Culling and cattle controls influence tuberculosis risk for badgers

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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^2) culling occurred, incidence was increased on neighboring unculled lands (3) and in areas where culling was restricted to small patches of land (average, 5.3 km^2) (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. 1*a*), 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

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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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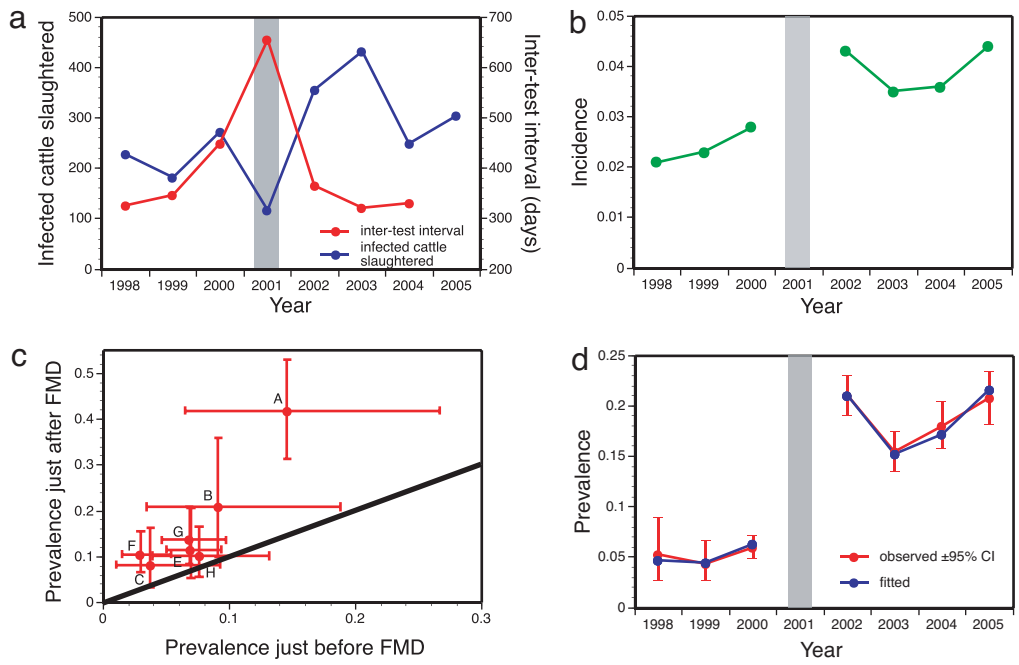

Fig. 1. Interannual variation in *M. bovis* infection in badgers and cattle. Shading indicates the 2001 FMD epidemic. (*a*) Median interval between cattle tests in proactive areas enrolled in the RBCT, and total infected cattle slaughtered in all proactive areas. (*b*) National incidence of cattle TB 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

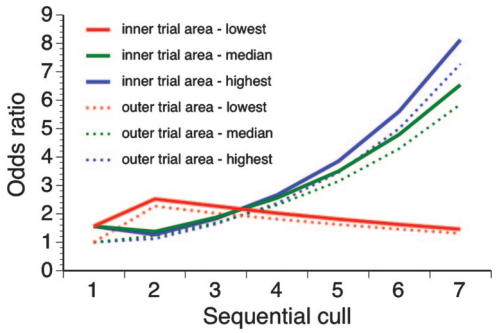

Fig. 2. Effect of repeated proactive culling on *M. bovis* infection prevalence in adult badgers. Prevalence estimates are derived from the model in Table 1 and are represented as odds ratios for badgers captured in inner (≥2 km inside, solid lines) and outer (<2 km inside, dashed lines) regions of trial 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 × 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 unculled 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.)	χ^2	df	*P*
Base model covariates				
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. ≤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
Interannual variation				
FMD		18.19	1	<0.001
2002 vs. other years	1.70 (1.33–2.16)			
Boundary permeability				
Permeability × cull sequence-1 interaction†		3.16	1	0.076
Permeability × cull sequence-2 interaction†		14.99	1	<0.001
Capture location within trial area				
Capture location†		9.49	1	0.002
Capture location × cull sequence-1 interaction†		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.

