	293.6 ± 1.5
interim	6	10.0 ± 0.4	9.4 ± 0.3	9.4 ± 0.3	8.7 ± 0.3	8.9 ± 0.3	9.2 ± 0.3
	8	13.1 ± 0.3	12.6 ± 0.4	12.8 ± 0.3	12.7 ± 0.4	12.4 ± 0.3	11.9 ± 0.4
	10	16.5 ± 0.4	16.4 ± 0.4	16.0 ± 0.4	16.6 ± 0.4	15.8 ± 0.4	16.0 ± 0.4
live test	6	26.6 ± 2.8	26.4 ± 2.6	33.2 ± 2.0	35.7 ± 1.7	38.3 ± 1.8	39.0 ± 1.3
	8	36.0 ± 3.4	48.6 ± 2.4	55.9 ± 1.7	51.5 ± 1.8	56.5 ± 1.5	54.6 ± 1.2
	10	46.8 ± 3.8	62.4 ± 3.5	70.9 ± 1.9	68.3 ± 2.2	71.6 ± 1.5	72.6 ± 1.4

Table 6. *The mean percentages of infected badgers among those killed per control operation under the different culling-based strategies*

(Results are based on 50 simulations. Means are shown ± standard error.)

control strategy	disease-free equilibrium group size	initial prevalence (%)					
		10	20	30	40	50	60
gassing	6	4.1 ± 0.2	8.1 ± 0.4	11.2 ± 0.5	16.4 ± 0.6	19.4 ± 0.6	23.1 ± 0.7
	8	4.6 ± 0.3	8.7 ± 0.3	12.6 ± 0.4	17.3 ± 0.5	22.1 ± 0.5	25.0 ± 0.6
	10	4.5 ± 0.4	8.8 ± 0.4	13.9 ± 0.5	17.9 ± 0.6	22.5 ± 0.5	25.0 ± 0.6
clean ring	6	1.8 ± 0.3	3.3 ± 0.4	4.2 ± 0.3	6.3 ± 0.3	6.5 ± 0.3	8.2 ± 0.3
	8	1.7 ± 0.2	3.3 ± 0.2	4.6 ± 0.3	5.8 ± 0.3	7.0 ± 0.3	8.6 ± 0.3
	10	2.0 ± 0.3	3.6 ± 0.2	5.3 ± 0.3	6.9 ± 0.3	8.4 ± 0.3	9.5 ± 0.2
interim	6	7.4 ± 1.3	17.6 ± 1.7	25.0 ± 2.2	37.4 ± 2.4	42.0 ± 2.3	38.3 ± 2.4
	8	11.7 ± 1.4	18.3 ± 1.5	22.1 ± 1.4	30.8 ± 1.7	37.8 ± 1.7	47.4 ± 2.3
	10	10.0 ± 1.2	16.7 ± 1.3	30.3 ± 1.7	35.3 ± 1.7	43.3 ± 2.5	45.1 ± 1.9
live test	6	5.6 ± 0.8	11.6 ± 1.4	13.3 ± 1.1	19.9 ± 1.2	22.1 ± 1.3	26.1 ± 1.1
	8	5.3 ± 0.6	10.8 ± 0.9	15.3 ± 0.7	19.5 ± 1.0	24.2 ± 0.9	27.9 ± 1.1
	10	6.0 ± 0.6	11.0 ± 0.9	16.4 ± 0.7	21.1 ± 0.9	25.0 ± 0.8	30.3 ± 0.9

probability of the disease persisting beyond five years after a control operation under the gassing strategy ranged between 0.06–0.08 for the central nine groups and between 0.06–0.24 for the surrounding ones. The equivalent ranges of probabilities for the clean ring strategy were between 0.00–0.16 for the central nine groups, and between 0.00–0.06 for the surrounding ones. The clean-ring strategy was more efficient than the gassing strategy in eradicating the disease from surrounding groups because of the greater potential area included in each control operation. However, this was reflected in the far greater number of badgers killed during control operations under the clean-ring strategy. The mean number of badgers killed per control operation ranged between 129.7 ± 11.8 and 297.1 ± 6.1 ($n = 50$) under the clean ring strategy compared with between 59.8 ± 0.6 and 99.8 ± 0.7 ($n = 50$) for the gassing strategy for an initial prevalence level of 20%. The number of badgers killed was positively related to the disease-free equilibrium group size for all strategies, but unrelated to the initial prevalence level. In contrast, the proportion of positive badgers among those killed during the different strategies was related to the initial prevalence level, but unrelated to the disease-free equilibrium group size. The mean proportion of positive badgers among those killed was smallest under the clean ring strategy, ranging between 3.3 ± 0.4 and 3.6 ± 0.2% ($n = 50$) for an initial prevalence level of 20%, and highest under the interim strategy, ranging between 16.7 ± 1.3 and 18.3 ± 1.5 ($n = 50$) for the same initial prevalence level.

