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Notes:

# Culling and cattle controls influence tuberculosis risk for badgers

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Contributed by David R. Cox, July 26, 2006

Human and livestock diseases can be difficult to control where infection persists in wildlife populations. In Britain, European badgers (*Meles meles*) are implicated in transmitting *Mycobacterium bovis*, the causative agent of bovine tuberculosis (TB), to cattle. Badger culling has therefore been a component of British TB control policy for many years. However, large-scale field trials have recently shown that badger culling has the capacity to cause both increases and decreases in cattle TB incidence. Here, we show that repeated badger culling in the same area is associated with increasing prevalence of *M. bovis* infection in badgers, especially where landscape features allow badgers from neighboring land to recolonize culled areas. This impact on prevalence in badgers might reduce the beneficial effects of culling on cattle TB incidence, and could contribute to the detrimental effects that have been observed. Additionally, we show that suspension of cattle TB controls during a nationwide epidemic of foot and mouth disease, which substantially delayed removal of TB-affected cattle, was associated with a widespread increase in the prevalence of *M. bovis* infection in badgers. This pattern suggests that infection may be transmitted from cattle to badgers, as well as vice versa. Clearly, disease control measures aimed at either host species may have unintended consequences for transmission, both within and between species. Our findings highlight the need for policymakers to consider multiple transmission routes when managing multihost pathogens.

behavior | bovine tuberculosis | epidemiology | *Meles meles* | perturbation

Many pathogens that can influence human and domestic animal health are sustained in wildlife populations (1). *Mycobacterium bovis*, the causative agent of bovine tuberculosis (TB), is one such pathogen. In Britain, testing and slaughter of infected cattle eradicated the infection in many areas, but control was not achieved where populations of European badgers (*Meles meles*), widespread but protected wild animals, sustain endemic infection (2). Between 1975 and 1997, >20,000 badgers were culled by the British government in a series of attempts to limit TB transmission to cattle (2). Nevertheless, the national incidence of cattle TB has been increasing since the 1980s (2).

A large-scale field trial, the Randomised Badger Culling Trial (RBCT), recently showed that although culling badgers reduced cattle TB incidence where widespread (100 km<sup>2</sup>) culling occurred, incidence was increased on neighboring uncultured lands (3) and in areas where culling was restricted to small patches of land (average, 5.3 km<sup>2</sup>) (4). These detrimental effects of badger culling were attributed to disruption of badgers' territorial organization and expansion of ranging behavior, which were documented in and around culling areas. This social perturbation potentially increased contact with cattle (5), but is likely to

have also influenced contact rates within the badger population (5, 6). Whereas epidemiological models usually assume that depressing host population density will reduce disease transmission through lower contact rates, social perturbation could lead badger culling to generate only small reductions, or even increases, in rates of disease transmission, with concomitant effects on infection prevalence (7). Such effects could be particularly marked where repeated culling and continued immigration prevent reestablishment of a stable spatial organization (6, 8).

Substantial interannual variation in prevalence has been observed in the absence of culling (9), and it is therefore important to distinguish hypothetical culling effects from other factors that could influence TB dynamics in badgers, such as the local prevalence of infection in cattle. However, even though patterns of *M. bovis* infection in cattle and badgers are associated in space (10), distinguishing badger-to-cattle transmission of infection, which has been demonstrated experimentally (3, 4, 11), from cattle-to-badger transmission is problematic in observational studies, especially where badgers are being sampled destructively. While the RBCT was in progress, measures to control a nationwide epidemic of foot-and-mouth disease (FMD) led to a 9-month suspension of routine cattle TB testing (12). This suspension delayed the removal of *M. bovis*-infected cattle (Fig. 1a), increasing the potential for spread among cattle (13), and offering an opportunity to assess the risks of transmission to badgers.

We used statistical models to investigate the effects of badger culling and cattle controls on the prevalence of *M. bovis* infection in badgers. We predicted that prevalence would be higher: (i) in areas that had been culled repeatedly; (ii) where geographical features allowed badgers to recolonize culled areas, promoting social perturbation and hence elevating contact rates; and (iii) when removal of infected cattle had been delayed by suspension of cattle testing. We also investigated an alternative hypothesis that interannual variation in prevalence was related to climate. Weather conditions have been linked to both geographical (14) and temporal (15) variation in cattle TB, as well as to badger population dynamics (16) and could plausibly influence TB dynamics in badgers. The North Atlantic Oscillation (NAO) is a major determinant of weather conditions in Western Europe and