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

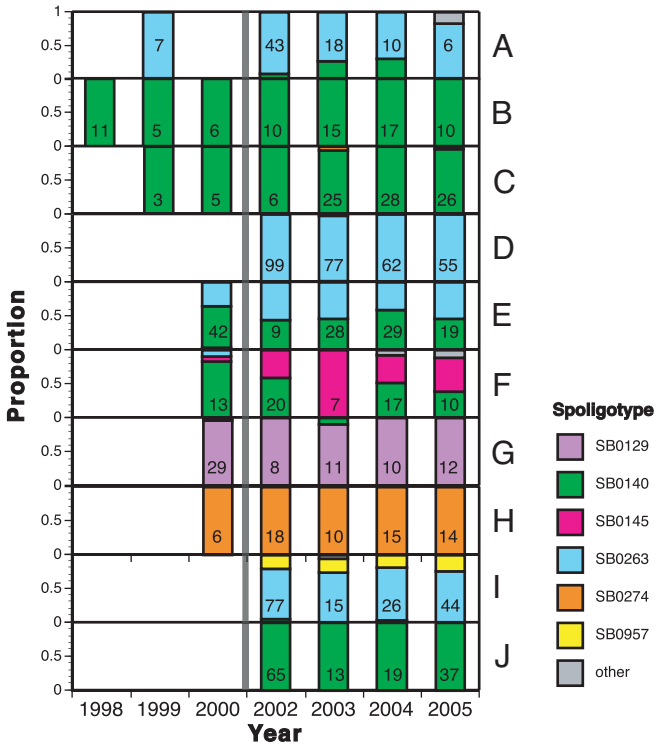

Fig. 3. Strain types (spoligotypes) (35) of *M. bovis* detected in badgers in 10 proactive culling areas (A–J). The solid vertical line indicates the 2001 FMD epidemic. Numbers inside bars give sample sizes. Spoligotypes were available for 1,167 of 1,203 infected badgers.

on such land, but our finding that prevalence increased more markedly among badgers caught closest to such land suggests that detrimental effects in cattle might have partially been caused by increased prevalence in badgers as well as by increased badger-to-cattle contact (5).

A similar study in Ireland did not record increasing *M. bovis* prevalence on successive badger culls (20). This difference in findings may be partly because of the intentional placement of Irish study sites in areas where geographical barriers would impede badger immigration (details in supporting information).

Our results also show an association between suspension of cattle TB controls and increased *M. bovis* prevalence in badgers. These observational data do not conclusively demonstrate causality; however, several alternative explanations are inconsistent with observed patterns. One possibility is that the increased *M. bovis* prevalence observed after the FMD epidemic represented cyclic dynamics, as predicted by some models of badger TB (21). However, it is unlikely that such cyclicity would be synchronized across multiple sites (as was observed) unless the populations were either linked by movement of animals (22) or synchronized by external factors (23). Regular movement of badgers between sites is unlikely, because the distances between culling areas (mean nearest neighbor distance, 31 km; range, 5–99 km) were large relative to the distances moved by badgers in the British Isles during normal ranging (mean home range size, 0.2–2.1 km^2) (24) during dispersal (0.3–3.0 km) (25, 26), inside areas disrupted by culling (maximum 2.2 km (5, 27) and on infrequent temporary movements (up to 7.8 km) (28). Moreover, molecular typing of *M. bovis* isolates demonstrated a highly heterogeneous distribution of strain types across trial areas, which did not change when prevalence increased (Fig. 3). This pattern indicates a widespread increase in local transmission rather than a single epidemic affecting all trial areas.

Like cattle testing, badger culling was suspended during the FMD epidemic, and the consequent delay to repeat culling in proactive areas offers an alternative explanation for the observed increase in prevalence, albeit through an unknown mechanism. Analyses, however, provide no support for this explanation (details in supporting information). Moreover, similar high prevalence after FMD was detected in badgers killed in road traffic accidents in areas not subjected to culling (see supporting information), reinforcing the conclusion that culling did not cause the pattern observed.

Although the suspension of cattle testing during the FMD epidemic was associated with increased *M. bovis* infection prevalence in badgers, this increase would not be expected to undermine the beneficial effects of badger culling on cattle TB incidence. Indeed, because high prevalence was recorded after FMD in both culled and unculled badger populations, the expected benefit of removing badgers by culling could, if anything, have been increased.