The interim strategy also resulted in the lowest number of badgers killed, but it was by far the least successful of the four strategies considered. For an initial prevalence of 20%, the probability of the disease persisting beyond five years after a control operation under the interim strategy ranged between 0.54 and 0.90 in the central nine groups and between 0.22 and 0.70 in the surrounding ones. The live-test strategy was more effective at controlling the spread of disease than the interim one, but not nearly so effective as the gassing and clean-ring ones. Under the live-test strategy, the probability of the disease persisting beyond five years from an initial prevalence of 20% ranged between 0.36 and 0.52 in the central nine groups and between 0.20 and 0.32 in the surrounding ones. However, the mean number of badgers killed per control operation under the live-test strategy was approximately three times higher than under the interim strategy for low initial prevalence levels, and

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Table 7. *The mean percentages of infected badgers among those killed per control operation under the interim strategy conducted at different time periods after the initial infection.*

(Results are based on 50 simulations. Means are shown ± standard error.)

disease-free equilibrium group size	timing of control after initial infection/months	initial prevalence (%)					
		10	20	30	40	50	60
6	7	7.9 ± 1.1	21.5 ± 1.7	23.7 ± 1.7	33.3 ± 2.1	41.5 ± 2.6	42.8 ± 2.3
	13	8.4 ± 1.2	16.0 ± 1.5	20.6 ± 1.6	27.0 ± 2.0	31.3 ± 2.4	30.1 ± 1.9
	19	5.9 ± 1.3	17.0 ± 1.9	14.2 ± 2.1	28.2 ± 2.3	30.5 ± 2.8	33.6 ± 3.4
	25	6.6 ± 1.0	10.5 ± 1.4	12.0 ± 1.4	22.4 ± 2.2	23.1 ± 2.2	19.0 ± 2.0
	31	4.9 ± 1.3	14.7 ± 1.9	12.9 ± 1.9	21.9 ± 2.3	22.9 ± 2.4	23.7 ± 2.7
	37	2.9 ± 0.9	8.8 ± 1.6	11.8 ± 1.7	16.7 ± 2.8	18.5 ± 1.9	17.2 ± 2.2
8	7	11.1 ± 1.0	19.3 ± 1.5	21.6 ± 1.8	33.5 ± 1.8	37.9 ± 2.0	47.2 ± 2.6
	13	10.8 ± 1.3	12.8 ± 1.5	18.2 ± 1.2	24.5 ± 1.7	30.8 ± 2.0	36.0 ± 2.3
	19	9.3 ± 1.5	13.7 ± 1.6	20.9 ± 2.4	26.9 ± 1.9	33.4 ± 2.9	37.5 ± 2.4
	25	5.7 ± 0.9	11.8 ± 1.7	15.1 ± 1.6	22.7 ± 2.8	26.1 ± 2.4	28.3 ± 2.3
	31	6.3 ± 1.3	12.2 ± 1.7	17.9 ± 2.2	25.2 ± 2.3	26.7 ± 2.6	30.7 ± 2.8
	37	6.0 ± 1.0	10.9 ± 1.6	13.0 ± 1.7	20.5 ± 2.1	18.0 ± 2.0	25.2 ± 2.5
10	7	10.9 ± 1.0	17.7 ± 1.5	28.3 ± 1.8	35.6 ± 1.8	39.7 ± 1.9	46.2 ± 2.0
	13	8.7 ± 1.1	15.1 ± 1.2	24.1 ± 2.0	27.8 ± 1.7	34.7 ± 2.0	38.2 ± 2.3
	19	8.0 ± 1.1	15.1 ± 1.8	28.0 ± 1.9	32.8 ± 2.6	36.2 ± 2.5	38.7 ± 2.5
	25	9.3 ± 1.3	13.7 ± 1.4	20.9 ± 1.7	22.2 ± 2.0	25.3 ± 2.1	33.1 ± 2.8
	31	12.4 ± 1.9	14.8 ± 2.0	26.8 ± 2.7	25.6 ± 2.3	29.5 ± 2.4	33.2 ± 2.6
	37	9.1 ± 1.3	12.0 ± 1.5	20.8 ± 2.2	22.9 ± 1.9	27.6 ± 2.3	27.3 ± 2.7

Table 8. *The percentage of control operations conducted under the interim strategy at various time intervals after the initial infection in which no infected badgers were caught*

(Results are based on 50 simulations.)

disease-free equilibrium group size	timing of control after initial infection/months	initial prevalence (%)					
		10	20	30	40	50	60
6	7	40	2	4	2	0	0
	13	38	14	4	4	2	0
	19	60	20	32	10	8	4
	25	48	26	24	8	4	10
	31	68	32	36	14	18	16
	37	74	50	34	32	12	18
8	7	10	6	2	0	0	0
	13	26	18	0	2	0	2
	19	44	16	6	6	0	4
	25	44	28	16	6	6	4
	31	48	26	24	8	12	8
	37	46	30	26	8	16	8
10	7	14	4	2	0	0	0
	13	24	6	2	0	0	0
	19	36	18	4	2	0	0
	25	26	12	2	6	8	0
	31	18	22	4	8	0	2
	37	30	18	12	6	6	12

up to 4.5 times higher for higher initial prevalences, though less than under both the gassing and clean-ring strategies. The proportion of infected badgers among those killed under the live test strategy was almost half that under the interim strategy, and only slightly in excess of that under the gassing strategy.

(*c*) *Effects of timing of control on the efficacy of control measures under the interim strategy*

The timing of the onset of control operations under the interim strategy had little effect on the probability of disease spread and persistence, either within the central nine groups or in the surrounding ones. However, it did have an effect on the proportion of infected badgers among those killed during control operations (see table 7). The most extreme decreases with time of the proportion of infected badgers killed occurred with the highest initial prevalence levels and the smallest disease-free equilibrium group sizes. Thus for an initial prevalence level of 60% and a mean disease-free equilibrium group size of six adults and yearlings, the mean percentage of infected badgers killed during control operations fell from 42.8 ± 2.3 % ($n = 50$) in operations initiated seven months after the initial infection to 17.2 ± 2.2 % ($n = 50$) for operations conducted 37 months after the initial infection.

