When to kill a cull: factors affecting the success of culling wildlife for disease control

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Abstract

Culling wildlife to control disease can lead to both decreases and increases in disease levels, with apparently conflicting responses observed, even for the same wildlife-disease system. There is therefore a pressing need to understand how culling design and implementation influence culling's potential to achieve disease control.

We address this gap in understanding using a spatial metapopulation model representing wildlife living in distinct groups with density-dependent dispersal and framed on the badger-bovine tuberculosis (bTB) system. We show that if population reduction is too low, or too few groups are targeted, a 'perturbation effect' is observed, whereby culling leads to increased movement and disease spread. We also demonstrate the importance of culling across appropriate time scales, with otherwise successful control strategies leading to increased disease if they are not implemented for long enough. These results potentially explain a number of observations of the dynamics of both successful and unsuccessful attempts to control TB in badgers including the Randomised Badger Culling Trial in the UK, and we highlight their policy implications.

Additionally, for parametrisations reflecting a broad range of wildlife–disease systems, we characterise 'Goldilocks zones', where, for a restricted combination of culling intensity, coverage and duration, the disease can be reduced without driving hosts to extinction.

Keywords: *Mycobacterium bovis*, culling, disease control, tuberculosis, badgers, bovine TB

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1 Introduction

Population reduction through culling is often used to control disease in wildlife populations, especially when those populations act as a reservoir for livestock disease [1]. Culling has been successful in reducing infection levels in wildlife–disease systems such as bovine tuberculosis (bTB) in possums (*Trichosurus vulpecula*) [2]. However, culling has also been found to be ineffective or even detrimental to disease control in many disease systems, including: rabies and foxes (*Vulpes vulpes*), rabies and bats (*Desmodus rotundus*), classical swine fever (CSF) and wild boar (*Sus scrofa*); and devil facial tumour disease and Tasmanian devils (*Sarcophilus harrisii*) [3–7]. For a given host-pathogen system, culling can also lead to an increase or decrease in disease levels under different scenarios, as has been observed for bTB in badgers (*Meles meles*) [8].

Culling can lead to changes in behaviour, immunological response, movement frequency and dispersal propensity, and such perturbations are seen in many wildlife populations [5, 9–12]. Where increases in disease transmission via these social processes outweigh the reductions in transmission anticipated from lower numbers of susceptible and infected hosts, causing levels of disease to increase post-culling, this is referred to as the 'perturbation effect' [13]. There is a need to understand what elements of culling implementation influence the presence or absence, and magnitude, of any resultant perturbation effect.

The Randomised Badger Culling Trial (RBCT) in the UK is the best-documented example of wildlife culling for disease control and investigated the effects of culling badgers on bTB levels [14]. Implemented between 1998 and 2005, and covering 3000 km^2 in the southwest of England [15], the RBCT is one of the largest-ever controlled veterinary epidemiology field experiments. The aim of the RBCT was to quantify the impact of culling badgers on the number of *Mycobacterium bovis* herd breakdowns in cattle [14]. In the RBCT, it was found that culling could increase disease incidence in cattle populations [16], increase disease prevalence in badgers in the target area [17] and increase disease in surrounding areas [8]. These influences of population reduction extended beyond

the culling period, with increased disease prevalence also found in the following year [18]. However, such rises in disease are not observed in all badger-bTB systems [19]. Reductions in bTB breakdowns in sympatric cattle herds have been seen within core areas in the RBCT, following consecutive years of proactive culling [18]. Reductions in bTB outbreaks in cattle have also been observed following proactive culling of sympatric badgers in Ireland [20, 21].

As the badger-bTB system highlights, differing responses to population reduction are encountered within the same host-pathogen system [22]. It is not fully understood what determines the efficacy of disease control. Culls vary in their intensity, spatial implementation, and duration. The removal rate is dependent on a combination of the resources available and the efficacy of culling techniques. Spatial heterogeneities in culling (measured by proportion of groups within the target population where culling is applied) are influenced by variations in land accessibility, landowner permissions, and knowledge of territory use. The duration of culls is also variable, often driven by resources and policy recommendations, and in some cases have been halted when results appeared to increase disease risk [16].

A number of species-specific models have explored the increases in disease levels following population reduction. Abdou *et al.* [23] and Smith *et al.* [24] incorporated a phenomenological 'perturbation parameter', based on *a priori* assumptions of how population reduction will affect disease transmission. However, some authors have approached the problem mechanistically. Birth rates are density dependent for numerous wildlife species [25], so one proposed mechanism for the perturbation effect is the compensatory increase in birth rates following population reduction in host populations. Using a non-spatial transmission model exploring a system with density-dependent host birth rates, Choisy *et al.* [26] demonstrated that interactions between population reduction and density-dependent effects can lead to an increase in infective individuals following culling.

A second proposed mechanism for the perturbation effect is spatial perturbation observed following population reduction. Dispersal is an important mechanism in disease transmission, as it is at least partially responsible for geographic disease spread. Dispersal is the movement of individuals between social groups, in search of new resources and mating opportunities. In wildlife, the rate of (successful) dispersal may typically depend on the density of the target population [27, 28], in which case culling can lead to an increase in dispersal into targeted groups from neighbouring areas, a phenomenon called the 'vacuum effect' [13]. The vacuum effect has been implicated in several disease systems [5, 9–13, 29–31] in which culling failed to control the disease.

Lintott *et al.* [32] found that, in spatial two-patch systems, increases in dispersal in response to culling can limit the efficacy of control at the metapopulation level. Prentice *et al.* [33] consider density-dependent dispersal rates and the population-level implications of individual responses to the disruption of social and spatial structures induced by population reduction measures. Enhanced transmission was shown to be an emergent property in systems where density dependence limits dispersal in 'full' stable spatially structured populations. They found that when population reduction is not sufficiently severe, enhanced transmission can lead to perturbation effects that are largest for systems with low levels of heterogeneously distributed disease. In such systems, insufficiently severe population reduction destabilises this structure, leading to elevated rates of dispersal that spreads infectious individuals to uninfected social groups. This leads to a rise in the effective transmission rate and rapid increases in the area affected by disease (potentially expanding risks to sympatric species). This previous work has demonstrated that social perturbations arising from the interaction between density-dependent dispersal and population reduction can result in an increase in disease levels. However, it has not explored how spatial and temporal elements of culling implementation alter the outcome of control programmes and the size of the perturbation effect.

