Exploring the Efficiency of Badger Culling in Preventing the Spread of Bovine Tuberculosis in the UK

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Abstract

In the UK, incidence of Bovine Tuberculosis (bTB) in cattle has been found to be correlated with incidence of the disease in badgers (*Meles meles*). Different methods of badger culling have been employed for the control of bTB, but disease incidence has continued to increase. Social perturbation of badger populations in response to culling has been proposed as an explanation for the failure of culling to consistently demonstrate significant reductions in bTB in cattle. Field studies indicate that culling disrupts badger social structure, leading them to behave in a manner that increases contact rates and hence disease transmission. This paper will demonstrate that culling is indeed likely to increase disease incidence and that this is largely due to social perturbation. This is consistent with the results from other studies including a recent large-scale field trial. After discussing the effects of social perturbation and results from other studies, I conclude that badger culling is not an efficient method of controlling the spread of bTB in the UK, and more research should be carried out looking for alternative control strategies.

1 Introduction

Bovine Tuberculosis (bTB), caused by the bacterium *Mycrobacterium bovis*, is a serious infectious disease that affects cattle [2]. Regular testing of cattle for bTB and subsequent slaughter of individuals found positive has been an effective way of controlling the disease[5]. However, this method of control becomes more complicated when wildlife is acting as a reservoir for the disease, thereby re-infecting cattle. This is the case in the UK where badgers have been found to be the main causative agent of the disease [10]. It follows that the badger population must also be culled, but this has been a controversial matter, particularly because badgers have been statutorily protected in Britain since 1973 [9]. Various methods for badger culling have been employed in various studies, but disease levels have only kept increasing, particularly in the Southwest of England [3].
Population reduction is the most commonly employed strategy used to control wildlife [16] with the aim of reducing the total population and the number of individuals infected in order to reduce transmission rates, disease prevalence and risk to other populations [12]. This method is supported by the theoretical evidence of a persistence threshold, a population threshold below which the infection would be unable to persist and die out [1]. The assumption here is that the transmission rates vary positively with population abundance, so that reducing population numbers will reduce transmission rates [3]. However, there is actually little evidence for a persistence threshold in wildlife populations [11], and there is evidence that the relationship between host abundance and transmission rate

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is non-linear [13]. For example the Randomised Badger Control Trial (RBCT)[2] showed that reactive culling of badgers in response to a confirmed bovine tuberculosis herd breakdown in cattle, was associated with a 27% increase in the incidence of confirmed breakdowns, relative to control trials. A herd is said to experience a bTB "breakdown" if one or more members of a cattle herd fail the conventional TB skin test or show evidence of TB lesions at slaughterhouse inspection [4].

This counterintuitive trend whereby culling is increasing the incidence of disease has been termed the "perturbation effect," as it is believed to depend on social perturbation. Carter's 2007 paper [3] defines social perturbation as a substantial disruption in social structure of host populations in response to culling, and this is known to have serious implications for disease persistence and prevalence. In the same paper he outlines how there are three broad processes by which this may occur: the vacuum effect, territorial disruption and increased ranging behaviour. The vacuum effect is the tendency for individuals neighbouring a culled area to disperse inwards to seek new home ranges. Territorial disruption is a breakdown of the discrete pattern of group territories typical of medium-to-high-density badger populations, and may be accompanied by an increase in the frequency of movements of individuals between social groups. An increased ranging behaviour is an increase in how far or often an individual will travel. Any of these processes may increase contact rates between badgers, potentially increasing disease transmission in the population and the likelihood that infected animals become infectious through stress-induced immunosuppression [9]. Both phenomena could lead to an immediate increase in the risk of infection in cattle [3].

In this paper I will be exploring how badger culling affects disease incidence by replicating a model to show how varying levels of social perturbation affect the number of infectious badgers. I will also investigate the suitability of this simple model for evaluating the effects of culling.

I chose to reproduce this simple non-spatial model because an investigation like this includes many parameters the values of which are not always certain. This is even more true of more complicated spatial models, where the number and uncertainty of the parameters is greater, thereby reducing the credibility of the results. Instead a simpler model can be used to gain a better understanding of the general behaviours and interactions between variables, which can then be compared to those seen in the field. From this simple model one can also identify more easily which aspects of the model need improving or further investigation. It is nevertheless necessary to have a simple model on which to construct more complex ones. By comparing the results derived from the different models I will be able to better determine whether the simple model is a viable method of analysing whether culling will indeed result in an increase of disease prevalence.