The proportion of control operations in which no infected badgers were caught was negatively related to the initial prevalence level, and positively related to the time between the initial infection and the onset of control. Table 8 shows the relation between the percentage of control operations in which no infected badgers were caught and the timing of the onset of control following the initial infection. The effects of timing of control on the probability of infected badgers being caught were more pronounced for lower initial prevalences and smaller group sizes. For an initial prevalence level of 10% in a population with a disease-free equilibrium group size of six adults and yearlings, the percentage of control operations in which no infected badgers were caught rose from 40% for operations commenced seven months after the initial

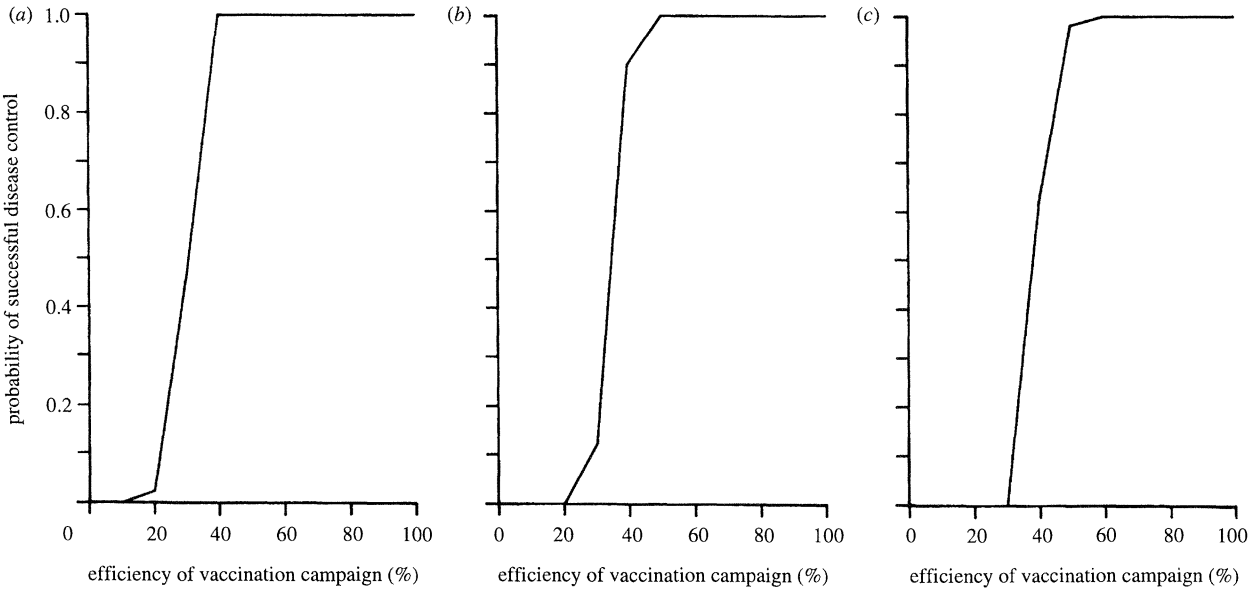

Figure 3. The relation between the efficiency of repeated annual vaccination and the probability of successful control of endemic bovine tuberculosis in badger populations with disease-free equilibrium group sizes of (*a*) six, (*b*) eight and (*c*) ten adults and yearlings. Results are based on 50 simulations.

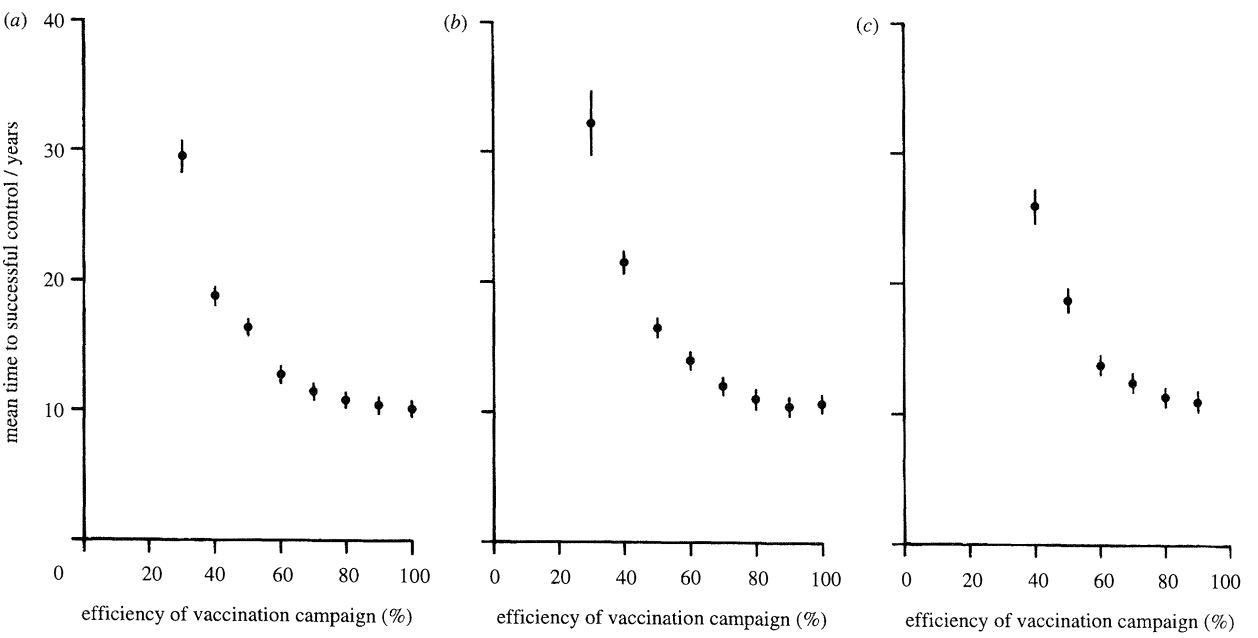

Figure 4. The relation between the efficiency of repeated annual vaccination and the mean time taken to successful control of endemic bovine tuberculosis in badger populations with disease-free equilibrium group sizes of (*a*) six, (*b*) eight and (*c*) ten adults and yearlings. Results are based on 50 simulations. Vertical bars denote standard errors.

infection to 74% for those commenced 37 months afterwards.