It is important to understand what elements of culling implementation maximise the chances of a positive outcome, and how to minimise unintended consequences of enhanced transmission. The aim of this study is to determine how the outcome of disease control programmes can be affected by the rate of population reduction achievable, the proportion of land that is accessible, and the duration of culling programmes. We use a spatial metapopulation model that incorporates density-dependent dispersal, a known mechanism of spatial perturbation following population reduction.

2 Methods

We use the spatially explicit stochastic susceptible-infective SI metapopulation model introduced by Prentice *et al.* [33], to investigate the influence of spatial and temporal aspects of culling implementation. We explore the impact of spatial coverage, within-group removal rate, and duration of culling on disease levels. This model captures a generic host-pathogen system, and we use bTB as a framing in this study with parameter values chosen to reflect badger-bTB dynamics, as this is one of the best-studied systems.

Results are obtained from the mean of 1000 realisations of the stochastic model. The model was written in `C++` and the results plotted in GNU Octave [34] and Julia [35].

2.1 Demography and epidemiology

The model consists of a metapopulation divided into multiple sub-populations (groups) on a 10×10 lattice, each indexed by subscript i. As each group in the model is small (around 18 individuals at equilibrium), group-level stochastic pathogen fade-out and stochastic host extinction might have important effects on dynamics, necessitating a stochastic model. Demographic stochasticity is represented using a discrete Markov process in continuous time with exponentially distributed waiting time between events. The model is simulated using the Gillespie algorithm [36, 37] with events and their corresponding rates shown in Table 1. When fewer than two individuals

Table 1: Default event rates for the stochastic SI model. Event rates during time interval $(t, t+\delta t)$ and corresponding effects in the spatial stochastic model. Groups are indexed by i, and neighbouring groups are indexed by j. $z = 1/4$ is the reciprocal of the number of neighbouring groups. Note that the birth rate can take a minimum value of zero.

Event	**Rate**	δS_i	δI_i	δS_j	δI_j
Birth of S_i	$rN_i(1-N_i/c)$	+1	0	0	0
Death of S_i	$(\mu+p_2)S_i$	-1	0	0	0
Death of I_i	$(\mu+\nu+p_2)I_i$	0	-1	0	0
Infection of S_i	βSI	-1	+1	0	0
Dispersal of S_i to group j	$\kappa z S_i f(N_j)$	-1	0	+1	0
Dispersal of I_i to group j	$\kappa z I_i f(N_j)$	0	-1	0	+1

remain, the population is considered to be extinct, and the simulation is stopped.

We assume density-dependent (logistic) growth, with natural mortality at rate μ (i.e. mortality for any reason other than via disease or as a consequence of population reduction), and intrinsic reproduction rate r (the maximum rate that individuals can reproduce in optimal circumstances) limited by a carrying capacity c (the population size for which the density limited *per capita* birth rate reaches zero – note this is not necessarily the same as the population equilibrium, because mortality, including that induced by disease and population reduction, will prevent the population from attaining this maximum). In the stochastic model at equilibrium and in the absence of disease and culling, the density-dependent birth rate (averaged over sufficiently long time scales) will match the death rate and the population will fluctuate around the disease free (DF) equilibrium population size of $N^*_{\mathrm{DF}} = c(r-\mu)/r$. The size of the demographic fluctuations is determined by the ratio $(r-\mu)/r$, so the meaning of the parameter r is best understood in terms of its effect on density-dependent regulation of the population size; for a given μ and $r > \mu$, increasing r will lead to smaller stochastic fluctuations as the population gets closer to its carrying capacity c.

Infection is modelled with an SI framework. Hosts are either susceptible to the disease (S), or infected (I), without the possibility of recovery, so that the total population size in group i at any one time is $N_i(t) = S_i(t) + I_i(t)$. For infected individuals, excess disease-induced mortality occurs at rate ν, i.e. in addition to the natural mortality at rate μ. Disease transmission is density-dependent within groups, and occurs at rate βSI, where β is the horizontal transmission coefficient.

We used a von Neumann neighbourhood to simulate movement occurring between neighbouring groups along adjoining edges of a square lattice. Each sub-population is connected with neighbouring groups via dispersal at an intrinsic rate κ. The population boundaries are periodic (i.e. opposite edges of the lattice connect to each other, allowing movement across the borders), in order to remove boundary effects. Robustness testing showed that our findings were qualitatively robust to the way that spatial relationships were explored. For example, the results were not sufficiently different if a negative exponential dispersal kernel was used, where dispersal was possible between any two groups but more likely for nearby groups.

Dispersal rates are dependent on the density of the target group, and entry is only possible into a group that is below a certain threshold proportion α of the DF equilibrium size, N^*_{DF}. The density dependence function that gives this behaviour is

$$f(N_j) = \begin{cases} 1 & \text{if } N_j \leq \alpha N^*_{\mathrm{DF}} \\ 0 & \text{if } N_j > \alpha N^*_{\mathrm{DF}} \end{cases} \quad (1)$$

where $N_j(t)$ is the size of the neighbouring group, indexed by j. Therefore, dispersal of susceptible and infected individuals between groups occurs at rates $\kappa z S_i f(N_j)$ and $\kappa z I_i f(N_j)$ respectively, where κ is the intrinsic dispersal rate, and $z = 1/4$ is the reciprocal of the number of neighbouring groups and is used to normalise dispersal rates.

A core set of model parameter values are chosen which reflect the badger-bTB system. These parameter values are detailed in Table 2 (for additional details on parameterisation see [33]). These parameter values capture key demographics of badger populations, with an average group size of 18 [38] and individual lifespan of 10 years [39]. Badger territories are actively defended, and in the absence of culling population size within social groups fluctuates around a stable equilibrium with very low levels of dispersal between groups, which is typical of high-density badger populations [38, 40, 41]. Disease is relatively stable once established in a given social group and therefore the disease is patchily distributed and stable across groups, as is observed in bTB in badger metapopulations [40, 42–44]. Density-dependent movement has been widely observed in badger populations [40, 45], and increased movement (social perturbation) has been observed following population reduction [13, 40, 46]. Our parameterisation captures this density-dependent dispersal. To explore the robustness of our findings across a broad set of wildlife-disease systems, we also conduct a sensitivity analysis around this core parameterisation by varying each parameter in turn, across a wide range.