Our results illustrate the need to consider all transmission routes in planning control policies for multihost pathogens. Badger culling apparently has the capacity to increase badger-to-badger transmission of infection, potentially undermining anticipated reductions in badger-to-cattle transmission. Likewise, cattle-to-badger transmission appears to be influenced by cattle testing regimes, which suggests that improved cattle controls might not only have immediate benefits through reduced cattle-to-cattle transmission (29), but could also ultimately reduce the probability of reinfection from wildlife. These results suggest that it may be helpful, in this case, to replace the traditional paradigm of a wildlife "reservoir host" from which infection "spills over" into livestock with a more dynamic picture, including substantial transmission both within and between alternative host species.

Materials and Methods

Data Collection. We investigated *M. bovis* infection prevalence among badgers culled in the proactive treatment of the RBCT, a large-scale study of the effectiveness of badger culling as a control measure for cattle TB in Britain (3, 4). Thirty 100-km^2 RBCT trial areas were situated in areas of high cattle-TB risk and recruited sequentially as 10 triplets (designated A–J) with one trial area per triplet randomly allocated to proactive culling (10 areas total). The other trial areas received either reactive (localized) or no badger culling. Attempts were made to place trial area boundaries along geographic features that might impede badger movement, but doing so was rarely possible and trial area boundaries mainly followed property boundaries. We measured the permeability of trial area boundaries for badgers as the proportion of the boundary not composed of coastline, major rivers, dual carriageways, motorways, or conurbations.

The proactive treatment involved a single initial cull across all accessible land in each area, with follow-up culls repeated approximately annually thereafter. Of 41 follow-up culls, 37 were implemented as single operations. The remainder involved 2–6 operations conducted sequentially in different trial area sectors. Initial culls were conducted from 1998 to 2002, and the last follow-up culls occurred in 2005.

Badgers were captured in cage traps, most of which were placed near setts (dens), and then killed by gunshot. The majority of badgers received no injuries because of confinement in the trap (30), and independent audit deemed the dispatch methods humane (31). No culling occurred from February to April of each year to avoid killing females with young cubs confined to the sett (32). Culling was also suspended from May 2001 to January 2002 because of the FMD epidemic.

Each badger was chilled after death and necropsied (at one of nine laboratories), usually within 72 h of dispatch. A proportion (9.8%) of carcasses were stored (nearly always frozen) for >7

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 (χ^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.

