

The impact of localised reactive badger culling versus no culling on TB incidence in British cattle: a randomised trial

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Abstract

Background

Badger (*Meles meles*) culling has formed part of the British government's strategy to control cattle tuberculosis (TB) since the 1970s when *Mycobacterium bovis*, the aetiological agent of bovine TB, was isolated in a badger carcass. Transmission between badgers and cattle has been demonstrated, but the dynamics remain unknown. The ongoing randomised badger culling trial, which began in 1998, was designed to compare two badger culling strategies, subject to welfare constraints, against a control and assess the impact of these strategies on cattle TB incidence.

Methods

Ten matched triplets, each consisting of three trial areas of approximately 100 square kilometres, were identified in areas of high TB incidence in cattle. Within a triplet, trial areas were randomly allocated to one of the two culling strategies or the control with no badger culling. Proactive culling aims to reduce badger densities to low levels across entire trial areas. Localised reactive culling was undertaken in response to the disclosure of TB in cattle herds. Analyses of data on TB incidence in cattle herds in the trial areas are undertaken every six months and will continue throughout the trial. The reactive treatment was suspended on 4 November 2003 based on a previous analysis; the current analysis uses an additional year of incidence data. Triplets were enrolled for an average of 3.6 years at the time of the current analysis. Log-linear regression was fitted to the incidence data and confidence intervals were adjusted for overdispersion.

Results

There were 358 confirmed TB cattle disclosures in the control areas and 356 in the areas receiving localised reactive culling up to 22nd August 2004. After adjustment for covariates, localised reactive badger culling was associated with an estimated 25% increase in the number of cattle herds disclosing TB with 95% confidence interval (CI) 2.6 to 52% increase using the pre-specified time-scale (that is from the end of the initial proactive cull in each triplet). For comparison, the estimate based on the data from the end of the first reactive cull in each triplet was 21% with 95% CI: 1.0% decrease to 48% increase. This paper presents many previously unpublished extensions to these comparisons.

Conclusions

We conclude that reactive culling as performed in the randomised badger culling trial cannot contribute constructively to the control of bovine TB in Britain.

Background

Bovine tuberculosis (TB), caused by *Mycobacterium bovis*, is primarily a disease of cattle. However, other mammals including humans, badgers (*Meles meles*), and deer can be infected. In the 1930s an estimated 40% of dairy cattle in the UK were infected with *M. bovis* [1] and bovine TB was responsible for about 2,000 human deaths annually in the UK, which was 6% of the total deaths from TB at that time [2]. Since then, pasteurization of milk, meat inspection, regular tuberculin testing of cattle, and compulsory slaughter of animals showing evidence of exposure has greatly reduced the risk. This approach, focussing on both public and animal health, has successfully controlled bovine TB in many countries. However in certain regions of the British Isles the incidence in cattle has ceased to fall and has been rising steadily over recent years. Badgers are strongly implicated in transmitting *M. bovis* to cattle, and badger culling has formed a component of British TB control since 1973 [3, 4]. Elsewhere other wildlife reservoirs (possums in New Zealand and deer in Michigan, United States) have been associated with difficulties of controlling bovine TB.

Badgers were first linked to cattle TB in the UK in 1971 when *M. bovis* was isolated in a badger carcass. In response, badger culling was undertaken in localities where TB occurred, though in the past 30 years the culling strategies have changed several times [3, 4]. Initially farmers were licensed to cull badgers. From 1975-1981 badgers were gassed in their setts by Ministry of Agriculture, Fisheries and Food staff. This approach was replaced by cage trapping, initially based on the “clean ring” strategy, designed to identify and remove clusters of infected badgers. From 1986 culling was more limited in scope and took place only on land used by cattle in which *M. bovis* was isolated. This was intended solely as an “interim strategy” pending development of a more selective culling policy [5] but continued until 1997 when a major review of TB control was undertaken [4].

Despite a number of reviews [4-6], the effectiveness of badger-targeted strategies at reducing TB in cattle remains uncertain, since none of the historical badger removal strategies was accompanied for comparison by a control strategy of no culling. Following a recommendation of the Krebs report [4], the Independent Scientific Group (ISG) on Cattle TB was set up and charged with designing and overseeing a large-scale field trial aimed at evaluating two different approaches to badger culling as a means of reducing TB incidence in cattle [7-9].

This ongoing trial is known as the randomised badger culling trial (RBCT). Started at the end of 1998, it investigates 10 matched triplets (A to J), each consisting of three trial areas of approximately 100 square kilometres. Within each triplet, trial areas were randomly allocated to one of three experimental treatments: proactive culling; localised reactive culling in response to TB being confirmed in a cattle herd (a so-called “confirmed herd breakdown”); or the control with no badger culling, which is the policy throughout the rest of Great Britain. Of these treatments, reactive culling was most similar to past policies [4], in that it used cattle TB breakdowns as a sentinel for the presence of potentially infectious badgers, whose removal might be expected to reduce the risk of future breakdowns in that locality. A calculation made when the trial was planned showed that the data from 50 triplet-years (e.g. 10 triplets over a 5-year period) should provide a study of at least 90% power to detect a 20% reduction

in TB incidence [8], a reduction in TB incidence then considered large enough to be of potential policy relevance.

TB incidence in trial areas is analysed and reported to an independent statistical auditor on a regular basis, and this has been the procedure since the first interim analysis in October 2000 [10]. Findings on localised reactive badger culling were first released when this treatment was suspended by UK Ministers on 4 November 2003. The finding [11] was based on data of TB incidence in cattle up to 31 August 2003 when 26.5 triplet-years had accumulated, 11.9 of these being prior to the end of the first reactive cull in each triplet. The analysis revealed that the reactive treatment was associated with an increase in the incidence of confirmed cattle herd breakdowns of 27% with the overdispersion-adjusted 95% confidence interval ranging from a 2.4% decrease to a 65% increase. This apparent increase in the risk of herd breakdowns was an effect in the opposite direction to that expected and, because the 95% confidence interval excluded decreases in incidence greater than 2.4%, the result provided strong evidence that reactive culling, as implemented in the RBCT, had no worthwhile beneficial effect over the time scale considered.

In this paper we present detailed results on the effects of reactive culling on TB incidence in cattle. This paper builds on published analyses of data obtained up to August 2003 [11] by including an additional year of data and presenting a more wide-ranging set of analyses. As yet, the results of proactive culling are undisclosed and will remain so until they are informative.

Methods

Trial Design

The 30 trial areas, were located in areas of the highest incidence of TB in cattle in England as revealed by the most recent three years of breakdown data available at the time of selection [7]. Initial surveys for badger activity were undertaken in all trial areas before the random allocation of treatments, with the trial areas being constructed as 10 sets of matched triplets, identified for convenience with the letters A to J. The three treatments were randomised to trial areas within each triplet except in the case of triplet I, where security considerations led to a specific allocation. (Table 1 gives data on the area and number of active main setts identified at the initial survey for reactive and no-cull areas.)