(*d*) *Vaccination as a potential strategy for the control of endemic and localized disease*

(i) *Control of endemic disease*

Single vaccination campaigns did not control endemic disease in any of the simulations conducted, even at the smallest disease-free equilibrium group size of six adults and yearlings, and were therefore disregarded from future analysis. Figure 3 shows the relation between the efficacy of repeated annual vaccination and the probability of successful disease control for the disease-free equilibrium group sizes considered. The frequency distribution of time taken to control the endemic disease is shown in table 9 and the mean time taken to control the disease if successful in figure 4.

The probability of success for a given vaccination efficiency was higher for a smaller disease-free equilibrium group size. However, repeated vaccination had a high probability of success in controlling the disease at all group sizes considered, even where the level of efficiency during each separate operation was relatively low. Time to successful control was slightly greater for larger mean group sizes, and was generally less for increased vaccination efficiencies. For a vaccination efficiency of 40%, the mean time to successful control

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Table 9. *Frequency distribution of the percentage of simulations in which successful control of endemic disease was achieved within specific time periods following repeated vaccination of different efficiencies in badger populations with different disease-free equilibrium group sizes*

(Results are based on 50 simulations.)

disease-free equilibrium group size	vaccination efficiency (%)	time taken for successful control of endemic disease/years				
		< 5	5–9	10–14	15–19	⩾ 20
6	10	0	0	0	0	0
	20	0	0	0	0	2
	30	0	0	0	2	44
	40	0	2	18	44	36
	50	0	0	40	40	20
	60	0	16	54	28	2
	70	0	28	62	8	2
	80	0	32	62	4	2
	90	0	44	50	4	2
	95	0	38	62	0	0
8	10	0	0	0	0	0
	20	0	0	0	0	0
	30	0	0	0	0	12
	40	0	0	12	24	54
	50	0	0	38	44	18
	60	0	4	46	46	4
	70	0	18	62	18	2
	80	0	28	64	6	2
	90	0	40	54	6	0
	95	0	30	64	6	0
10	10	0	0	0	0	0
	20	0	0	0	0	0
	30	0	0	0	0	0
	40	0	0	4	8	50
	50	0	0	24	38	36
	60	0	4	60	28	8
	70	0	16	60	20	4
	80	0	24	58	16	2
	90	0	28	64	8	0
	95	0	54	42	4	0

ranged between 18.8 ± 0.8 years ($n = 50$) and 26.1 ± 1.3 years ($n = 31$). This decreased to between 9.9 ± 0.3 and 10.7 ± 0.3 years (both $n = 50$) for a vaccination efficiency of 95%. However, the rate of decrease was not constant, and appeared to reach an asymptote at around 10 years after the onset of vaccination. This asymptote was reached at lower vaccination efficiencies for smaller disease-free equilibrium group sizes. The mean population size at the end of the simulations showed the inverse of the relations in figure 4, being positively but non-linearly correlated with vaccination efficiency. For vaccination efficiencies of 60% and above, for all disease-free equilibrium group sizes considered, the population size had recovered to within 15% of the theoretical disease-free levels at the end of the simulations.

(ii) *Control of localized disease*

A single vaccination operation, even at very high efficiencies, showed negligible improvements over a no control strategy in containing the spread and persistence of bovine tuberculosis throughout the badger population. The only real detectable differences occurred at the lowest initial prevalences in the central groups, and were greatest for the smallest equilibrium group sizes and the highest vaccination efficiencies. For an initial prevalence of 10%, the probability of the disease persisting in the central nine groups beyond five years after a single vaccination campaign of 95% efficiency ranged between 0.28–0.62, compared with between 0.48–0.72 where no control was conducted. The probabilities of the disease spreading to the surrounding groups in the same period after a 95% efficient single vaccination campaign and after no control were identical, ranging from 0.20–0.30.

The effects of repeated vaccination on a localized outbreak of the disease with an initial prevalence of 20% are shown in detail in figure 5, with summary data for all the different initial prevalence levels considered in tables 10*a* and *b*. All vaccination efficiencies resulted in a substantial long-term decrease in the probability of disease persistence in the central groups compared with the no control option. However, this decrease was not apparent until approximately ten years from the onset of control. Thus for the first five years, probabilities of disease persistence in the central groups were only slightly lower than with no control, even for repeated vaccination with an efficiency of 95%, where the probabilities of disease persistence beyond five years ranged between 0.22 and 0.58 for an initial prevalence of 20%. However, by the end of the 20-year simulation period, probabilities of persistence in the central groups had fallen to levels similar to those achieved by the specific control operations examined in §3*b*. For these central groups, the efficacy of a strategy of repeated vaccinations with an efficiency of 20% or 40% was an improvement over the interim strategy, and equivalent to the live test strategy, and repeated vaccinations with an efficiency of 60% or above were equivalent to the gassing or clean ring strategies. For the surrounding groups at the end of 20 years, repeated vaccinations with 60% efficiency gave results similar to those obtained by the live test; only vaccination efficiencies of 80% or greater achieved similar results to the gassing strategy, and none were so successful as the clean ring strategy.