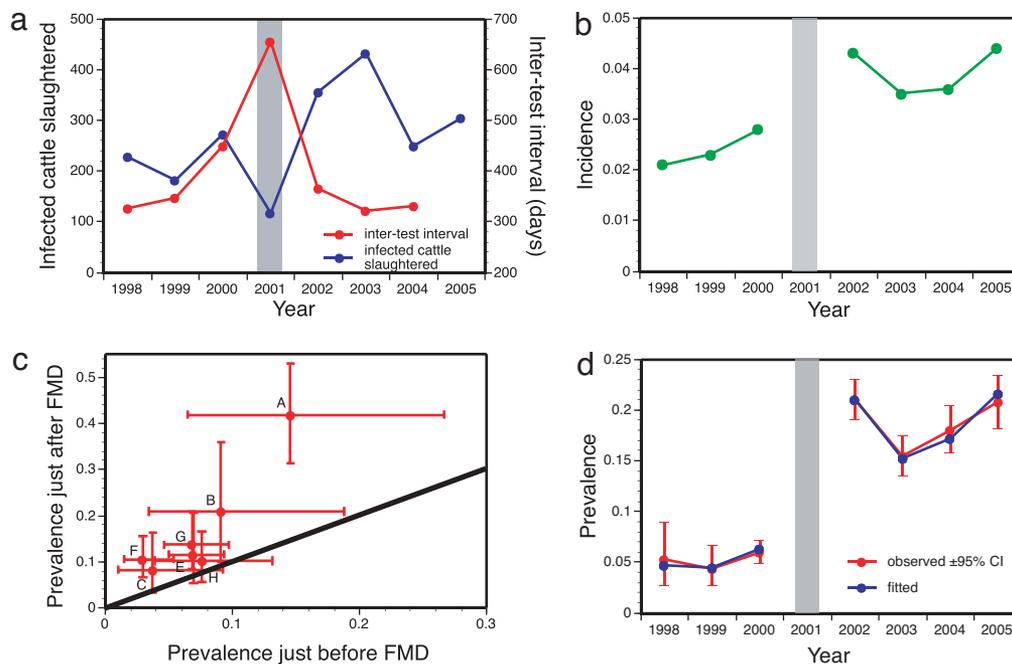
Conflict of interest statement: No conflicts declared.

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Abbreviations: FMD, foot-and-mouth disease; NAO, North Atlantic Oscillation; RBCT, Randomised Badger Culling Trial; TB, tuberculosis.

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**Fig. 1.** Interannual variation in *M. bovis* infection in badgers and cattle. Shading indicates the 2001 FMD epidemic. (a) Median interval between cattle tests in proactive areas enrolled in the RBCT, and total infected cattle slaughtered in all proactive areas. (b) National incidence of cattle TB per calendar year. (c) Prevalence recorded in adult badgers in seven proactive areas before (1999 for area A, 2000 for areas B–H) and after (2002) the FMD epidemic. Error bars give exact binomial 95% confidence intervals, and the black line indicates equal prevalence. (d) Prevalence in proactively culled adult badgers, with fitted values from the model shown in Table 1.

influences a wide array of ecological traits (17), predicting ecological responses more reliably than do local weather conditions (18). We therefore investigated relationships between the NAO and TB dynamics in badgers.

## Results

Our primary analyses concerned adult badgers taken on successive culls in 10 RBCT trial areas. Analyses of other data sets are provided as supporting information, which is published on the PNAS web site.

We constructed a “base model,” including covariates likely to influence observed prevalence (see *Materials and Methods*). This model suggested that, as predicted, the prevalence of *M. bovis* infection increased with successive culls. This increased prevalence was particularly marked when multiple operations were used to complete each annual cull (details in supporting information). However, adding a categorical variable “year” to this base model significantly improved model fit ( $\chi^2 = 35.49$ ,  $df = 6$ ,  $P < 0.001$ ) indicating additional unexplained interannual variation. We therefore explored other covariates that might influence *M. bovis* infection prevalence in badgers.

The national incidence of cattle TB was rising throughout the period of the RBCT (Fig. 1*b*). We therefore hypothesized that *M. bovis* infection prevalence in badgers might also be increasing. However, a simple linear temporal trend, although significant ( $\chi^2 = 14.46$ ,  $df = 1$ ,  $P < 0.001$ ), left much of the observed interannual variation in prevalence unexplained ( $\chi^2 = 21.03$ ,  $df = 5$ ,  $P = 0.001$ ).