Data collected in trial areas include information on land occupiers and their links to cattle herds, badger setts and other field signs, trapping operations and culled badgers. This trial database is maintained by the Veterinary Laboratories Agency (VLA) on behalf of the British Department for Environment, Food and Rural Affairs (Defra). This paper presents analysis of data from the trial database up to 7 September 2004. Predominantly the trial data, separate from the cattle incidence data, were used to test for covariate interactions with the treatment comparison.

The data collection and trial implementation are undertaken by two geographically separate branches of the Defra Wildlife Unit (WLU). One is responsible for triplets A, D, E, G and I and the other for triplets B, C, F, H, and J.

Animal welfare considerations favoured the use of cage trapping to capture badgers [7]. Also for welfare reasons, no culling was undertaken in any trial area between February and April [7]. This was to avoid culling females with young cubs still confined to the sett that would starve if their mothers were killed [12]. Trial operations such as cage trapping, humane despatch and surveying are implemented according to standard operating procedures. Many facets have been subjected to independent audit [10, 13-15] as well as analysis by the ISG [12, 16].

As each triplet was enrolled in the trial, an initial cull was undertaken across the area allocated the proactive treatment with the aim of reducing badger densities to low levels across this entire area. Each triplet started contributing data to the study at the completion of its initial proactive cull, the dates ranging from 1998 to 2002 (Table 2). Standard operating procedures called for a major 'follow-up' cull in each proactive area some six to nine months after the initial proactive cull, with follow-up culling annually thereafter to maintain badger densities at low levels.

Reactive culling took place in response to the disclosure of TB in a cattle herd and sought to remove badgers, whose estimated home ranges overlapped the land used by the breakdown herd or herds. For each breakdown, the State Veterinary Service identified 'reactor land' as any parts of a property from which *M. bovis* infection might have originated, either directly through cattle's presence on the land, or indirectly through fodder having been collected there. Badger home ranges that overlapped this reactor land were identified based on the locations of badger setts, latrine sites and landscape barriers such as large roads and rivers. Initially, culling areas were delineated using survey data collected at the time the trial area was recruited; additional field surveys were carried out where subsequent culling was suspected to have disrupted badger spatial organisation. Independent audit showed that this technique successfully identified home range boundaries when main setts were correctly identified (Cresswell, 2001). Trapping then occurred at or near all active setts falling within home ranges which overlapped the reactor land. Hence, culling operations covered the reactor land itself as well as parts of neighbouring properties.

The reactive treatment operated from the first day on which any new confirmed breakdown may have been reported in order to trigger a cull, i.e. the end of the initial proactive cull. The inevitability of delays between reporting of breakdowns and implementation of reactive culling was recognised due to the closed season, the delay in establishing confirmation of disease, as well as administrative and operational demands, as would be the case with implementing this strategy as a TB management policy. As part of their role in the management of the disease the State Veterinary Service (SVS) identified the land associated with herds experiencing confirmed breakdowns and the information was passed to the WLU to trigger a reactive cull. Prior to culling, limited resurveying for badger activity was undertaken to guide trap siting, and over the course of the trial this occurred up to a maximum of 2km from the boundaries of the land associated with the breakdown herd. No reactive culling was undertaken in response to some breakdowns (e.g. because of lack of consent from land owners).

Depending on the triplet, the first reactive cull was conducted between six and 25 months after the initial proactive cull, with major delays in some triplets due to the

field restrictions in place during the FMD epidemic. In triplet J, no reactive culling had been carried out by the time of the ministerial decision to suspend all reactive operations in November 2003. Key information on reactive trial areas is presented in Tables 1 and 2.

Trial areas that were allocated to the no culling treatment have received regular field surveys assessing badger activity and looking for signs of interference. They will continue to do so throughout the trial to ensure that the “control” observations provide a valid basis for comparison against the areas where badgers are actively culled. Since the suspension of the reactive treatment, all reactive trial areas continue to be monitored in the same way as no-cull areas.

During the 2001 FMD epidemic, field work was suspended in all trial areas. No culling took place in the culling period which ran from 1 May 2001 to 31 January 2002. This delayed some proactive follow-up culls and reactive culls in all seven triplets that were active over this time period. Initial proactive culls in triplets D, I and J were delayed until 2002.

Cattle TB incidence data

Cattle herds are subject to regulations set out in the Animal Health Act [17] requiring regular TB tests. Cattle TB tests are arranged by the SVS and conducted by veterinary surgeons. The country is divided into counties and parishes. Within parishes are a number of holdings, typically a single farmed unit, and one or more herds within a holding. Each parish is assigned a parish testing interval of between four years and one year, where parishes with the lowest incidence are assigned four yearly testing and parishes with the highest incidence are assigned annual testing. Routine whole herd tests are carried out in accordance with the parish test interval and additional tests can be conducted at any time, for example, in response to slaughterhouse checks or breakdowns in neighbouring herds. All herds within the RBCT are required to have annual whole herd tests for TB throughout the trial.

All animals are given the single comparative intradermal tuberculin test, which involves injecting purified protein derivative from *M. bovis* and *M. avium* into the skin of the animal at two sites on the neck. Three days later the test is interpreted based on the size of reaction in the skin. If the reaction to *M. bovis* is more than 4mm (under so-called standard interpretation) or 2mm (under severe interpretation) larger than the reaction to *M. avium*, then the animal is categorised as a ‘reactor’. The herd is put under movement restrictions, all reactors are compulsorily slaughtered and subject to post-mortem, and tissue samples cultured for *M. bovis*. If either lesions characteristic of TB are identified at post-mortem or the *M. bovis* organism is cultured, the breakdown is classified as “confirmed” and the severe interpretation of the skin test applied to remaining members of the herd. Otherwise breakdowns are classed as “unconfirmed”. In addition to regular herd testing, the Meat Hygiene Service inspects all cattle sent for slaughter and if suspected cases of TB are identified, they are reported to the SVS. During the 2001 FMD epidemic, few routine cattle TB tests were conducted, delaying the detection of infected herds. All breakdowns are included in the analyses without respect to any, possibly subjective, assessment of the breakdown’s source.

The data from cattle TB tests are managed within the Defra animal health information system VETNET, operated and maintained by the SVS. The VLA receives these cattle data monthly, which provide information on TB tests and breakdowns throughout Great Britain. This paper presents analyses of the cattle TB incidence data up to 22 August 2004.

Data on land owners within and around trial areas (held within the trial database) are cross-referenced to TB test data (held within the VETNET database) to evaluate the treatments. Initially, data identifying farm holdings were collected by Defra WLU staff and checked using the grid references recorded in VETNET to establish the corresponding herd IDs that were inside trial areas. Since then, monthly checks are made to identify and link new holding IDs in the database, and also to identify new herd IDs for existing herds.

As a check, a geographical information system (ArcGIS 9.0) was also used, as an alternative to the “trial database”, to identify herds within trial areas. Herds with VETNET grid references that fell within trial area boundaries were classified as herds inside trial areas and cross-linked to TB test data. This method was independent of the list of land owners used by Defra WLU staff.