4. DISCUSSION

The results of any modelling exercise which attempts to replicate field-based control techniques will always be a poor substitute for good field data. However, such data rarely exist. The modelled environment is, by necessity, a more-or-less idealized representation of reality. In real environments, environmental heterogeneity will affect badger density, the relative spatial positions of group territories, the degree of interaction between them, and probably also the manner in which control efforts are conducted and where they are concentrated. However, spatial stochastic models, such as the one employed here, offer several advantages over non-spatial, deterministic approaches. First, because probabilistic models incorporate chance effects, they

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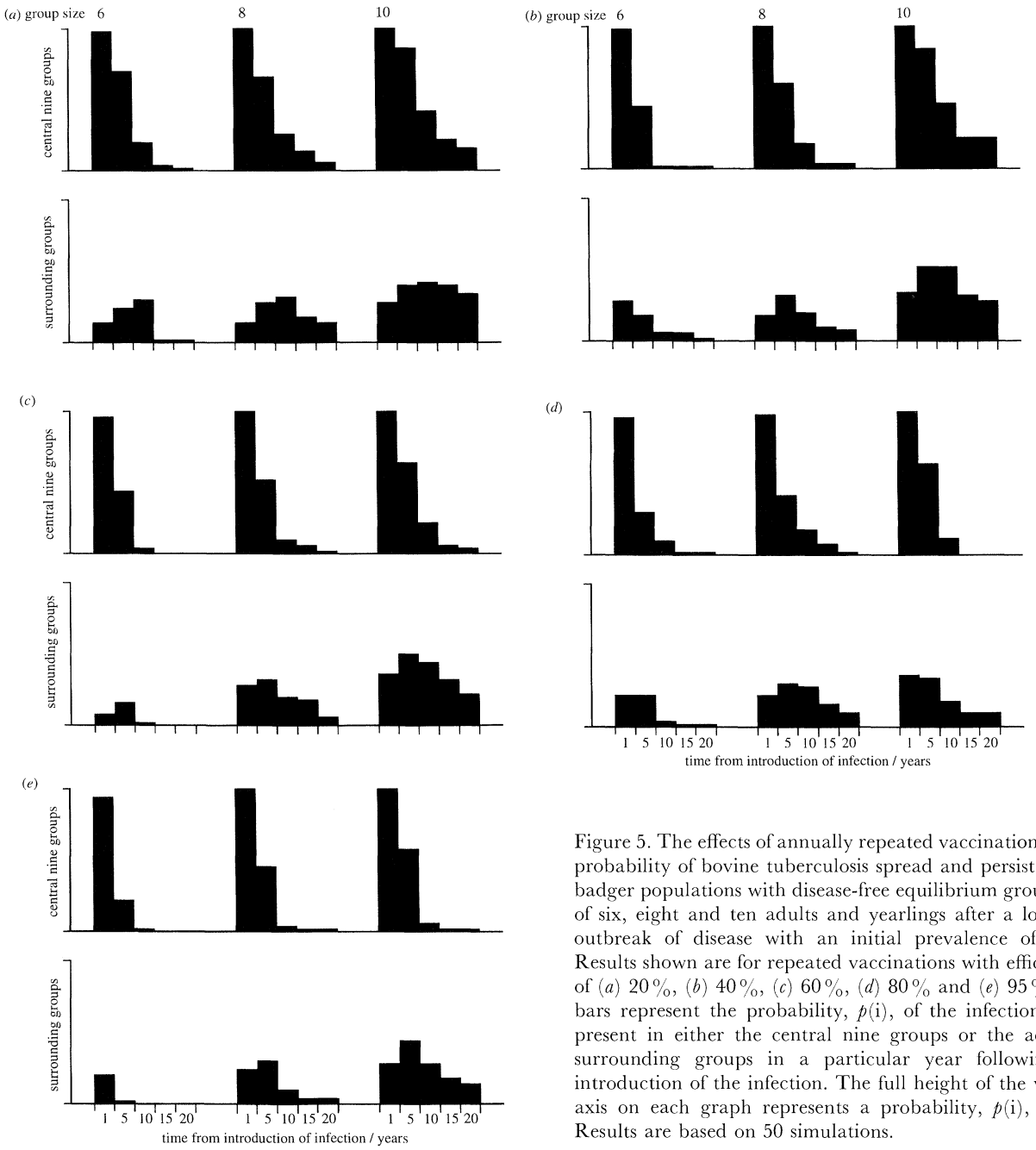

Figure 5. The effects of annually repeated vaccination on the probability of bovine tuberculosis spread and persistence in badger populations with disease-free equilibrium group sizes of six, eight and ten adults and yearlings after a localized outbreak of disease with an initial prevalence of 20%. Results shown are for repeated vaccinations with efficiencies of (*a*) 20%, (*b*) 40%, (*c*) 60%, (*d*) 80% and (*e*) 95%. The bars represent the probability, $p(\mathrm{i})$, of the infection being present in either the central nine groups or the adjacent surrounding groups in a particular year following the introduction of the infection. The full height of the vertical axis on each graph represents a probability, $p(\mathrm{i})$, of 1.0. Results are based on 50 simulations.

can be used to estimate the probability that a certain control measure will be successful; this can not be done in a deterministic setting. Secondly, spatial models can incorporate the spatial structure of both the environment and the host population, and so will provide a far more reliable indication of the comparative efficacies of different control methods in real environments. Furthermore, any predictions from such models can be tested relatively easily in the field.