In addition to the state variables in Table 1, the mean number of infective individuals per group $I(t)$ and proportion of infected groups $G(t)$ at time t are also tracked in these simulations. The effective dispersal rate is the average *per capita* host dispersal rate across all groups. This is measured directly from simulation, and is indicative of the total level of dispersal occurring within the population. The effective

Table 2: Summary of parameters, symbols, and default values used. Values provided are either annual rates or dimensionless.

Parameter	Symbol	Value
Intrinsic reproduction rate	r	1
Carrying capacity	c	20
Natural mortality rate	μ	0.1
Disease-induced mortality rate	ν	0.1
Within-group transmission coefficient	β	0.1
Intrinsic dispersal rate	κ	1.0
Dispersal threshold	α	0.7
Disease free equilibrium	N^*_{DF}	18
Population reduction rate		
coverage	p_1	1
within-group removal rate	p_2	0.7

transmission rate is given by

$$\beta_{\text{eff}} = \frac{\sum_i \beta S_i I_i}{\sum_i S_i \sum_i I_i}$$

which indicates the average risk of infection to each susceptible. It is also the effective contact rate of the disease dynamics in the analogous non-spatial model that assumes complete mixing. In Prentice *et al.* [33], it was shown that β_{eff} increases under culling when the proportion of groups containing infected individuals, G, increases (because more susceptibles are exposed to infection), but returned to expected values after culling stopped. A high β_{eff} during population reduction demonstrates higher than expected disease transmission, and is therefore indicative of the perturbation effect.

2.2 Quantifying the perturbation effect

We define the perturbation effect as the increase in disease levels following control (population reduction), compared to disease levels if no control had been carried out. We explore two measures of disease levels; the number of infective individuals, and the number of infected groups.

The size of the perturbation effect is defined as

$$\Pi(t;p) = I(t;p) - I(t;0)$$

where p represents the average rate of population reduction under a given culling strategy, and t is the time. A similar measure, based on the proportion of groups containing infectives $G(t)$, is

$$\Pi_{\text{groups}}(t;p) = G(t;p) - G(t;0)$$

Π_{groups} is useful because it reflects the geographical spread of disease; an increase in G is typically a precursor to an increase in the global number of infectives when the sub-populations return to equilibrium (see [33]).

Presenting the changes in number of infectives rather than the change in prevalence provides the most conservative measure of changing disease levels. As culling decreases the overall population size, an increase in prevalence could conceal an overall reduction in the number of infectives, overestimating changes to average risk of infection.

2.3 Implementation of population reduction strategies and their spatial design

In the implementation of population reduction schemes, there is considerable variation in the proportion of land that is accessible, and the rate of removal within targeted groups. The dynamic spatially explicit model used in this paper allows these elements of wildlife culling to be explored.

Culling may be applied to any subset of groups (corresponding to variation in the proportion of accessible land), and with any within-group removal rate within the selected groups (corresponding to variation in the level of population reduction achievable). Here we use two parameters to characterise the population reduction strategy:

1. Coverage $p_1 \in [0,1]$ is the proportion of targeted groups in which culling takes place. The higher the value of p_1, the higher the spatial coverage of culling effort.
2. Within-group removal rate p_2 models culling as an additional mortality and represents the rate at which individuals are removed from targeted groups.

Individuals are removed continuously by adding, for the duration of the cull within a targeted group, the effect of culling to the natural mortality rate in targeted groups, i.e. the mortality rate in such groups becomes $\mu + p_2$. When the coverage is incomplete (i.e. $p_1 < 1$) then targeted groups are chosen randomly with probability p_1. After a period of time, a switch event is triggered, the mortality rates in targeted groups are returned to normal, and a new set of groups is targeted. The time between such events is referred to here as the 'switch time'. By default, the switch time is 1 year. In the simulations shown below, the model is initialised at $t = -5$ and the metapopulation is allowed to stabilise prior to the onset of culling at $t = 0$. Where a single snapshot is used the population is measured at time $t = 20$.

The specified coverage is obtained by randomly selecting groups to target. The within-group removal is the same throughout all targeted groups. Therefore, we measure the overall culling effort by

$p = p_1 \times p2$. Within such a design, it is possible to obtain the same overall culling effort for different combinations of p_1 and p_2 by keeping p fixed and choosing, for example, $p_1 = p/p_2$ (e.g. culling is evenly distributed across all sites when $p_1 = 1.0$, $p_2 = 0.2$, or has lower spatial coverage (greater spatial heterogeneity) when $p_1 = 0.2$, $p_2 = 1.0$, but both strategies have the same overall culling rate of $p = 0.2$). Therefore, assuming the effort and cost required depends only on p, it is possible to evaluate the results of different strategies for any given level of resources (total culling effort).

3 Results

Culling can be an effective form of disease control. Fig. 1 shows the behaviour of an infected population before, during, and after implementation of population reduction. A culling effort of $p = 0.7$ was spread uniformly across all groups. This level of effort reduces the population to around 50% of pre-cull levels after 1 year and to around 30% (i.e. a 70% reduction) after 3 years. All other parameter values are as detailed in Table 2. Groups are classed as disease-free or infected, with G representing the proportion of infected groups, and I the mean number of infective individuals per group. During the culling period, the level of disease within infected groups typically decreases; however, population reduction disrupts the existing demographic structure, leading to an increase in dispersal. This increased movement between groups leads to introduction of infection into previously uninfected groups, and a short-term increase in the number of infected groups. Continued culling then leads to a decline in both the number of infected individuals and groups, with disease levels being lowest at the end of the culling period. When population reduction ceases, there is a small increase in the number of infected groups, and disease levels in the newly infected groups increases towards an equilibrium level.

Fig. 1 demonstrates that for the chosen parameter values, a culling effort of $p = 0.7$ carried out over 10 years, where all groups are targeted, can result in a decline in the levels of disease. However, it is not always possible to cull evenly across all animal territories within an area.