Statistical Analysis

In the primary comparison of treatments the outcome variable for analysis is the number of new confirmed herd breakdowns per trial area. All new breakdowns from the start of the study in each area are counted and all three treatments are included in the analysis, although as discussed earlier the proactive results are not presented. Log-linear Poisson regression [18] was used to analyse the TB incidence data implemented in SAS PROC GENMOD [19]. Supplementary analyses are also reported which have used the number of breakdowns of all types, including both confirmed and unconfirmed. Further checks were made analysing the dataset that excluded the proactive treatment (which, when included, contributed information to the estimation of triplet effects). The exclusion of the proactive data results in less data contributing to the analysis thereby reducing power to detect effects.

Table 3 shows the total numbers of confirmed and unconfirmed breakdowns in all reactive and no-cull areas for the 10 experimental triplets. Note that triplets have been under study for different lengths of time (see Table 2). A simple comparison of total numbers suggests similar breakdown rates in control and reactive areas. That comparison, however, ignores the substantial differences between triplets in the no-cull versus reactive comparisons. These differences vary significantly more than expected had breakdowns occurred at random in each trial area ($p < 0.001$ for a test based on the magnitude of the model deviance allowing for the residual degrees of freedom). Such variation if unexplained is known as extra-Poisson overdispersion. There are two possible explanations for such variation and the need for careful study of these accounts for much of the apparent complexity of the resulting analyses.

The first possible explanation is that within each triplet, the no-cull and reactive areas were not as similar at the start of the study as would in principle have been desirable. While randomisation aims to ensure that any such differences have no systematic effect overall, differences could remain between triplets in the apparent no-cull versus reactive comparison. Features of the areas relating to their status before randomisation

may legitimately be used to adjust for an initial lack of similarity, and we have used two broad types of such measures. First are the numbers of cattle herds at the start of the trial (baseline herds), the initial numbers of cattle (baseline cattle), the number of herd tests or the number of cattle tested. Table 3 shows that there are substantial individual differences between trial areas in terms of the number of baseline herds. The second measure concerned the history of the region before inclusion in the trial, in particular the number of breakdowns in a three-year period before the initial proactive cull, or before the 2001 FMD epidemic in the UK for triplets initially culled in 2002. In the case of the primary comparison, allowance was made for the numbers of confirmed historic breakdowns. The duration over which historic incidence was calculated, three years, was chosen to match that used in the original selection of trial areas (though the triplets were selected based on earlier three year periods). These data are comparable between proactive, reactive and no-cull areas because herds were equally like to have 1, 2, 3 or more herd tests in this three-year time period ($p=0.214$ with an average of 2.4 herd tests in each of the treatments over the three-year interval). Again there are quite substantial differences between trial areas. To preserve the linearity of the Poisson model, these covariates required log transformation in the analyses.

A quite different explanation, which could be additional to the above, is that the effectiveness of the reactive strategy really did vary between different triplets, and that this can be explained in terms of a feature of the triplet or of the implementation of the strategy. In statistical terms, this would appear as an interaction between the no-cull versus reactive comparison and the feature in question. An intensive search for such interactions has been made.

In assessing the effect of the reactive strategy an important issue concerns the time origin from which the reactive strategy is judged to operate and from which new cattle breakdowns are to be counted. There are two different approaches. As described above, technically speaking the reactive strategy as a means of managing TB begins at the time breakdowns can be reported to trigger a cull, i.e. at the end of the initial proactive cull. On the other hand, any plausible biological effect of the strategy cannot start until at least the first reactive culling takes place in each area. We have assessed the outcomes of reactive culling using both these starting points. However, the key objective of the trial was the comparison of the two strategies operated under field conditions and consequently on the analysis of the entire data set.

Further consideration was given to a possible starting point some time after the completion first reactive cull. However, there is an immediate effect on the behaviour of remaining badgers (both in targeted social groups and those nearby) following culling [20]. Thus, given that there is likely to be as little as 3 weeks from infection to cattle to a positive result for a tuberculin skin test (personal communication from Chris Howard, Institute for Animal Health: Cattle inoculated intranasally with virulent *M. bovis* showed strong skin test responses by 3 weeks but showed no response at 2 weeks, with the comparative intradermal skin test as currently used.), 3 weeks following reactive culling is arguably a starting point for the referee's "biologically plausible" comparison. However, because reactive culling operations typically ran over a period of between 1 and 2 weeks from the first to the last badger trapped, this translates to only 1.5 weeks following the completion of the first reactive cull (3 weeks to tuberculin test positivity minus 1.5 weeks culling) from the

completion of the first reactive cull until possible positive tuberculin test results among cattle infected by badgers affected by nearby reactive culling. The exclusion of only 1.5 weeks' data would have virtually no effect on the estimates so we did not present this as an alternative analysis. However, we recognize that opinions will vary on whether a different starting point is more biologically plausible.

Only three triplets (A, B and C) provided useful data from the period both before and after the 2001 FMD epidemic. An assessment of the effectiveness of the reactive strategy was undertaken including only breakdowns either before the FMD epidemic or after it. In considering these different time origins it is important that when smaller amounts of data are analysed confidence intervals inevitably expand as a consequence of the decreased amount of information available.

With complete consent and adequate resources, every confirmed breakdown in a reactive area would lead to subsequent reactive culling. Thus, these breakdowns all contribute to the comparison of reactive areas with no-cull areas when the start point is the end of the initial proactive cull. However, in the analyses relating to time periods starting either after the first reactive cull or after the FMD epidemic, many early reactive culls will have been triggered by breakdowns that took place prior to the start point of the period.

Standard errors, and therefore confidence intervals, were adjusted for overdispersion by using an overdispersion-adjustment factor (or inflation factor), which is the square root of the model deviance divided by the degrees of freedom. For most of the analyses the inflation factor was relatively small, less than 1.5, and sometimes only on the borderline of significance; however, in the interests of caution it has been used to inflate standard errors and hence widen confidence limits in all cases where it was greater than 1.

In addition to the main analyses further possibilities have been investigated. To assess the impact of individual triplets on the overall estimate of the reactive treatment, the primary comparison was repeated excluding each triplet in turn. To assess the impact of repeat breakdowns the primary comparison was carried out using the numbers of herds with at least one confirmed breakdown, rather than the number of confirmed breakdowns.

The duration of breakdowns and time to a repeat breakdown were also analysed. Survival analysis was an appropriate method for these outcomes, where the time to the event, defined as either "time to the end of the breakdown" or "time to the repeat breakdown", was analysed [18]. Herds contribute varying amounts of time until either of the above events occurs or the herd becomes censored. When the event was "time to end of the breakdown", the herd was censored if the breakdown did not have an end date recorded on the VETNET system by August 22 2004; and similarly for the event the "time to the repeat breakdown", the relevant time was censored if no repeat breakdown had been recorded by the 22nd August 2004. Weibull regression implemented in SAS PROC LIFEREG [19] was used to estimate the survival parameters, and in both cases adjusted for triplet, treatment, herd type (beef, dairy or other) and herd size.