In the comparative analyses of the different strategies employed by MAFF, we have had to assume certain trapping efficiencies and, in the case of the live test, different efficiencies for different stages of a control operation. The efficiencies used have been based on data from various sources, and the choice of values has deliberately erred on the optimistic side. Thus the estimated efficiencies of the different strategies will tend to be overestimates rather than underestimates. Furthermore, we have used single mean values rather than ranges of efficiencies for each control strategy. Badgers are not easy animals to trap, and their ease of trapping varies at different times of the year. Of all control operations conducted by MAFF in 1992 and 1993, 12% failed to catch any badgers at all, and it is likely that a certain percentage of operations under all the strategies failed to cull any animals. However, assigning statistical distributions of trapping efficiencies ranging from zero to some maximum value for each strategy would have required far greater assumptions than calculating a single mean value. Such an approach would also have complicated the comparison of the efficacy of the different strategies in the preliminary theoretical analysis presented here. Various factors such as badger density, the use of setts in a particular area, the previous exposure of the animals to trapping and habitat structure and com-

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Table 10*a*. *Probabilities of localized disease persisting to year five after control through annually repeated vaccination*

(Results are based on 50 simulations.)

vaccination efficiency (%)	disease-free equilibrium group size	initial prevalence (%)					
		10	20	30	40	50	60
in the central nine social groups							
20	6	0.22	0.70	0.74	0.86	0.88	0.84
	8	0.52	0.66	0.88	0.94	0.98	0.98
	10	0.74	0.86	0.98	1.00	0.98	1.00
40	6	0.32	0.44	0.60	0.76	0.86	0.76
	8	0.44	0.60	0.74	0.90	0.94	0.96
	10	0.56	0.84	1.00	0.98	1.00	0.96
60	6	0.20	0.44	0.48	0.76	0.62	0.78
	8	0.34	0.52	0.70	0.82	0.86	0.90
	10	0.42	0.64	0.86	0.98	0.96	0.88
80	6	0.24	0.30	0.42	0.66	0.58	0.72
	8	0.26	0.42	0.58	0.70	0.84	0.90
	10	0.48	0.64	0.78	0.96	0.88	0.96
95	6	0.12	0.22	0.36	0.72	0.66	0.68
	8	0.22	0.46	0.56	0.70	0.90	0.84
	10	0.36	0.58	0.76	0.84	0.96	0.90
outside the central nine social groups							
20	6	0.08	0.24	0.22	0.22	0.24	0.28
	8	0.26	0.28	0.48	0.64	0.60	0.72
	10	0.32	0.40	0.62	0.74	0.82	0.86
40	6	0.12	0.18	0.24	0.36	0.34	0.36
	8	0.30	0.32	0.42	0.50	0.72	0.60
	10	0.18	0.52	0.64	0.80	0.78	0.84
60	6	0.08	0.16	0.26	0.32	0.38	0.38
	8	0.20	0.32	0.56	0.54	0.38	0.58
	10	0.30	0.50	0.66	0.80	0.70	0.74
80	6	0.14	0.22	0.22	0.20	0.16	0.36
	8	0.28	0.30	0.50	0.38	0.52	0.54
	10	0.12	0.34	0.54	0.62	0.72	0.70
95	6	0.10	0.02	0.14	0.22	0.38	0.30
	8	0.10	0.30	0.44	0.56	0.56	0.48
	10	0.28	0.44	0.52	0.56	0.68	0.76

Table 10*b*. *Probabilities of localized disease persisting to year 20 after control through annually repeated vaccination*

(Results are based on 50 simulations.)

vaccination efficiency (%)	disease-free equilibrium group size	initial prevalence (%)					
		10	20	30	40	50	60
in the central nine social groups							
20	6	0.00	0.02	0.02	0.04	0.02	0.08
	8	0.06	0.06	0.10	0.10	0.12	0.24
	10	0.08	0.16	0.40	0.38	0.50	0.40
40	6	0.00	0.02	0.02	0.00	0.00	0.02
	8	0.04	0.04	0.00	0.06	0.12	0.14
	10	0.16	0.22	0.20	0.10	0.18	0.22
60	6	0.00	0.00	0.00	0.02	0.00	0.02
	8	0.00	0.02	0.02	0.06	0.08	0.04
	10	0.00	0.04	0.10	0.12	0.10	0.06
80	6	0.00	0.02	0.00	0.02	0.00	0.00
	8	0.00	0.02	0.00	0.00	0.00	0.06
	10	0.00	0.00	0.08	0.02	0.10	0.10
95	6	0.00	0.00	0.00	0.00	0.00	0.00
	8	0.00	0.02	0.00	0.00	0.08	0.00
	10	0.02	0.02	0.02	0.10	0.04	0.02
outside the central nine social groups							
20	6	0.00	0.02	0.04	0.08	0.08	0.08
	8	0.10	0.14	0.14	0.20	0.20	0.42
	10	0.18	0.34	0.60	0.58	0.62	0.62
40	6	0.04	0.02	0.02	0.02	0.04	0.04
	8	0.14	0.08	0.06	0.14	0.24	0.24
	10	0.20	0.32	0.38	0.38	0.44	0.44
60	6	0.02	0.00	0.02	0.04	0.02	0.08
	8	0.06	0.06	0.14	0.12	0.14	0.22
	10	0.08	0.22	0.28	0.48	0.36	0.30
80	6	0.04	0.02	0.04	0.08	0.04	0.04
	8	0.08	0.10	0.02	0.08	0.10	0.18
	10	0.06	0.10	0.20	0.18	0.24	0.36
95	6	0.00	0.00	0.00	0.00	0.04	0.06
	8	0.00	0.04	0.08	0.08	0.14	0.14
	10	0.10	0.14	0.18	0.28	0.28	0.28

position are all likely to have some effect on the efficiencies of trapping operations in specific areas. Data quantifying these various effects are as yet unavailable, although they could be readily incorporated into the model. Were such data available, this would considerably strengthen the predictive power of the model as it could be refined to match specific locations and the results could then be used to design control operations to suit particular locations and circumstances. A flexible approach such as this would be likely to yield significantly better results than the blanket-type approach currently employed.