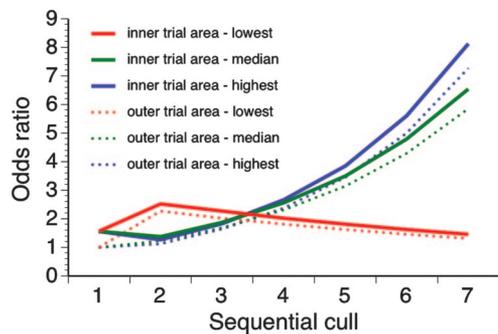
The delayed removal of TB-affected cattle caused by the FMD epidemic was associated with a significant rise in *M. bovis* prevalence in badgers. For each trial area, adding the median interval between the previous year’s cattle tests significantly improved the fit of the base model ( $\chi^2 = 25.38$ ,  $df = 1$ ,  $P < 0.001$ ), with a 1-year delay in cattle testing associated with a 2-fold increase in badger prevalence (odds ratio = 2.00, 95%

confidence interval = 1.52–2.61). Adding this continuous variable explained most of the observed interannual variation ( $\chi^2 = 10.11$ ,  $df = 5$ ,  $P = 0.072$ ). Because the intertest interval was markedly higher in 2001 (the year of the FMD epidemic) than at other times (Fig. 1*a*), the effect of suspended cattle testing could also be represented as a binary variable, distinguishing prevalence in 2002 (after the FMD epidemic) from other years. The fit of the base model was significantly improved by adding this binary FMD variable ( $\chi^2 = 31.94$ ,  $df = 1$ ,  $P < 0.001$ ; odds ratio = 1.86, 95% confidence interval = 1.50–2.31), leaving no interannual variation in prevalence unexplained ( $\chi^2 = 3.55$ ,  $df = 5$ ,  $P = 0.62$ ). This binary FMD variable was preferred over the continuous intertest interval variable because it could be used consistently across multiple data sets (see supporting information). The FMD-associated increase in *M. bovis* infection prevalence in badgers was observed consistently across trial areas enrolled in the RBCT at the time (Fig. 1*c*).

Adding the previous year’s NAO index improved the fit of the base model ( $\chi^2 = 9.32$ ,  $df = 1$ ,  $P = 0.002$ ; odds ratio = 0.48, 95% confidence interval = 0.30–0.77) but left much of the observed interannual variation in prevalence unexplained ( $\chi^2 = 26.17$ ,  $df = 5$ ,  $P < 0.001$ ). Adding the FMD variable improved the fit of the base + NAO model ( $\chi^2 = 23.89$ ,  $df = 1$ ,  $P < 0.001$ ), but adding NAO did not improve the fit of the base + FMD model ( $\chi^2 = 1.27$ ,  $df = 1$ ,  $P = 0.26$ ). These analyses indicate that the FMD variable provided a superior explanation for the interannual variation observed.

Adding the FMD variable did not affect the trend of increasing prevalence on successive culls. Moreover, adding a linear “year” variable to the base + FMD model did not improve model fit ( $\chi^2 = 0.12$ ,  $df = 1$ ,  $P = 0.72$ ) and did not affect the increasing prevalence trend associated with repeated culling, suggesting that the changes observed were not due to an underlying gradual increase in prevalence.

Model fit was further improved by adding terms for the interactions between repeated culling and the permeability of



**Fig. 2.** Effect of repeated proactive culling on *M. bovis* infection prevalence in adult badgers. Prevalence estimates are derived from the model in Table 1 and are represented as odds ratios for badgers captured in inner ( $\geq 2$  km inside, solid lines) and outer ( $< 2$  km inside, dashed lines) regions of trial areas with the lowest (red, permeability = 0.55), median (green, permeability = 0.94), and highest (blue, permeability = 1.0) observed boundary permeability. Odds ratios are calculated relative to initial culls in outer trial areas.

trial area boundaries ( $\chi^2 = 14.39$ ,  $df = 2$ ,  $P < 0.001$ ), showing that successive culls led to increased prevalence only in less geographically isolated areas (Fig. 2). We also detected a significant interaction between badger capture location and the variable describing initial vs. follow-up culls. Prevalence increased more rapidly among badgers captured close to ( $< 2$  km inside) the culling area boundary, although prevalence was initially lower in these regions (Fig. 2). Both of these patterns would be expected if badgers recolonizing culled areas from

neighboring land contributed to social perturbation, and hence to disease transmission.

Conclusions from the final model are summarized in Table 1, and Fig. 1*d* compares fitted values from this model with observed values. Fitted values for each trial area are in the supporting information. Some differences in the year-to-year variation between different trial areas remain unexplained (trial area  $\times$  year  $\chi^2 = 81.08$ ,  $df = 31$ ,  $P < 0.001$ ), but this pattern does not affect the precision of the comparisons that are the primary focus of this study.

## Discussion

Our findings demonstrate the difficulties associated with managing host-pathogen systems with complex dynamics. Simple dynamic models predict that reducing badger population density should reduce disease transmission and hence, prevalence (19). However our results show that repeated badger culling was associated with increasing prevalence of *M. bovis* infection, probably because disruption of territorial organization elevated contact rates (5). As predicted, prevalence increases were most marked close to the borders of culling areas and in areas lacking geographical barriers to badger immigration.