Results

Reactive culling operations

Of the 266 notifications (of confirmed breakdowns eligible for reactive culling) received by the WLUs prior to 4 November 2003, 164 (62%) had received reactive culling with an additional 18 (7%) having been partially culled in other reactive culling operations. Only 16 (6%) of the farms with notified breakdowns refused consent to cull, whereas 57 (21%) were abandoned on (or had not been timetabled by) 4 November 2003. Of the remaining 11 notifications, no badger activity was found on one farm, and the remaining 10 were abandoned after the long delay imposed by the FMD epidemic so that resources could be concentrated on more recent breakdowns.

Primary comparison and extensions

The analysis of confirmed breakdowns for the 10 triplets from their enrolment in the trial to 22nd August 2004 indicates that the reactive treatment is associated with an estimated increase of 25.0% compared to the no-culling treatment areas (overdispersion adjusted 95% CI: 2.6 to 52.2% increase; $p=0.027$) based on 36.3 triplet-years of data. Adjustment for the initial numbers of cattle herds and historical breakdowns in each trial area, as discussed earlier, accounts for nearly all of the overdispersion; the overdispersion inflation factor was 1.22, $p=0.091$. Adjustment for average herd size was also considered but had virtually no effect on the estimates obtained for the comparison of reactive and no-cull areas. When estimating the effects of both treatments relative to the control, adjustments for systematic differences among triplets were made.

As stated above, we also investigated the effect of the reactive treatment from when we might consider the biological effect of the strategy to start, i.e. from the end of the first reactive cull. Analyses relating to the time period before the first reactive cull included data from triplet J up to 4 November 2003, the date the reactive treatment was suspended. Although no reactive culling took place in Triplet J, analyses relating to the time period since the end of the first reactive cull included data from triplet J from 5 November 2003, to be conservative. Table 4 demonstrates that the inclusion of data from triplet J has decreased the magnitude of our estimates.

As expected, the estimate of the effect of the reactive treatment since the end of the first reactive cull, 21.0%, was similar to the primary comparison (95% CI: 1.0% decrease to 47.8% increase; $p=0.062$, the CI was not adjusted for overdispersion because the inflation factor was less than one). These data covered 24.4 triplet-years of which 8.0 years followed the reactive suspension. In addition, we also compared the effect of the reactive treatment from the trial commencement to the end of the first reactive cull, a total of 11.9 triplet-years. The confidence limits for this comparison were wide namely, from a 9.9% decrease to a 96% increase in herd breakdowns and there was significant overdispersion, $p=0.009$, signifying that the model did not fit the data well. The overall estimate was a 32.8% increase in herd breakdowns in reactive areas, and although this estimate was non-zero, it was imprecisely estimated ($p=0.167$), and the confidence interval included the biologically plausible result of no difference between reactive and survey only areas during this time period. There is no significant difference in the effect of the reactive treatment before and after the treatment was suspended, that is, before and after 4 November 2003 ($p=0.935$).

In the primary comparison, repeat breakdowns on the same farm are included. The Poisson model assumes independence between events but this assumption could be violated because repeat breakdowns could be due to the same on-farm factors. Therefore we analysed the primary comparison with the number of herds that had confirmed breakdowns, i.e. rather than the number of confirmed breakdowns. The reactive treatment was associated with an increase of 28.2% in the number of herds with confirmed breakdowns compared to no-cull areas (95% overdispersion adjusted CI: 4.7 to 57.1% increase $p=0.016$). We also investigated the time to repeat breakdowns using survival analysis but found no significant difference ($p>0.55$ adjusting for different covariates) in the time to second breakdown between reactive and no-cull areas. In the dataset analysed 56 herds in reactive areas and 59 herds in no-cull areas had experienced more than one breakdown during the RBCT.

As an alternative method of identifying herds inside trial areas, the grid references recorded in the VETNET system were used. When the primary comparison was performed on this dataset, the reactive treatment was associated with an increase in herd breakdowns of 17.8% compared to no-cull areas. The overdispersion adjusted 95% confidence limits ranged from a decrease of 1.2% to an increase of 40.4%, $p=0.068$.

The estimates for the primary comparison, excluding each triplet in turn, are presented in Table 4. The reactive treatment was associated with an increase in herd breakdowns as low as 20% when either triplets C or H were excluded and an increase as high as 29% when either triplets B or G were excluded. Confidence intervals (adjusted to account for overdispersion) indicated that the reactive treatment was associated with *at best* a 4% reduction in herd breakdowns (when triplet C was omitted from the analysed dataset) and *at worst* a 61% increase (when triplet B was omitted from the analysed dataset). Based on the analysis of data collected only since the end of the first reactive cull, the confidence intervals indicated that the reactive treatment was associated with *at best* a 6% reduction in herd breakdowns (when triplet H was omitted from the analysed dataset) and *at worst* a 66% increase (when triplet B was omitted from the analysed dataset).

The duration of completed breakdowns was analysed, adjusting for triplet, herd size and herd type. There was no significant difference in the duration of breakdowns in reactive and no-cull areas ($p=0.830$). The number of reactor animals per breakdown, in breakdowns that were not ongoing, was also examined. A negative binomial distribution was used to look at the effect of treatment because there is dependence among reactors within a herd and more variation than expected under a Poisson distribution [18]. The model, adjusting for triplet, the log of historical numbers of reactors, and baseline cattle, found no association between the number of cattle reactors in completed breakdowns and reactive areas compared to no-cull areas ($p=0.154$).

Figure 1 illustrates the relationship between the number of confirmed breakdowns per herd over the study period and the number predicted had the areas not been subject to culling. Points above the line show that reactive trial areas had increased incidence. Nine of the reactive trial areas had more breakdowns than expected using the data to

22 August 2004. Triplet J had received no reactive culling before that treatment was suspended in November 2003.

Figure 2 shows, for each triplet, the ratio of observed breakdowns per herd in reactive relative to no-cull areas calculated for two different periods; the ratio over the study period (vertical axis) is plotted against the equivalent historical ratio as observed prior to the start of the study (horizontal axis). Ratios greater than 1 indicate that there were more breakdowns per herd in reactive areas than in no culling areas after commencement of the trial. An increase in the ratio would suggest an association between the reactive treatment and increased TB incidence and this occurred in seven of the 10 triplets.

Alternative models

Alternative analyses explored the robustness of the overall findings, presented in Tables 5, 6 and 7. Different time periods and different measures of the size of the population at risk were utilised. The differences in confirmed herd breakdowns between reactive and no-cull areas are shown in Table 5 and the primary comparison is included for completeness. In Table 6 the differences in all herd breakdowns, both confirmed and unconfirmed are presented. The models in Table 7 are confirmed herd breakdowns in the dataset that does not include the proactive treatment (which, when included, contributed information to the estimation of triplet effects); when the proactive data are excluded, less data were contributing to the analyses and so power to detect effects was reduced.