The theoretical comparisons of the different strategies presented here also assumed an island of initial infection against a disease-free background. This will not be the case in real environments where endemic diease is likely to exist in the surrounding groups (which will therefore serve as a reservoir for reinfection of the controlled groups). Thus in real environments, in the absence of control, the disease would persist endemically throughout the grid with a probability close to 1.0 for at least the period of the simulations. It is important to note therefore that the results of these analyses, although they are valid for comparative purposes, are not intended to be used as quantitative predictors of the successes of specific control strategies in real environments. In such environments, where endemic disease is present in surrounding groups, the effectiveness of all strategies will be reduced considerably below the levels indicated in this paper.

Of the culling-based control strategies employed over the past twenty years, the most effective in terms of reducing the probability of spread and persistence of the infection in the badger population were probably the gassing and clean ring strategies. The results from the model for the efficacy and number of badgers killed in each operation under the clean ring strategy are likely to be overestimates. This is because the more complicated control protocol associated with the clean ring strategy can be followed rigourously in the artificial environment created by the model, whereas a variety of complications will undoubtedly arise on the ground, thereby limiting the application of the strategy. However, even allowing for this, it is still likely to have been a very effective strategy, mainly due to the large area potentially encompassed by the control operations and the long time period over which they were conducted. Although the proportion of each

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badger group killed under the clean ring strategy was less than under the gassing strategy, the dynamic element in the clean ring strategy was its strength. If control efforts moved outwards from a source of infection at a faster rate than the infection itself, the probability of successful control, both in the groups immediately surrounding the source, but especially in the groups surrounding these, would be greatly increased. None of the other strategies had this dynamic element to them, and thus once the infection spread into the badger population beyond the designated control zone, the long-term probability of successful control would be much reduced. Any infection in the surrounding areas would serve as a reservoir to reinfect any badgers remaining within the control area, or which could be reintroduced by colonizing animals. Although the area subjected to control was monitored for signs of badger activity after the control operation had been completed, given the slow colonisation rate shown by badgers and the chronic nature of the infection, a maximum monitoring period of twelve months was probably too short to ensure that reinfection would not occur. This is likely to explain, at least in part, the pattern of reoccurrence of the disease in the same locations, although local environmental conditions promoting the transmission of the disease or badger population density *per se* will also undoubtedly contribute to this pattern (White & Harris 1995).

The results of the model show that the interim strategy has been the least effective of those employed to date. Indeed, where there was a large disease-free equilibrium group size or where the initial prevalence of infection was high, control operations under the interim strategy offered little or no improvement over a no control option. The main reason for the lack of efficacy of the interim strategy lies in its application in a restricted area which encompassed an average of just two group territories, although not necessarily the entire territories. Furthermore, interim trapping operations conducted some time after the initial infections had a relatively high probability of not catching any infected badgers, and this is the pattern of events shown by MAFF culling records. Although it is possible that this indicates that there were no infective badgers to be caught in these areas, it is more likely that the infection continued to persist in badgers in the area, but that it did so in badgers in adjacent groups or in badgers in the same groups which did not use the specific areas where trapping was conducted. Subdominant individual badgers will not necessarily use the entire group territory for foraging, and the spatial use of the range by all members of the group may vary seasonally (Cresswell & Harris 1988; Brown 1993). It is, therefore, highly probable that infected badgers would be able to persist in the immediate vicinity of farms where infection has been detected despite control operations conducted under the interim strategy.

The timing of interim control operations following an initial outbreak generally had no effect on the probability of disease persistence or spread due to the basic inadequacy of the strategy. Indeed, delays in the onset of control operations are likely to have been of far greater significance under the gassing and clean ring strategies due to their inherently greater efficacies. The fact that some operations have taken place long after the initial outbreak of bovine tuberculosis in the cattle or in a piecemeal fashion over extended periods is likely to be one reason why the disease continues to persist in the badger population despite all the control efforts over the years.

The live test offered some improvement over the interim strategy in controlling the spread and persistence of the infection in badger populations, especially where the disease-free equilibrium group size and/or the prevalence of infection were high. However, the live-test strategy still failed to prevent the spread or persistence of the infection in the badger population in approximately half the simulated control operations. Furthermore, it was responsible for the deaths of approximately four times as many badgers each year as the interim strategy, and the proportion of those killed which were infected was approximately half that obtained from control operations conducted under the interim strategy.

The potential for any culling-based control operation to cause perturbation to the badger population has been ignored in this analysis because precise effects have not yet been quantified. However, perturbation is known to increase intergroup movements in badger populations (Cheeseman *et al.* 1988), and disruption to existing social hierarchies may lead to an increased potential for disease spread (Brown *et al.* 1994; MAFF 1994; White & Harris 1995). Thus perturbation may reduce the efficacy of any culling-based control operations. Any such effect will be more pronounced for control operations conducted under those strategies which are inherently less effective, such as the interim and live test strategies, and for those control operations which are conducted over a long period of time.