2 Methods

The model I will be replicating was derived from Prentice's 2014 paper [12]. As outlined in the paper, the model will examine a generic single-pathogen wildlife disease system with a fluctuating host population. The number of susceptible and infected individuals in the population at time t are $S(t)$ and $I(t)$ respectively, and the total population size is given by $N(t) = S(t)+I(t)$. It assumes density dependent (logistic) growth, with intrinsic reproduction rate r, limited by a carrying capacity c. Natural mortality occurs at a constant per-capita rate of d, while disease induced mortality occurs occurs at a constant per-capita rate of e. The rate of infection is a combination of susceptibility and contact rates between susceptible and infective individuals and here density-dependent infection is being considered (as is the case for bTB in badgers) with horizontal transmission rate β (that is, transmission by any means excluding that of mother-to-offspring).

The population reduction is an additional con-

stant per-capita death rate p which applies to the entire population. To include social perturbation in response to culling, the horizontal transmission rate was modelled as $\beta + kp$ where $k > 0$ represents any mechanism or combination of mechanisms that lead to an increase in contact rate or susceptibility. The formulation of a simple non-spatial deterministic model that encapsulates the above assumptions can be found in the appendix of Prentice's 2014 paper[12]. The rescaled deterministic ordinary differential equations (ODEs) that combine the demography and disease dynamics described above with population reduction and a corresponding enhanced transmission resulting from explicit behavioural and implicit ecological (system) responses are given by:

$$\frac{dS}{dt} = N(1-N) - (d+p)S - (\beta + kp)SI \quad (1)$$

$$\frac{dI}{dT} = -(d+e+p)I + (\beta + kp)SI \quad (2)$$

These are the final equations that will be used to model the change in number of infected individuals over time for varying values of disease enhancement values k. This will occur within an emergent disease situation (starting near the disease-free equilibrium [12]), which is why the initial number of infected animals is so low (See Fig.1), and at a constant culling rate of p (constant culling is often referred to as proactive culling[2]).

The equations were solved numerically using the odeint function of scipy, an adaptive step size Runge-Kutta algorithm [8]. The parameters used were: reproduction rate $r = 1$, carrying capacity $c = 1$, natural mortality rate $d = 0.2$, disease induced mortality rate $e = 0.1$, horizontal transmission rate $\beta = 0.5$. For the graph on the right in Fig.1, there was no culling culling for the first 5 years, so when $t < 5$the population reduction rate $p = 0$, number of infectives $I = 0.05$, number of susceptibles $S = 0.55$ and total population $N = 0.6$. After the 5 years, culling began, so when $t > 5$, $p = 0.2$, $I = 0.05$, $S = 0.828$ and $N = 0.778$.

3 Results

I was mostly successful in reproducing the same graph from the Prentice 2014 paper [12] found in Fig.1. An extra value for k, found by trial and error was added to the reproduced graph in order to investigate which approximate value of k would determine the boundary above which culling becomes inefficient.

In Fig.2, the number of infected individuals rises above the control group when $k \geq 4.1$. This demonstrates that when the disease enhancement level is above a certain threshold (in this case 4.1), culling becomes counterproductive and we can observe the perturbation effect occurs. It appears that what determines the efficiency of the cull is the disease enhancement value or level of Social Perturbation that arises in response to the cull. It stands to reason that if the efficiency of a cull were known, one could potentially derive a disease enhancement value. If we were then able to calculate this value independently from the cull, we could derive disease enhancement values for different scenarios and predict what level of social perturbation a cull would result in.

Overall, the graph appears to agree with what was mentioned earlier about how culling can lead to the perturbation effect and increase rather than decrease the level of infection.

3.1 Limitations

In attempting to reproduce the graph, some constraints came to light. The graph depended on sensitive parameters, such that there was no room for much variation in the values for the initial condition when attempting to obtain the same effect. Values used and explanations for that choice were not clear in the original paper [12], therefore my initial conditions were derived by trial and error which accounts for the discrepancies between the graph in Fig.1 and that in Fig.2. It follows that the results may not be as compelling or oust as they might appear at first. The model reproduced is a general model for the average wildlife disease where infection