The estimates in Table 5 are consistent over the different time periods and adjusted for different measures of the size of the population at risk. All models show an increase of at least 18% in confirmed herd breakdowns for the reactive treatment compared to the no-cull areas. In Table 6, estimates show a comparable increase for all breakdowns, both confirmed and unconfirmed, of at least 13% for reactive areas compared to no-cull areas. When the data analysed exclude the proactive treatment (results shown in Table 7), again all point estimates show an increase in confirmed herd breakdowns associated with the reactive treatment, although in this case some of the estimates were close to zero. Further, when considering the number of confirmed breakdowns since the end of the first reactive cull and the different adjustments, the confidence intervals include the possibility of a reduction in herd breakdowns of at most 20% and an increase in breakdowns of 48%. However, those findings do not change the overall interpretation that there was no evidence that the reactive treatment had any beneficial effect on herd breakdowns over the timescale under study.

Analyses were also conducted using the different time periods for the dataset sourced completely using VETNET. These are presented in Table 8. Again, the findings support the main interpretation.

Stability of treatment effect

Many features which could differ by triplet or trial area were investigated for covariate interaction with the treatment comparison. These are shown in Table 9 and include time-, cattle-, badger-, and historically-based variables. The search has not revealed any significant associations. Variables apart from most time-based ones were added individually to the primary comparison model as main effects and the interaction term with the treatment comparisons was examined for statistical

significance. When we examined changes over time, the data were split into incidence per year. The models were adjusted for either the number of days in the year or the number of tests conducted in the year as well as for all the variables in the primary comparison and the year.

Discussion

The more comprehensive analyses presented here (including consideration of an additional year's data) are consistent with the main findings and conclusions reported in late 2003 [11]. The estimated effect of the reactive treatment is also consistent among triplets with nearly all reactive trial areas demonstrating more breakdowns than expected after adjustment in various ways. Further, the use of different time periods and different variables for estimating the size of the population at risk did not change the findings. Tests for covariate interactions with treatment additionally did not yield any significant results, indicating that the effect associated with the reactive treatment did not vary systematically among triplets.

None of the results suggests that the 16.4 triplet-years of reactive culling carried out in this trial would usefully reduced the incidence of TB in British cattle over the timescale under study. Although analyses of the effects of past, more intensive badger culling interventions indicated a lag of years before a positive effect was clear [21-23], there was no suggestion at any stage in these studies of a detrimental impact of badger culling.

The FMD epidemic affected the control of TB in the UK in many ways. Between 19 February and 28 November 2001 the RBCT field activity was suspended to avoid any risk of spreading FMD. As a result, no culling took place between February 2001 and April 2002. Additionally, to prevent the spread of the disease and due to the diversion of resources into controlling the epidemic, only limited cattle TB tests were conducted. A large backlog of tests had accumulated by the end of the epidemic, and delays in reporting breakdowns occurred, which therefore caused delay in the initiation of reactive culling. From 31 January 2002 movement restrictions were imposed on herds with overdue annual tests as these were at highest risk of having undetected TB. There was no evidence that the delays in testing were systematically different between reactive and no-cull trial areas. However, further detailed investigations of the trial farms culled out to control FMD are planned, e.g. to test whether restocking practices varied systematically

Regular field surveys indicate changes in badger activity consistent with the aims of the culling treatments; badger activity was lowest in areas subject to proactive culling and highest in no-culling areas [24]. Additional analyses of (as yet unpublished) further data confirm that badger culling was associated with badger density in a way agreeing with the different culling strategies, i.e. that badger density was lowest in proactive areas.

Spatial and temporal analyses are currently underway to examine further the association between badger removal and cattle TB breakdowns. The control of badger populations is known to disturb badger social groupings and change badger movement patterns [25-27]. In particular, long-distance movements and dispersal are associated with increased transmission of *M. bovis* among badgers [28]. The

additional analyses aim to quantify the risk of cattle breakdowns when badgers have been removed nearby.

At the time of the present analysis 36.3 triplet-years had accumulated among the 10 triplets, ranging from 5.7 years for triplet B to just over 1.7 years from triplets D, I and J. At the start of the trial it was estimated that a total of 50 triplet-years would detect a 20% reduction in TB incidence. These power calculations were used as a guide and consideration was also given to the replication of effects over sites (triplets) and to the precision of estimates [10]. 26.5 triplet-years had accumulated when the reactive treatment was suspended, 11.9 of these being prior to the end of the first reactive cull in each triplet. Analyses performed at that stage already provided evidence of consistency of the effect of the reactive treatment among triplets. Although the confidence interval did include the possibility of very small reductions in breakdowns due to reactive culling, this was not of sufficient size to suggest to policy makers that any alternative conclusions might be forthcoming had the reactive treatment continued. Further, there was no evidence of trends over time in the effect of the reactive treatment.

Conclusions

The reactive treatment was originally designed as a feasible potential future policy option, operating subject to the constraints of badger welfare, wildlife conservation and the resources available. Reactive culling was also the treatment most similar to badger control policies operating over the previous 30 years. In contrast, the proactive treatment is intended to provide an estimate of the effect on cattle TB of a more extreme approach to badger culling, and also to yield more complete epidemiological data on TB dynamics in badgers. The consistency of the reactive result between the data up to August 2003 and the data up to August 2004 suggests that the situation has not changed with the confidence limits providing no evidence for a beneficial effect of the reactive strategy over the time scale under study.

Competing interests

None declared.

Authors' contributions

AMLF undertook data processing and statistical analyses and drafted the manuscript. CAD and DRC were involved in the analysis plan and interpretation. JB, DRC, CAD, GG, JPM, WIM and RW are the Independent Scientific Group (ISG) on Cattle TB, the group responsible for the design and analysis of the Randomised Badger Culling Trial. RSCH suggested additional analyses and assisted in the interpretation of results. WTJ is a research associate working with the ISG and has assisted in the data processing. All authors have contributed to the drafting of the final manuscript.

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Figures

Figure 1 - Triplet-specific TB incidence in reactive trial areas observed and predicted had they received no culling

TB incidence is the number of confirmed breakdowns since enrolment in the trial to August 2004 divided by the number of baseline herds at risk. Points above the line indicate that increased incidence occurred in reactive trial areas.