Although it should be possible to control endemic bovine tuberculosis in badger populations by culling, a very high culling efficiency (in excess of 80%) is required for a reasonable degree of certainty of the outcome. Such high culls result in substantial reductions in overall badger population density which takes a very long time to recover. With a single cull of 95% over the entire grid, it took an average of eight years to erradicate the disease, and 40 years after the initial cull the badger population remained 85% below the disease-free equilibrium density. This very slow rate of recovery predicted by the model is supported by field data. In Norfolk and Suffolk, about 1450 badger social groups were exterminated by gamekeepers before World War I. Now there are only around 150 badger social groups in the two counties and there is little evidence that the badgers are recolonizing formerly occupied areas despite a dramatic reduction in the level of persecution (Harris 1993). These results reflect both the low intrinsic rate of increase of badgers and the slow rate of colonization of empty territories. The effects of an intensive culling strategy on the badger population would therefore be severe and long-lasting, and it is doubtful that such an extreme level of intervention would be acceptable given the importance of the British badger population

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from a European conservation perspective (Harris *et al.* 1995).

Disease control through vaccination offers great potential advantages over culling-based approaches as vaccination will not lead to dramatic reductions in badger population density. However, the control of localized disease outbreaks by single vaccination campaigns was unsuccessful. Campaigns involving repeated vaccinations did provide some degree of success for the control of localized disease when they were applied to the same size areas as covered by the gassing and live test strategies but would probably have been more effective if they had been conducted over a wider area.

In contrast, there is great potential for a vaccine to eradicate endemic infection from the badger population. With annually repeated vaccination campaigns, there is a high probability of successful control of endemic disease even with a relatively low (40–50%) efficiency for the vaccination programme, and it is likely that the efficiency would be far in excess of this. Vaccination would not adversely affect the overall density or social structure of the badger population, and successful control would be achieved more rapidly. In fact, in areas where endemic disease is currently reducing badger population density, vaccination should lead to significant population increases. However, even with repeated vaccination and a high efficiency, the mean time to successful control of the endemic disease never fell below about ten years. This is due both to the chronic nature of the infection in badgers and the longevity of badgers, which may live for up to 11 years in the wild (Neal & Cheeseman 1991). To conduct an intensive vaccination campaign throughout the southwest would be prohibitively expensive. However, bovine tuberculosis infection in badgers in the southwest of Britain appears to be confined to certain relatively restricted areas (MAFF, unpublished data). These coincide with the majority of the outbreaks of bovine tuberculosis in cattle, and so it may be possible to use vaccination to control endemic disease in badgers in these areas, which currently total around 12% of southwest Britain (White *et al.* 1993).

Although the current analyses have of necessity adopted a theoretical approach to compare the efficacy of different control strategies, they have served to highlight the strengths and weaknesses of the different badger control strategies and indicate possible ways forward. All past strategies have failed, to varying degrees, for the same fundamental reasons: they were reactive responses to infection in cattle and there was no attempt to monitor the success or progress of the control strategy. A proactive response would have a greater potential for success than such reactive responses, particularly if a monitoring programme and regular reassessment of progress are a central part of the control strategy. To achieve this goal, the spatial stochastic simulation model used in this paper could be matched to real environments by restructuring the spatial configuration of badger groups within the basic grid, controlling interactions between them and adjusting the disease-free equilibrium group sizes according to quantified relations between habitat and carrying capacities. Developing the model in this way would provide a basis for evaluating the best control strategies in specific locations, and as such would enable the development of an alternative proactive management strategy. The value of such an approach has been clearly demonstrated by the coypu (*Myocastor coypus*) eradication programme in Britain, where a simulation model was used to devise the control programme and subsequently monitor its success (Gosling & Baker 1989). Without a scientific basis to future badger control programmes in Britain, the chance of eliminating bovine tuberculosis from the badger population appears remote.

5. CONCLUSIONS AND RECOMMENDATIONS

1. The live test strategy will not lead to significant improvements in the overall disease situation in the southwest. It will result in more badgers being killed each year which will increase disruption to the badger population and possibly lead to local increases in the spread of infection following the cessation of control. Control efforts will be targeted less accurately towards infected badgers rather than more accurately as was the original intention.

2. An ecological-economic analysis should be conducted to assess whether the live test strategy should continue. This should measure the possible economic benefits in terms of potential reductions in the incidence of bovine tuberculosis in cattle and any consequent reductions in the total compensation paid to farmers against: (i) the economic costs of the continued deployment of resources on a strategy that will provide no long-term solution to the problem; (ii) the economic costs of artificially restocking the badger population with uninfected animals in those areas in which the population has been dramatically reduced by control measures; and (iii) the potential ecological costs of killing an increasing proportion of the badger population in southwest England.

3. No reactive strategy based on responsive action to control localized incidences of infection in badgers will provide a long-term solution to the problem of bovine tuberculosis in badgers, whether it is based on culling or vaccination. A proactive strategy directed in those areas with a recent history of bovine tuberculosis in badgers has a far greater potential for success.

4. The most effective strategy for the long-term eradication of bovine tuberculosis from the badger population in southwest England would involve repeated vaccination directed proactively in those areas with a history of bovine tuberculosis infection in badgers.

5. A cost-benefit analysis should be conducted to assess whether continued research into the development of a vaccine for badgers is likely to offer significant net economic benefits in the long-term over a strategy of no control.

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