Fig. 2 illustrates how the spatial coverage of a culling regime can influence whether disease levels rise or fall for a set culling effort ($p = 0.7$). Fig. 2A–D show the temporal evolution of disease levels, for varying levels of spatial coverage, i.e. varying p_1 and p_2, for a set culling effort ($p_1 \times p_2 = 0.7$). For clarity this figure includes a repeat of Fig. 1, showing uniform culling. A spatially heterogeneous culling design with high levels of culling targeted at a small proportion of sites (Fig. 2A), results in a dramatic increase in disease levels both during and after the cull. This approach creates a large vacuum effect in targeted areas and a cascade of movement from surrounding groups. Increased movement between groups leads to introduction of infection into uninfected groups, subsequently increasing rates of effective horizontal disease transmission β_{eff} within groups. When population reduction ceases, the typical prevalence in the newly infected groups increases towards the equilibrium level, which leads to an increase in the total number of infectives as there are now more infected groups. This increase in number of infected groups and individuals following implementation of population reduction represents a perturbation effect.

3.1 Effects of removal rate and spatial coverage on culling outcomes

The outcome of population reduction is influenced by the cull coverage, i.e. the proportion of groups in which culling takes place (p_1), and the within-group removal rate (p_2). Fig. 3 shows the size of the perturbation effect, measured with both Π_{groups} and with Π (for definitions, see Section 2). Hence, positive values represent scenarios where a perturbation effect occurs according to the measures Π_{groups} and Π, respectively. An increase in Π_{groups} corresponds to spreading the infection spatially, potentially increasing disease risk to sympatric species.

Fig. 3A,B has lines of constant total culling effort (p) overlaid for clarity. Although the lines of constant p initially seem to closely follow the contours of the plot, Fig. 3A,B highlights that the lines of constant p can actually intersect the different contours (representing different levels of perturbation). This is a pivotal result. If the contours of the plot did in fact follow the lines of constant p then varying the spatial design of culling would not affect the outcome for disease control. However, Fig. 3 reveals that the spatial design of culling strategies impacts on the size of the perturbation effect (reiterating the findings shown in Fig. 2). The outcome of culling depends on both the proportion of groups targeted (p_1) and the removal rate within each targeted group (p_2) and cannot be predicted purely by overall culling effort, p (red contour lines, Fig. 3A,B). Levels of culling effort below $p = 0.6$ are likely to result in a perturbation effect, regardless of the proportion of groups to which this is applied. Only for sufficiently high effort levels is it possible for culling to have a positive impact in the system studied here, and in these cases increased spatial coverage of effort gives the best results.

For a low population reduction effort ($p = 0.2$), the perturbation effect is maximised by an intermediate level of spatial coverage of culling (where 20–50% of the sites are targeted) (Fig. 3A,B). This intermediate maximum occurs as individual groups are targeted hard enough to reduce population size, so density-dependent dispersal increases, creating a vacuum effect and instigating a cascade of individuals drawn in from surrounding areas. On the other hand, individual groups are not targeted hard enough to make within-group disease extinction likely.

From Fig. 3A,B, it is not clear whether disease control is successful as a result of the reduction of disease in an extant population, or whether the disease was eliminated because all hosts were removed. For host species of conservation interest, complete removal is undesirable. Fig. 3C shows the population size following culling. Fig. 3D reveals three key areas: the green area where the perturbation effect occurs, the purple area, where the host population will likely go extinct (an average of fewer than 2 in-

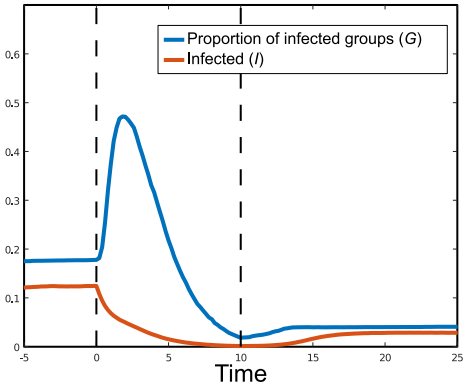

Figure 1: Disease dynamics before, during, and after population reduction, for parameter values given in Table 2, coverage $p_1 = 1.0$, removal rate $p_2 = 0.7$. The proportion of infected groups G (blue) and average number of infectives per group I, scaled by 20 (red).