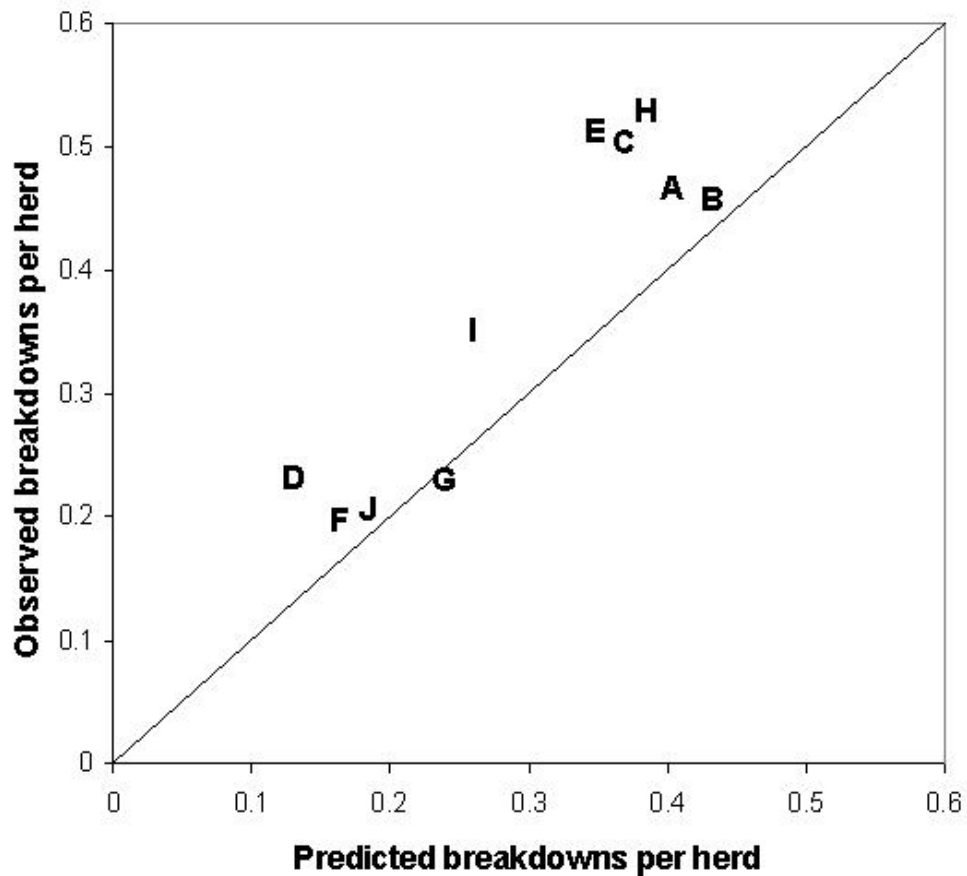
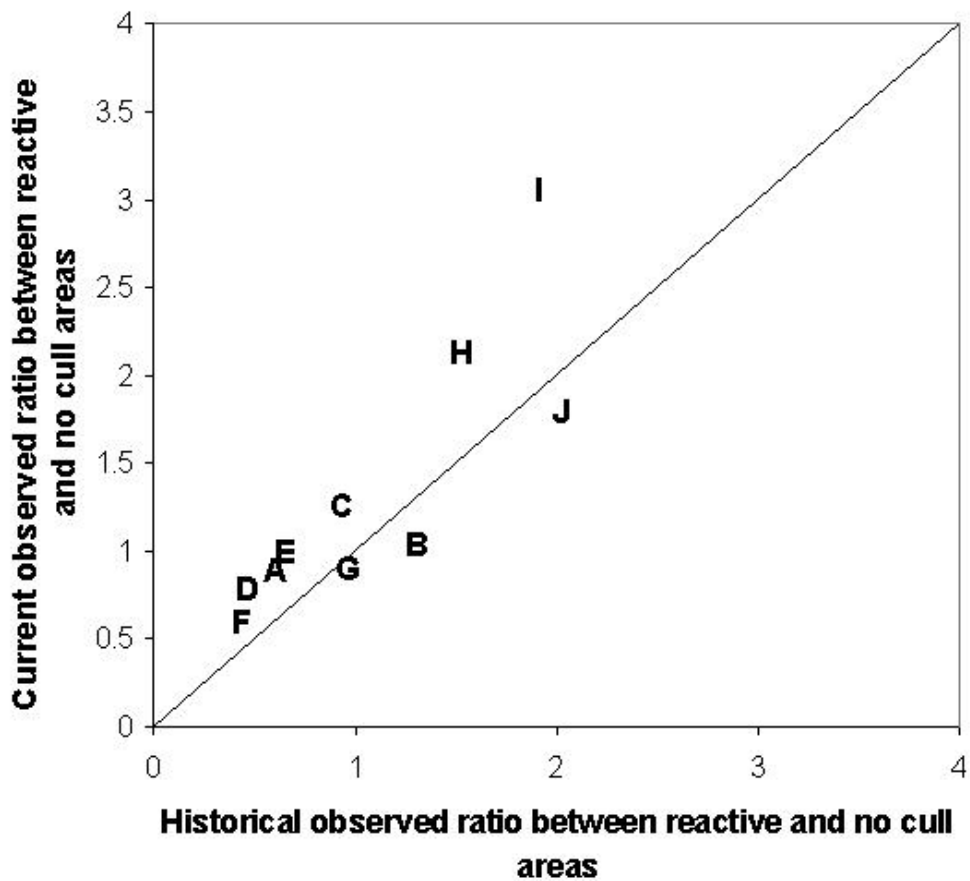


Figure 2 - Observed TB incidence in reactive to no-cull areas, during the trial and three years prior

TB incidence is the number of confirmed breakdowns divided by the number of herds at risk at the commencement of the period. Current data are those breakdowns since enrolment in the trial to August 2004. Historical data are those breakdowns in the three years prior to the initial proactive cull or for triplets D, I and J the three years prior to the 2001 FMD epidemic in the UK.



Tables

Table 1 - Key information on reactive trial areas in the randomised badger culling trial

Triplet	Total surface area (trial area and inner buffer) km ²		Number of active main setts (identified in the initial survey)		Number of active main setts with one or more badgers culled within 500 metres
	No-cull area.	Reactive area	No-cull area.	Reactive area	Reactive area
A	165	163	56	63	16
B	130	119	36	42	29
C	157	145	73	48	25
D	147	154	97	111	20
E	156	149	101	63	16
F	149	164	84	64	21
G	154	156	96	128	42
H	149	145	73	82	10
I	137	155	64	121	16
J	145	132	59	84	0
Total	1489	1485	739	806	195

Table 2 - Key information on reactive trial areas in the randomised badger culling trial

Triplet	Initial proactive cull	First reactive cull	Badgers culled in reactive treatment	Number of reactive operations	Triplet-years in the dataset analysed
A	Jan 2000	Jul 2000	117	10	4.57
B	Dec 1998	Jun 1999	301	10	5.69
C	Oct 1999	May 2000	394	20	4.82
D	Dec 2002	Sep 2003	122	4	1.68
E	May 2000	Jun 2002	188	10	4.24
F	Jul 2000	Aug 2002	435	10	4.10
G	Nov 2000	Aug 2002	256	7	3.78
H	Dec 2000	Jan 2003	159	4	3.69
I	Oct 2002	May 2003	94	3	1.87
J	Oct 2002		0	0	1.85
Total	-	-	2066	78	36.27

The dates of initial proactive culls and first reactive culls by triplet are shown. The total number of badgers culled in reactive trial areas and the total number of reactive operations carried out to 4 November are also given.