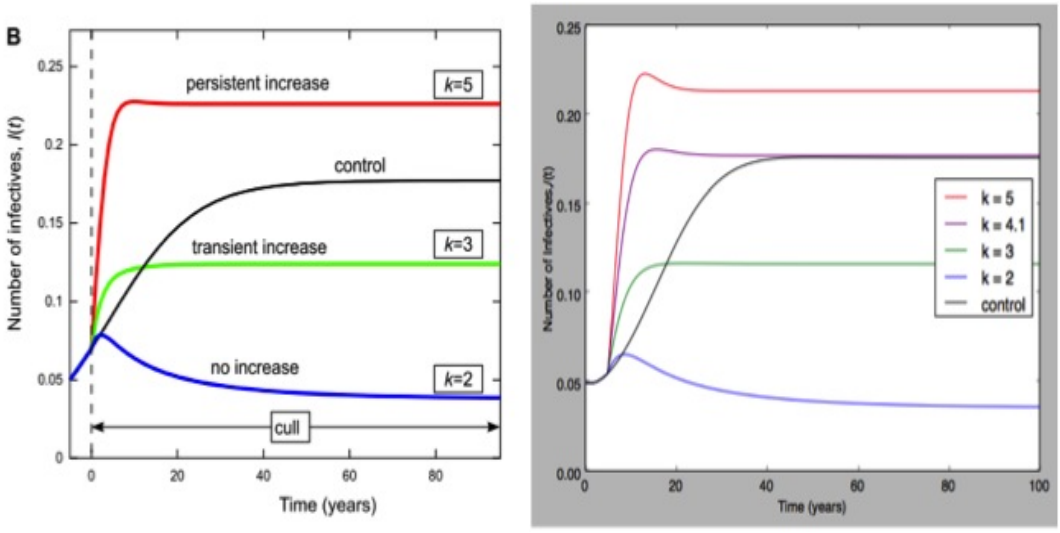

Figure 1: On the left, original graph from Prentice's 2014 paper. On the right, my reproduction of the graph on the left. The change in infectives is being modelled over time for varying disease enhancement values k. The dotted line represents the time when culling started. For $t < 5$ population reduction $p = 0$, for $t > 5$ population reduction $p = 0.2$. In this situation culling is a continuous process (proactive) occurring in an emergent disease situation (starting near the disease-free equilibrium). $k = 4.1$ represents the boundary above which the perturbation effect occurs [12]

is density dependent. For more case-specific results one would have to input more specific field data that I was unable to find.
The model is still a non-spatial model. Although it was chosen on purpose, it is still a limitation considering a major cause of the perturbation effect is dispersal, which could be better analysed with a spatial model. However k does try to simulate the effects of dispersal on the the number of infected individuals and plays a similar role as dispersal does in a spatial model [12].

4 Discussion

Based on a simple non-spatial deterministic model, the results presented in this paper show how culling can lead to an increase in disease incidence due to social disruption in the host population. Having evaluated some of the model's limitations, these results will now be compared with those of other more complex models and field studies.

Findings of a spatial model [12] show that there is no threshold below which the perturbation effect does not occur in populations where dispersal was density dependent. This would appear to contradict findings form the non-spatial model which in Fig.2 shows a threshold of $k < 4.1$. However the data from the spatial model might simply be showing that culling will always induce a disease endemic value of $k \geq 4.1$. That is to say, culling never a social perturbation small enough to prevent the perturbation effect. This is what most studies seem to be pointing towards [3][17][6].
The RBCT [2] found that reactive badger culling resulted in a 20% increase in bTB. In contrast, proactive culling induced a 23% reduction in bTB in the proactive area, but induced a 26% increase in the neighbouring area. Not only do these results show that culling induces the perturbation effect, but also how dispersal plays an important role in this. Therefore, the RBCT [2] also concludes that there are no culling methods that will induce no or a small enough level of so-

cial perturbation to prove an effective method of preventing the spread of bTB. We should therefor stop looking at badger culling as a solution for bTB control and start exploring alternative strategies.
Further work could consist on building a more complex spatial model that includes cattle herds and bTB infection in cattle to better analyse badger-to-cattle infection [13]. This can then be used to investigate methods of reducing infection.
More studies are also necessary into vaccination. Up until now studies show that vaccination is not as efficient as badger culling [14][15]. However these studies have not included social perturbation caused by badger culling into their studies. If the perturbation were added, perhaps the results would favour vaccination, which does not induce social perturbation.
Furthermore, it would be interesting to explore methods to reduce excretion in infected individuals. Since contact with faecal matter is one of the main sources of contagion [7]. By the same reasoning, more research should also be done on control strategies around latrines, such as fencing off.

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