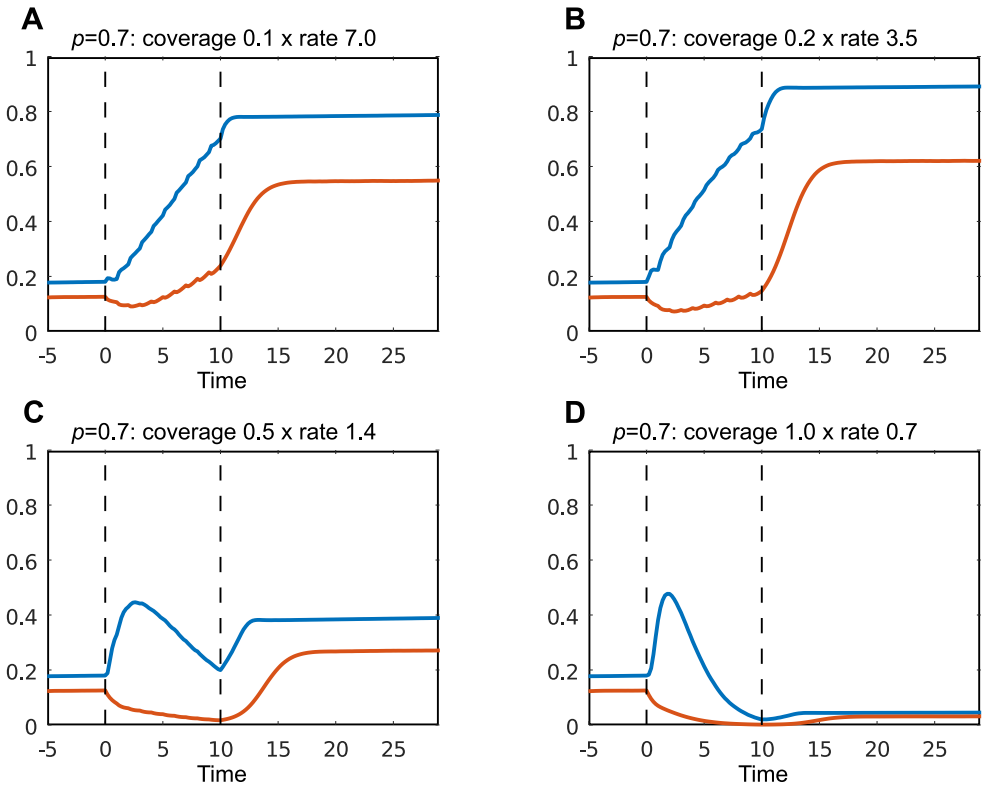

Figure 2: (**A**–**D**) Applying the same overall level of culling effort (constant $p = 0.7$), but varying the coverage of groups targeted (p_1) and removal rate (p_2). The proportion of infected groups G (blue) and average number of infectives per group I, scaled by 20 (red). The increases in G and I following spatially heterogeneous culling shows the size of the perturbation effect.

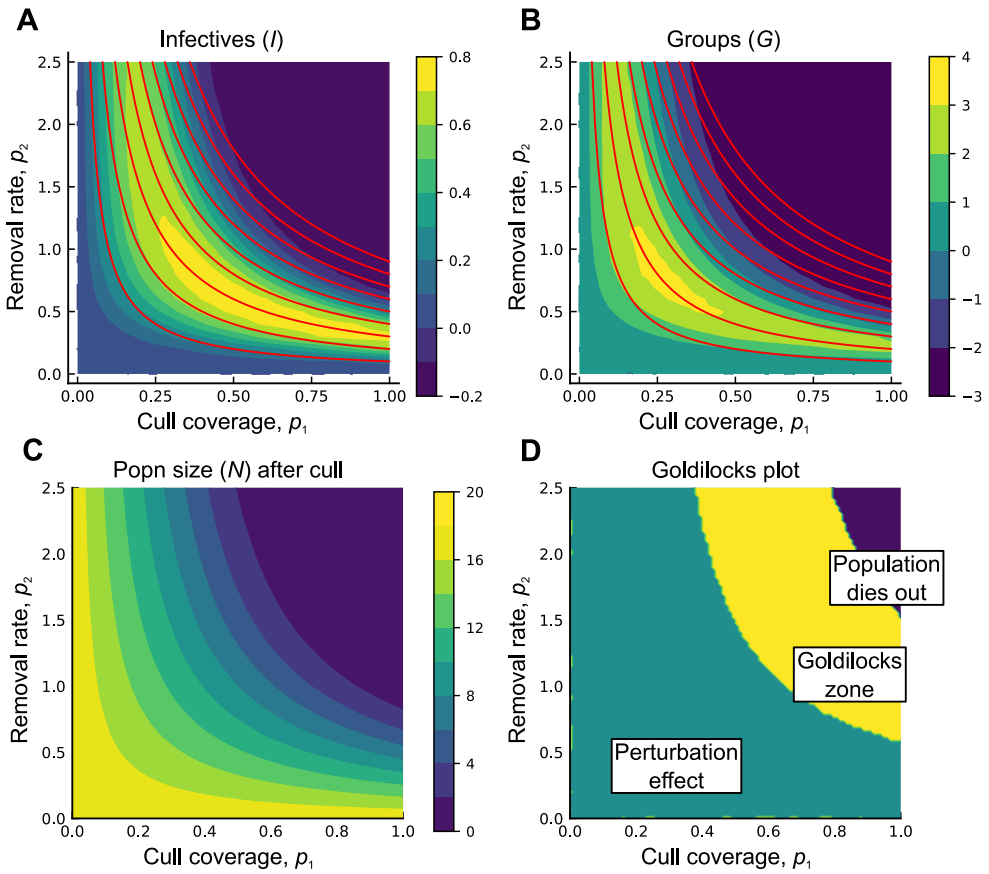

Figure 3: How total culling effort and the spatial distribution of culling effort affects both disease levels and population persistence. (**A,B**) the number of infective individuals ($\Pi(t)$) and infective groups ($G(t)$) for differing levels of coverage p_1, removal rate p_2, and with red lines of constant culling effort ($p = p_1 \times p_2$), from 0.1 to 0.9 in steps of 0.1 starting from the lower left. (**C**) Number of individuals per group ($N(t)$) following culling. (**D**) The outcomes of disease control for varying levels of p_1 and p_2, showing where culling results in increased disease levels (green), where culling leads to population extinction (purple), and the Goldilocks zone where culling leads to disease reduction without population extinction (yellow).

dividuals remaining in the entire metapopulation at $t = 20$), and the yellow 'Goldilocks zone' where conditions are just right [47], such that disease levels are reduced and the host population survives.

3.2 What affects the size of the 'Goldilocks Zone'?

The size of the Goldilocks zone is dependent on a number of key drivers, especially the nature of density-dependent dispersal operating in the focal wildlife species, and the level of disease-induced mortality. The sensitivity of our findings to variations in these parameters are shown in Fig. 4 and Fig. 5. Fig. 4 reveals that the threshold for density-dependent dispersal (α) has a substantial impact on the effectiveness of culling-based disease management. In Fig. 4B–D prior to culling, there is little contact between groups and disease is distributed patchily across the groups. The disease has space to expand and is able to do so when culling leads to an increase in density-dependent dispersal. During culling interventions, if high levels of culling are required before movement levels increase (low α), then culling can be effective for reducing disease levels for a wide range of effort levels and spatial designs (Fig. 4A). However, as α increases and population reduction leads to ever-increasing levels of dispersal, the range of scenarios for which culling leads to disease reduction are increasingly restricted (medium α, Fig. 4B,C). By contrast, Fig. 4E shows that all culling strategies can lead to disease reduction in scenarios where there is already substantial dispersal prior to culling. This is because, in such cases, the disease is already widespread across all groups and there is no scope for a perturbation effect.

The level of disease-induced host mortality also has a substantial effect on the potential outcomes of population reduction (Fig. 5). High disease-induced mortality selectively removes infective individuals. This selective removal of infective individuals increases the area of the Goldilocks zone, as culling becomes an increasingly effective form of disease control for a wider range of culling effort across a range of spatial designs.