Table 3 - Numbers of confirmed and unconfirmed herd breakdowns and important covariates in reactive and no-cull areas

Triplet	Confirmed breakdowns (since the end of the first reactive cull)		Unconfirmed breakdowns (since the end of the first reactive cull)		Baseline herds		Historic confirmed breakdowns	
	Reactive	No-cull	Reactive	No-cull	Reactive	No-cull	Reactive	No-cull
A	43 (39)	40 (34)	32 (25)	15 (15)	92	76	27	36
B	39 (31)	50 (49)	10 (10)	19 (19)	85	113	24	26
C	74 (55)	68 (60)	46 (43)	34 (31)	147	169	25	30
D	21 (8)	20 (7)	8 (4)	7 (4)	90	67	23	44
E	35 (20)	41 (16)	11 (10)	16 (9)	68	79	16	27
F	39 (21)	60 (32)	19 (13)	26 (18)	198	182	19	37
G	27 (14)	27 (15)	14 (8)	9 (5)	117	105	16	15
H	36 (18)	29 (15)	14 (3)	15 (8)	68	117	24	26
I	18 (10)	9 (5)	6 (3)	11 (6)	51	78	40	29
J	24 (10)*	14 (5)*	15 (6)	20 (7)	116	122	43	22
Total	356 (226)	358 (238)	175 (125)	172 (122)	1032	1108	257	292

Numbers of confirmed herd breakdowns in the primary comparison, that is since the end of initial proactive cull. The numbers of baseline herds are those herds inside trial areas at the time of the initial proactive cull. The numbers of historical breakdowns are those confirmed breakdowns in the three years prior to the initial proactive cull or for triplets D, I and J the three years prior to the 2001 FMD epidemic in the UK.

*Although no reactive culling took place in Triplet J, analyses relating to the time period since the end of the first reactive cull included data from triplet J from 5 November 2003, to be conservative.

Table 4 - Percentage difference in the incidence of confirmed herd breakdowns in reactive versus no-cull areas excluding each triplet in turn

Triplet excluded	Since the end of initial proactive cull		Since the end of the first reactive cull	
	Estimate (95% CI)	Inflation factor (p-value)	Estimate (95% CI)	Inflation factor (p-value)
A	26.6% (2.1, 57.1%)	1.25 (p=0.080)	20.4% (-3.3, 49.9%)	0.95 (p=0.552)
B	28.8% (3.3, 60.7%)	1.25 (p=0.081)	31.3% (4.1, 65.6%)	0.80 (p=0.830)
C	20.2% (-4.0, 50.5%)	1.25 (p=0.078)	20.5% (-4.3, 51.7%)	0.96 (p=0.542)
D	25.4% (5.8, 48.8%)	1.03 (p=0.390)	20.6% (-1.5, 47.7%)	0.94 (p=0.584)
E	23.9% (0.7, 52.5%)	1.25 (p=0.085)	16.9% (-4.7, 43.5%)	0.81 (p=0.814)
F	25.0% (1.1, 54.6%)	1.25 (p=0.078)	20.2% (-2.3, 47.9%)	0.94 (p=0.573)
G	28.7% (4.7, 58.2%)	1.20 (p=0.120)	25.2% (1.5, 54.4%)	0.93 (p=0.593)
H	19.9% (-1.4, 45.8%)	1.16 (p=0.178)	16.4% (-5.5, 43.4%)	0.88 (p=0.703)
I	21.8% (-1.3, 50.3%)	1.26 (p=0.073)	17.8% (-4.2, 44.8%)	0.93 (p=0.596)
J	28.1% (2.4, 60.2%)	1.29 (p=0.057)	21.3% (-1.6, 49.6%)	0.98 (p=0.501)

Percentage differences (and 95% confidence intervals) in the incidence of confirmed herd breakdowns between reactive and no-cull trial areas are presented. All confidence intervals are adjusted for overdispersion and the significance of the inflation factor is given. P-values less than 0.05 indicate significant overdispersion, while non-significant p-values indicate that the data fit the model well. The model considers all breakdowns from the initial proactive cull and in each case adjusts for triplet, baseline herd numbers and historic TB incidence. The analysed dataset includes the proactive treatment but results are not presented as that treatment continues.

Table 5 - Percentage difference in the incidence of confirmed herd breakdowns in reactive versus no-cull areas and measures of overdispersion

	Estimate (95% CI)	Inflation factor (p-value)
Since the end of the initial proactive cull		
(a) baseline herds	25.0% (2.6, 52.2%)	1.22 (p=0.091)
(b) baseline cattle	23.5% (-1.5, 54.9%)	1.40 (p=0.012)
(c) number of herd tests	22.5% (0.5, 49.4%)	1.23 (p=0.085)
(d) number of cattle tested	22.8% (-1.8, 53.5%)	1.38 (p=0.015)
Since the end of the initial proactive cull until the end of the first reactive cull		
(a) baseline herds	32.8% (-9.9, 95.6%)	1.42 (p=0.009)
(b) baseline cattle	31.4% (-12.5, 97.3%)	1.45 (p=0.006)
(c) number of herd tests	32.3% (-8.8, 92.1%)	1.37 (p=0.018)
(d) number of cattle tested	33.2% (-9.9, 96.7%)	1.43 (p=0.009)
Since the end of the first reactive cull		
(a) baseline herds	21.0% (-1.0, 47.8%)	0.91 (p=0.647)
(b) baseline cattle	18.9% (-4.8, 48.5%)	1.10 (p=0.246)
(c) number of herd tests	18.4% (-3.1, 44.8%)	0.95 (p=0.558)
(d) number of cattle tested	17.9% (-5.6, 47.3%)	1.10 (p=0.255)
Since the end of the initial proactive cull until the FMD epidemic		
(a) baseline herds	30.0% (-14.7, 98.3%)	1.08 (p=0.301)
(b) baseline cattle	42.8% (-6.8, 118.9%)	1.03 (p=0.386)
(c) number of herd tests	27.5% (-18.4, 99.1%)	1.08 (p=0.306)
(d) number of cattle tested	30.8% (-14.7, 100.4%)	1.09 (p=0.296)
Since the FMD epidemic		
(a) baseline herds	23.7% (-1.1, 54.7%)	1.22 (p=0.090)
(b) baseline cattle	22.5% (-4.1, 56.6%)	1.34 (p=0.027)
(c) number of herd tests	22.9% (-1.6, 53.6%)	1.22 (p=0.093)
(d) number of cattle tested	21.0% (-4.8, 53.8%)	1.32 (p=0.033)

Percentage differences (and 95% confidence intervals) in the incidence of confirmed herd breakdowns between reactive and no-cull trial areas are presented. All confidence intervals are adjusted for overdispersion and the significance of the inflation factor is given. P-values less than 0.05 indicate significant overdispersion, while non-significant p-values indicate that the data fit the model well. Different time periods were considered and in each case adjustments were made for triplet, historic incidence and (a) baseline herds, (b) baseline cattle, (c) number of herd tests conducted or (d) number of cattle tested. The analysed dataset includes the proactive treatment but results are not presented as that treatment continues.