Although proactive badger culling was associated with increasing *M. bovis* infection prevalence, badger densities were substantially lowered inside proactive areas (5) and, probably as a result, cattle TB incidence was consistently reduced (3). Increasing prevalence in badgers may, however, have diminished this beneficial effect. TB incidence was elevated for cattle herds resident on uncultured land neighboring proactive areas (3). We could not measure *M. bovis* infection prevalence among badgers

**Table 1. Predictors of *M. bovis* infection prevalence in adult badgers from 10 RBCT proactive culling areas, based on logistic regression**

Predictor	Odds ratio (95% C.I.)	$\chi^2$	df	P
<b>Base model covariates</b>				
Trial area		253.49	8*	<0.001
Gender		36.86	1	<0.001
Male vs. female	1.54 (1.34–1.76)			
Tooth wear		18.31	5	0.003
2 vs. 1	0.98 (0.54–1.78)			
3 vs. 1	1.24 (0.69–2.23)			
4 vs. 1	1.21 (0.67–2.19)			
5 vs. 1	1.64 (0.89–3.01)			
Not recorded vs. 1	0.45 (0.11–1.85)			
Carcass storage		3.86	1	0.050
>7 days vs. $\leq 7$ days	0.64 (0.41–1.01)			
Necropsy laboratory		26.69	9	<0.001
Culture laboratory		1.55	2	0.460
Cull sequence 1		0.727	1	0.394
Cull sequence 2		52.61	1	<0.001
<b>Interannual variation</b>				
FMD		18.19	1	<0.001
2002 vs. other years	1.70 (1.33–2.16)			
<b>Boundary permeability</b>				
Permeability $\times$ cull sequence-1 interaction <sup>†</sup>		3.16	1	0.076
Permeability $\times$ cull sequence-2 interaction <sup>†</sup>		14.99	1	<0.001
<b>Capture location within trial area</b>				
Capture location <sup>†</sup>		9.49	1	0.002
Capture location $\times$ cull sequence-1 interaction <sup>†</sup>		6.58	1	0.010

C.I., confidence interval.

\*The variable representing 10 trial areas is associated with only eight degrees of freedom because inclusion of the boundary permeability variable (a characteristic of each trial area) accounted for one of the trial area degrees of freedom.

<sup>†</sup>Odds ratios associated with the cull sequence variables and their interaction terms are in Fig. 2. Fitted values are in Fig. 1*d*.



days before necropsy. At necropsy, gender and tooth wear (a measure of age) were recorded (33), and one-half of each retropharyngeal lymph node, both bronchial lymph nodes, and the mediastinal lymph nodes were collected, as were any lesions suggestive of TB. Badgers were considered infected when *M. bovis* was detected in any sample by bacteriological culture (at one of three laboratories), or when acid-fast bacteria was detected in lesions by Ziehl–Neelsen staining (34). Isolates of *M. bovis* were strain-typed by spacer oligonucleotide typing (spoligotyping) (35).

**Statistical Analysis.** We used logistic regression models to investigate the probability of detecting *M. bovis* infection in individual badgers. Analyses presented here involve adults ( $n = 7,129$ ). Analyses of data on cubs are in the supporting information. We constructed a base model including three covariates considered likely to influence the probability of detecting infection (whether the carcass was stored >7 days, necropsy laboratory, and culture laboratory): one to represent geographical variation (trial area) and two previously found to influence prevalence (gender and tooth wear) (10).

The base model also accounted for successive culls conducted in the same area by including two variables indicating: (i) the difference between the initial and all subsequent follow-up culls, and (ii) a linear change between each cull after the first follow-up. The fit of a base model containing these two variables (detailed in supporting information) left little unexplained variation in prevalence resulting from culling sequence ( $\chi^2 = 6.85$ ,

$df = 4$ ,  $P = 0.14$ ). Exploratory analyses (detailed in supporting information) yielded no evidence of overdispersion in the data (as might have been caused by clustering of infection, for example), indicating that, depending on the variables included in the models, data from different badgers could be considered independent.

We assessed the contributions of other predictors of *M. bovis* infection by adding them to the base model and measuring improvements in model fit. “Badger years” were defined as February 1 to January 31 because most badger cubs are born in February (33). Unless otherwise stated, all references to “year” indicate badger years. For each herd, intervals between cattle tests were calculated as the time between the first test conducted after August 1 of each year and the most recent test conducted before that date. Annual NAO indices were calculated from monthly data available at [www.cru.uea.ac.uk/cru/data/nao.htm](http://www.cru.uea.ac.uk/cru/data/nao.htm).

Full models (including an alternate base model and alternate formulations of the FMD and permeability variables), comparisons with other data sets, and data to reconstruct the analyses presented here are presented in the supporting information.

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