The sensitivity of the results to birth rate r, carrying capacity c, natural mortality μ and within-group transmission β were also explored (see electronic supplementary material). The areas of the Goldilocks zone, perturbation zone and population extinction zone do not vary significantly across the parameter ranges explored. This demonstrates that the results are robust across a range of parameter values, and the Goldilocks zone is likely to be present for a wide range of systems.

3.3 The importance of cull duration

The duration of population reduction has an impact on its success for disease control. For high levels of sustained culling effort targeted across a large number of groups, disease levels can be reduced (Fig. 1). However, in the early stages of control, the number of infected groups increases due to increased dispersal. If the same culling strategy was instead applied for only 3 years, there would be an overall increase in disease levels (Fig. 6). This emphasises the importance of carrying out culling over appropriate timescales.

Fig. 6 also highlights the importance of measuring the success of a culling effort over the right timescale. The number of infectives is low at the point where culling ceases ($t = 3$). Sampling the population immediately on ending a cull could indicate that the strategy has been a success. However, the number of infected groups is higher than when culling began and when the new equilibrium is reached the overall levels of disease are higher after culling has ceased.

The minimum cull duration required for effective disease control is dependent on the spatial coverage of the control programme (Fig. 7). For a constant culling effort ($p = 0.7$), as the proportion of sites targeted decreases, the necessary duration of the cull increases. If only a low proportion of sites can be targeted, then culling may never be a viable option for disease control regardless of cull duration (Fig. 7).

4 Discussion

Failures or unanticipated consequences of disease control via population reduction in wildlife are widely reported [4, 5, 9, 13, 16, 48]. However, such effects are poorly understood and even within a given host-pathogen system (e.g. badgers and *M. bovis* (bTB)) the perturbation effect is found in some culling situations, but not others. For example, culling badgers has been associated with an overall increase [8, 17] and decrease [21] in bTB prevalence in different real-world systems. Our results provide potential process-based explanations for such observed differences in outcomes of different culling exercises. We found that the rate of population reduction, the proportion of the groups targeted, and the duration of the cull, can all be critical in determining the outcome of disease control efforts. With sustained high levels of population reduction across a sufficient number of sites, disease levels can be reduced. However, if high levels of population reduction effort are not applied, too few sites are targeted, or the cull duration is too short, then a perturbation effect is observed and disease levels increase.

Here we show that perturbation effects may arise from the mechanism of density-dependent dispersal (Fig. 2) in systems where transmission between undisturbed groups is limited and the disease is spatially heterogeneous. In such circumstances culling in selected groups leads to a 'vacuum effect', where the decrease in population density enables the influx of both susceptible and infectious individuals into targeted groups. The characteristics of density-dependent dispersal driven perturbation effects explored in this paper are broadly consistent with the effects of culling observed in the badger-TB system including: under low level heterogeneous (e.g. reactive) culling [16, 49] for uniform high removal rate culls (e.g. as conducted in Ireland [21]); and when the duration of the cull is insufficient [18].

Our results show that culling can be an effective form of disease control. Figure 1 shows a culling design based on a high removal rate ($p_2 = 0.7$) applied uniformly ($p_1 = 1$) (which would remove about

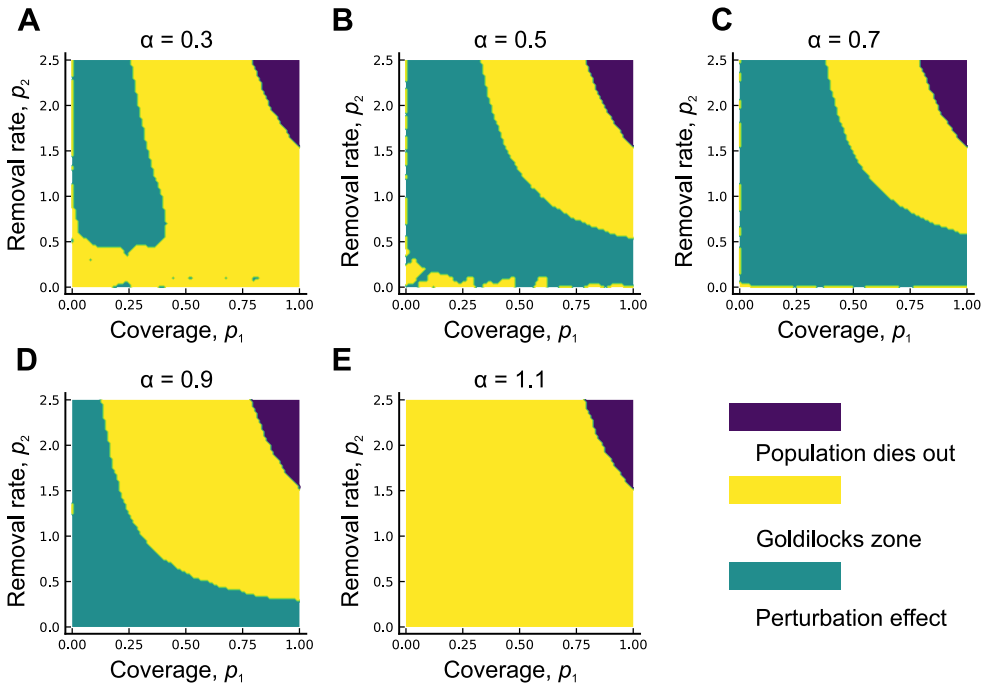

Figure 4: (**A**–**E**) The impact of density-dependent dispersal (α) on the Goldilocks zone. How total culling effort and the spatial distribution of culling effort affects population survival and disease eradication for host populations with varying levels of density-dependent dispersal (α).