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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1. Haydon DT, Cleaveland S, Taylor LH, Laurenson MK (2002) *Emerg Infect Dis* 8:1468–1473.
2. Krebs JR, Anderson R, Clutton-Brock T, Morrison I, Young D, Donnelly C, Frost S, Woodroffe R (1997) *Bovine Tuberculosis in Cattle and Badgers* (Her Majesty's Stationery Office, London).
3. Donnelly CA, Woodroffe R, Cox DR, Bourne FJ, Cheeseman CL, Wei G, Gettinby G, Gilks P, Jenkins H, Johnston WT, *et al.* (2006) *Nature* 439:843–846.
4. Donnelly CA, Woodroffe R, Cox DR, Bourne J, Gettinby G, Le Fevre AM, McInerney JP, Morrison WI (2003) *Nature* 426:834–837.
5. Woodroffe R, Donnelly CA, Cox DR, Bourne FJ, Cheeseman CL, Delahay RJ, Gettinby G, McInerney JP, Morrison WI (2006) *J Appl Ecol* 43:1–10.
6. Tuyttens FAM, Delahay RJ, Macdonald DW, Cheeseman CL, Long B, Donnelly CA (2000) *J Anim Ecol* 69:815–828.
7. Barlow ND (1996) *J Appl Ecol* 33:303–314.
8. Cheeseman CL, Mallinson PJ, Ryan J, Wilesmith JW (1993) in *The Badger*, ed Hayden TJ (Royal Irish Academy, Dublin), pp 78–93.
9. Delahay RJ, Langton S, Smith GC, Clifton-Hadley RS, Cheeseman CL (2000) *J Anim Ecol* 69:428–441.
10. Woodroffe R, Donnelly CA, Johnston WT, Bourne FJ, Cheeseman CL, Clifton-Hadley RS, Cox DR, Gettinby G, Hewinson RG, Le Fevre AM, *et al.* (2005) *J Appl Ecol* 42:852–862.
11. Griffin JM, Williams DH, Kelly GE, Clegg TA, O'Boyle I, Collins JD, More SJ (2005) *Prev Vet Med* 67:237–266.
12. Department for Environment, Food, and Rural Affairs, and Office for National Statistics, London (2004) *The Incidence of TB in Cattle—Great Britain.* Available at www.defra.gov.uk/animalh/tb/stats/index.htm.
13. Cox DR, Donnelly CA, Bourne FJ, Gettinby G, McInerney JP, Morrison WI, Woodroffe R (2005) *Proc Natl Acad Sci USA* 102:17588–17593.
14. Wint GRW, Robinson TP, Bourn DM, Durr PA, Hay SI, Randolph SE, Rogers DJ (2002) *Trends Microbiol* 10:441–444.
15. King EJ, Lovell DJ, Harris S (1999) in *Advances in Vertebrate Pest Management*, eds Cowan DP, Feare CJ (Filander, Fürth, Germany), pp 147–161.
16. Macdonald DW, Newman C (2002) *J Zool* 256:121–138.
17. Mysterud A, Stenseth NC, Yoccoz NG, Ottersen G, Langvatn R (2003) in *The North Atlantic Oscillation: Climatic Significance and Environmental Impact*, eds Hurrell JW, Kushnir Y, Ottersen G, Visbeck M (Am Geophys Union, Washington, DC), pp 235–262.
18. Hallett TB, Coulson T, Pilkington JG, Clutton-Brock TH, Pemberton JM, Grenfell BT (2004) *Nature* 430:71–75.
19. Swinton J, Tuyttens F, Macdonald DW, Nokes DJ, Cheeseman CL, Clifton-Hadley R (1997) *Philos Trans R Soc London B* 352:619–631.
20. Griffin JM, Clegg TA, Quigley F, Hammond RF, McGrath G, Sleeman DP, Costello E, O'Boyle I (2003) in *Selected Papers 2002–2003*, eds Collins JD, Hammond RF (Veterinary Epidemiology and Tuberculosis Investigation Unit, University College Dublin, Dublin), pp 1–12.
21. Anderson RM, Trewhella W (1985) *Philos Trans R Soc London B* 310:327–381.
22. Finkenstadt BF, Grenfell BT (1998) *Proc R Soc London Ser B* 265:211–220.
23. Ranta E, Kaitala V, Lindström J, Lindén H (1995) *Proc R Soc London Ser B* 262:113–118.
24. Woodroffe R, Macdonald DW (1993) *Symp Zool Soc London* 65:145–169.
25. Rogers LM, Delahay RJ, Cheeseman CL, Langton S, Smith GC, Clifton-Hadley RS (1998) *Proc R Soc London Ser B* 265:1–8.
26. Woodroffe R, Macdonald DW, da Silva J (1995) *J Zool* 237:227–239.
27. O'Corry-Crowe G, Eves J, Hayden TJ (1993) in *The Badger*, ed Hayden TJ (Royal Irish Academy, Dublin), pp 35–56.
28. Sleeman DP (1992) in *Wildlife Telemetry*, eds Priede IG, Swift SM (Ellis Horwood, Chichester, UK), pp 670–676.
29. Gilbert M, Mitchell A, Bourn D, Mawdsley J, Clifton-Hadley RS, Wint W (2005) *Nature* 435:491–496.
30. Woodroffe R, Bourne FJ, Cheeseman CL, Cox DR, Donnelly CA, Gettinby G, McInerney JP, Morrison WI (2005) *Anim Welf* 14:11–17.
31. Kirkwood JK (2000) *Humaneness of MAFF's Badger Despatch Procedures* (Department for Environment, Food, and Rural Affairs, London). Available at www.defra.gov.uk/animalh/tb/publications/auditor/report.htm.
32. Woodroffe R, Bourne FJ, Cheeseman CL, Cox DR, Donnelly CA, Gettinby G, McInerney JP, Morrison WI (2005) *Anim Welf* 14:19–25.
33. Neal E, Cheeseman C (1996) *Badgers* (Poyser, London).
34. Gallagher J, Clifton-Hadley RS (2000) *Res Vet Sci* 69:203–217.
35. Kamerbeek J, Schouls L, Kolk A, van Agterveld M, van Soolingen D, Kuijper S, Bunschoten AE, Molhuizen H, Shaw R, Goyal M, van Embden J (1997) *J Clin Microbiol* 35:907–914.