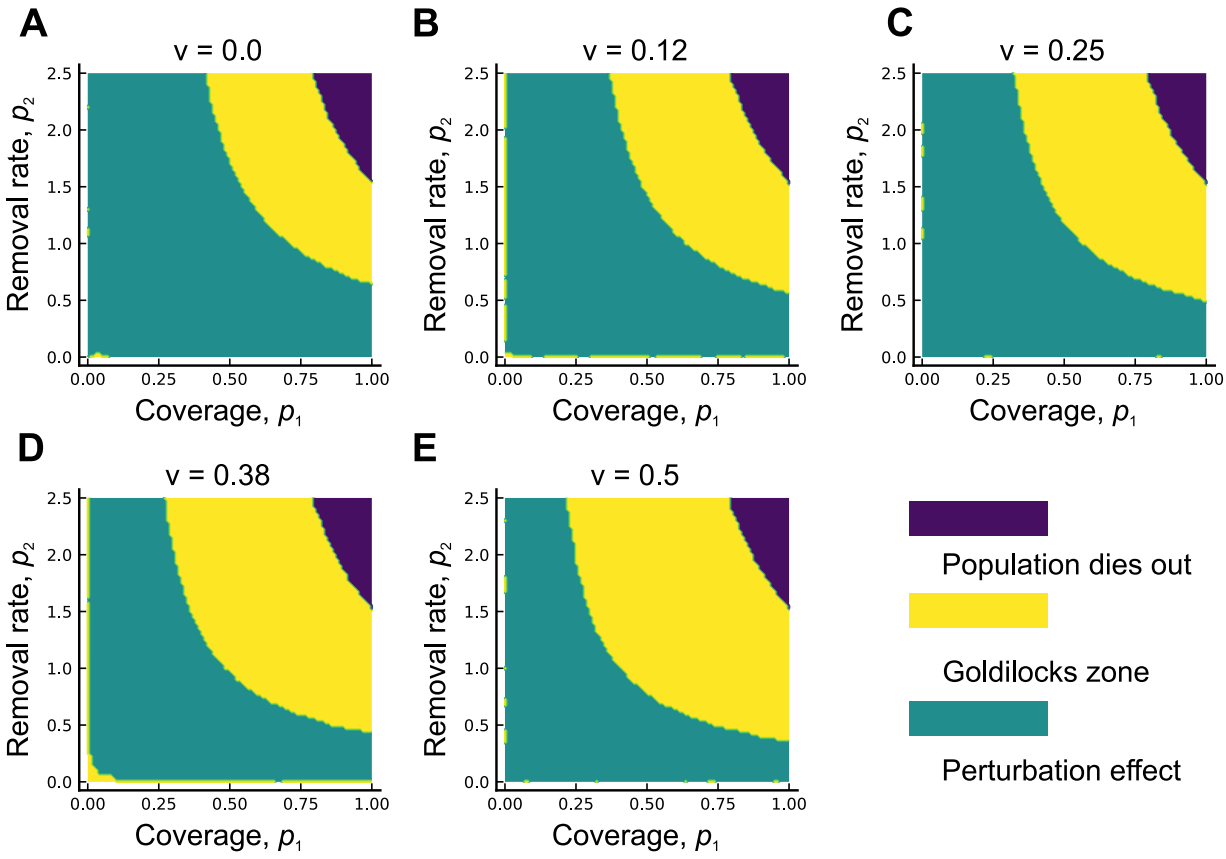

Figure 5: (**A**–**E**) The impact of disease-induced mortality (ν) on the Goldilocks zone. How total culling effort and the spatial distribution of culling effort affects population survival and disease eradication for host populations with varying rates of disease-induced mortality (ν).

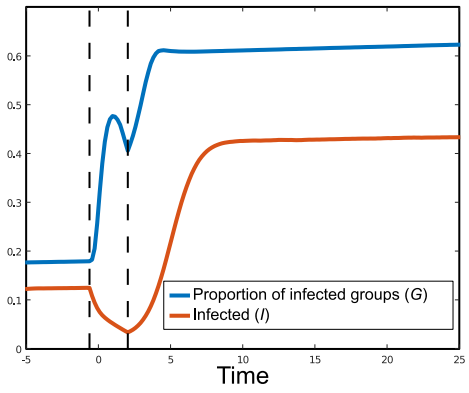

Figure 6: Disease dynamics for a 3 year cull. Parameter values given in Table 2, coverage $p_1 = 1.0$, removal rate $p_2 = 0.7$ (the same as for Fig. 1), however culling is suspended after 3 years. The proportion of infected groups G (blue) and average number of infectives per group I, scaled by 20 (red).

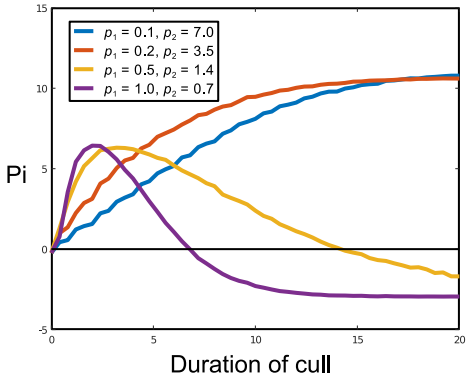

Figure 7: The impact of spatial coverage and duration of cull (t_1) on mean numbers of infectives per group. The number of infectives per group 10 years after culling has ceased ($\Pi(t_1 + 10)$), for $p = 0.7$ and varying levels of spatial heterogeneity in culling.

50% of the pre-cull population within the first year and over a 3 year period reduces the population by 70%) and carried out over an extended period, is sufficient to reduce the number of infected individuals and groups. This echoes the findings of a proactive badger culling campaign in Ireland, where bTB outbreaks in sympatric cattle were lower than in control regions of low-level reactive culling [21]. A number of factors consistent with our analysis could have contributed to the successful outcome of badger culling in Ireland. Firstly, wire restraints were used for badger trapping which is an efficient approach to badger removal (suggestive of high removal rates). Secondly, there was spatial homogeneity in culling implementation within the core areas, with permission for removal refused at only 0.42% of surveyed badger setts [21] suggesting $p_1 \approx 1$. Thirdly, the cull was carried out over a five-year period. Thus, success of this long-term, spatially homogeneous, high-intensity cull is consistent with our results.

The importance of a sustained high culling effort is reflected in policy guidelines, with the current policy for culling of badgers for bTB control in the UK stating that 70% of the badger population should be removed in target areas during the first year of operations and that culling should be sustained for at least 4 years [50]. However, it is not always possible to achieve such levels of population reduction. For example, efforts to remove 70% of the badger population in pilot culls in Somerset and Gloucestershire, UK, resulted in less than 48% and 39% of the respective pre-cull populations being removed by controlled cage trapping and shooting [51]. Our findings indicate that low levels of population reduction are unlikely to be sufficient for disease control.

Although our findings suggest that high levels of culling could be sufficient for disease control in the system modelled here, this success is dependent on uniform culling effort being applied equally across all groups (Fig. 2). Although the UK bTB policy states that limited land should be inaccessible for culling [50], there will be spatial variation in culling success due to variations in landowner permissions, site accessibility, and local knowledge of territory use. Sufficient resultant spatial heterogeneity in culling effort can lead to an increase in disease levels (Fig. 2, Fig. 3A,B). Model based analysis shows that the outcome of a cull is dependent on both the within-group removal rate and the proportion of groups targeted. At low levels of total culling effort (e.g. $p = 0.2$) the size of the perturbation effect is greatest for intermediate levels of spatial heterogeneity (where around 30% of the sites are targeted). This suggests that random culling targeted at a small number of groups could lead to the greatest rise in disease levels consistent with the results of the RBCT [49]. In such situations, a homogeneous culling design would be less deleterious. However, if only low levels of population reduction are achieved, then disease levels are likely to rise irrespective of the proportion of groups targeted.

In addition to considering spatial distribution of effort, culling design should also be considered across appropriate timescales. For culling strategies where successful disease control can be achieved (e.g. Fig. 1) there is an initial spike in dispersal leading to a temporary increase in the number of infected groups. If population reduction is stopped early (for example in response to such an increase in disease distribution), the equilibrium level of disease prevalence would be restored in these newly infected groups, leading to a perturbation effect and an overall increase in disease levels (Fig. 6). It is only by persisting with control over an appropriate time interval that overall disease reduction is achieved. The amount of time that a cull needs to be implemented for in order to successfully decrease disease levels is affected by the proportion of groups that are targeted. For a given culling effort, as the proportion of the sites targeted decreases, there is an increase in the amount of time needed to reduce disease levels (Fig. 7). If large areas of the land are inaccessible, culling is unlikely to be successful regardless of time frame.

The need for a minimum cull duration is acknowledged in the UK bTB control policy, which stipulates a commitment to four years of culling. Our findings on cull duration also qualitatively reflect those seen in the RBCT, where badger culling had a detrimental net effect on bTB levels between the first and second (approximately annual) culls, and a beneficial effect after the fourth year of culling [18]. Although our results qualitatively echo those observed in the field, they do not quantitatively match the timelines observed. This will be due in part to our parameterisations not perfectly capturing this specific system. There will also be confounding factors that affect the cull duration required to reduce disease levels. For example, Brunton *et al.* [52] found that badger culling led to a reduction in bTB levels (in sympatric cattle) after two years of culling, but partly attributed this to the implementation of an additional risk management programme in the second year of disease control.

Although disease reduction is maximised by high levels of population reduction over sufficiently long timescales, if this is achieved it can drive population extinction (Fig. 3C). This is an unwelcome consequence for many wildlife species, especially those of conservation concern. For example, the UK policy for badger culling states that cull efforts should ensure that a viable population remains [50]. We have identified Goldilocks zones (regions of culling-programme design space) of successful disease control, where culling effort is sufficient to reduce disease levels, whilst not being so high that it drives the population to extinction. The presence of the Goldilocks zone echoes empirical observations that population reduction can be used successfully to control disease in wildlife systems [53]. We have found this Goldilocks optimal culling zone to be robust to variations in parameterisation, suggesting that this finding is applicable to a range of disease systems (Fig. 4, Fig. 5, and supplementary material).

The position and breadth of this optimal culling zone is most strongly influenced by host dispersal (Fig. 4) and disease-induced mortality (Fig. 5). Distinct group structure and density-dependent dispersal are critical aspects of the model structure, and the tendency of a particular population to disperse in response to lower populations at neighbouring sites, represented here by the parameter α (dispersal threshold), has a significant impact on whether pop-

ulation reduction is a feasible form of disease control. In systems where culling leads to high levels of social perturbation, population reduction will likely lead to a perturbation effect or host extinction (Fig. 4B,C). The breadth of the Goldilocks zone is also influenced by the level of disease-induced mortality. The higher the rate of disease-induced mortality, the more opportunity for successful disease control through population reduction (Fig. 5). This is due to infected individuals being selectively removed and the population having a lower equilibrium disease prevalence. The Goldilocks zone is most narrow for systems with low disease-induced mortality, as there is limited selective removal of infected individuals. This highlights the difficulties faced when trying to control disease in asymptomatic reservoir hosts. The recognised difficulties in controlling *Mycobacterium avium* ssp. *paratuberculosis* in asymptomatic rabbit hosts [54, 55] supports this finding. There is also evidence that bTB and badgers represents a system where the Goldilocks zone is slim. At high densities, and in the absence of culling, badgers form stable populations with limited movement [39, 41, 42]. However population reduction leads to social perturbation and an increase in dispersal [13, 40, 54]. This limits the options for successful disease control.

In summary, variations in the spatial and temporal aspects of culling implementation could drive the different outcomes of culling seen within host-pathogen systems. The success or failure of disease control can hinge on the rate of population reduction, the proportion of the metapopulation targeted, and the duration of the cull. Although disease reduction is possible, our results indicate that implementation of uninformed population reduction could have a more detrimental outcome than if no action was taken. It is imperative to consider the rate of within-group removal that is achievable, the proportion of land accessible, and the expected cull duration given the resources available. It is only by appropriate modelling of the target species, paying particular attention to social structure and density-dependent dispersal, that control strategies can be tuned to achieve disease reduction without population elimination. In scenarios where hosts live in distinct groups, have density-dependent dispersal and low disease-induced mortality, there is only a restricted combination of culling intensity, coverage and duration where there is a good likelihood of success; culling that fails to meet these criteria is likely to have adverse consequences for disease spread.

Data accessibility

All code required to run the model is available at https://github.com/spaceLem/badgers, including instructions on how to compile and use it.

Author contributions

The work was planned by JCP, NJF, MRH, PCLW, RD, and GM. All code was written by and simulations were performed by JCP. The manuscript was prepared by JCP, NJF, MRH, PCLW, RD, and GM. All authors gave final approval for publication.

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