Table 6 - Percentage difference in the incidence of all herd breakdowns (confirmed and unconfirmed) in reactive versus no-cull areas and measures of overdispersion

	Estimate (95% CI)	Inflation factor (p-value)
Since the end of the initial proactive cull		
(a) baseline herds	22.6% (4.7, 43.6%)	1.22 (p=0.094)
(b) baseline cattle	19.8% (-3.4, 48.7%)	1.67 (p<0.001)
(c) number of herd tests	19.0% (2.1, 38.8%)	1.19 (p=0.128)
(d) number of cattle tested	18.0% (-4.6, 46.0%)	1.65 (p<0.001)
Since the end of the initial proactive cull until the end of the first reactive cull		
(a) baseline herds	31.3% (-6.8, 84.9%)	1.49 (p=0.003)
(b) baseline cattle	31.2% (-9.2, 89.5%)	1.59 (p=0.001)
(c) number of herd tests	30.4% (-4.8, 78.5%)	1.38 (p=0.016)
(d) number of cattle tested	28.7% (-9.0, 82.1%)	1.53 (p=0.002)
Since the end of the first reactive cull		
(a) baseline herds	18.2% (-2.5, 43.3%)	1.22 (p=0.094)
(b) baseline cattle	14.9% (-8.4, 44.3%)	1.43 (p=0.008)
(c) number of herd tests	13.6% (-3.7, 33.9%)	1.04 (p=0.365)
(d) number of cattle tested	12.9% (-10.0, 41.7%)	1.41 (p=0.010)
Since the end of the initial proactive cull until the FMD epidemic		
(a) baseline herds	19.8% (-13.6, 66.0%)	1.06 (p=0.347)
(b) baseline cattle	20.8% (-18.0, 77.9%)	1.16 (p=0.201)
(c) number of herd tests	16.1% (-18.6, 65.5%)	1.10 (p=0.277)
(d) number of cattle tested	18.1% (-21.4, 77.4%)	1.15 (p=0.208)
Since the FMD epidemic		
(a) baseline herds	22.0% (2.3, 45.6%)	1.21 (p=0.101)
(b) baseline cattle	19.9% (-4.0, 49.8%)	1.53 (p=0.002)
(c) number of herd tests	19.5% (1.3, 41.0%)	1.14 (p=0.184)
(d) number of cattle tested	18.1% (-5.2, 47.1%)	1.52 (p=0.002)

Percentage differences (and 95% confidence intervals) in the incidence of all herd breakdowns (both confirmed and unconfirmed) between reactive and no-cull trial areas are presented. All confidence intervals are adjusted for overdispersion and the significance of the inflation factor is given. P-values less than 0.05 indicate significant overdispersion, while non-significant p-values indicate that the data fit the model well. Different time periods were considered and in each case adjustments were made for triplet, historic incidence and (a) baseline herds, (b) baseline cattle, (c) number of herd tests conducted or (d) number of cattle tested. The analysed dataset includes the proactive treatment but results are not presented as that treatment continues.

Table 7 - Percentage difference in the incidence of confirmed herd breakdowns in reactive versus no-cull areas and measures of overdispersion. The analysed dataset excludes the proactive treatment.

	Estimate (95% CI)	Inflation factor (p-value)
Since the end of the initial proactive cull		
(a) baseline herds	14.3% (-7.0, 40.5%)	0.92 (p=0.546)
(b) baseline cattle	13.1% (-6.2, 36.4%)	0.92 (p=0.548)
(c) number of herd tests	11.9% (-7.7, 35.8%)	0.91 (p=0.562)
(d) number of cattle tested	13.6% (-5.2, 36.2%)	0.92 (p=0.545)
Since the end of the initial proactive cull until the first reactive cull		
(a) baseline herds	24.6% (-18.1, 89.5%)	1.27 (p=0.125)
(b) baseline cattle	37.1% (-13.8, 117.9%)	1.29 (p=0.110)
(c) number of herd tests	30.7% (-13.7, 98.1%)	1.30 (p=0.106)
(d) number of cattle tested	36.5% (-10.1, 107.2%)	1.29 (p=0.115)
Since the end of the first reactive cull		
(a) baseline herds	8.6% (-20.1, 47.6%)	1.13 (p=0.260)
(b) baseline cattle	2.9% (-21.2, 34.4%)	1.15 (p=0.233)
(c) number of herd tests	5.5% (-20.9, 40.7%)	1.15 (p=0.240)
(d) number of cattle tested	3.2% (-20.7, 34.4%)	1.15 (p=0.232)
Since the end of the initial proactive cull until the FMD epidemic		
(a) baseline herds	61.7% (-33.2, 291%)	1.32 (p=0.137)
(b) baseline cattle	47.0% (-29.9, 209%)	1.34 (p=0.127)
(c) number of herd tests	42.6% (-32.2, 200%)	1.35 (p=0.123)
(d) number of cattle tested	44.5% (-30.3, 200%)	1.34 (p=0.125)
Since the FMD epidemic		
(a) baseline herds	9.9% (-13.4, 39.5%)	1.06 (p=0.348)
(b) baseline cattle	9.9% (-12.4, 37.8%)	1.05 (p=0.353)
(c) number of herd tests	9.0% (-13.2, 36.9%)	1.05 (p=0.363)
(d) number of cattle tested	10.9% (-10.5, 37.3%)	1.06 (p=0.349)

Percentage differences (and 95% confidence intervals) in the incidence of confirmed herd breakdowns between reactive and no-cull trial areas are presented. All confidence intervals are adjusted for overdispersion and the significance of the inflation factor is given. P-values less than 0.05 indicate significant overdispersion, while non-significant p-values indicate that the data fit the model well. Different time periods were considered and in each case adjustments were made for historic incidence and (a) baseline herds, (b) baseline cattle, (c) number of herd tests conducted or (d) number of cattle tested. The analysed dataset excludes the proactive treatment.

Table 8 - Percentage difference in the incidence of confirmed herd breakdowns in reactive versus no-cull areas and measures of overdispersion. Herds were identified using the VETNET grid references and GIS software.

Time period	Estimate (95% CI)	Inflation factor (p-value)
Since the end of the initial proactive cull	17.8% (-1.2, 40.4%)	1.13 (p=0.195)
Since the end of the initial proactive cull until the end of the first reactive cull	25.4% (-10.2, 75%)	1.22 (p=0.094)
Since the end of the first reactive cull	14.4% (-5.3, 38.1%)	0.93 (p=0.612)
Since the end of the initial proactive cull until the FMD epidemic	11.0% (-21.9, 57.9%)	0.88 (p=0.662)
Since the FMD epidemic	17.9% (-2.3, 42.4%)	1.07 (p=0.300)

Percentage differences (and 95% confidence intervals) in the incidence of confirmed herd breakdowns between reactive and no-cull areas are presented. Herds were identified using the VETNET grid references and GIS software. Models adjusted for the number of baseline herds and historical breakdowns. When inflation factors are less than one the confidence intervals are not adjusted using the inflation factor.

Table 9 - Covariates tested for interaction with the treatment comparisons

Covariate	Description
1. Historical variables	
Number of historical breakdowns	Confirmed herd breakdowns in the three years prior to the initial proactive cull or prior to the 2001 UK FMD epidemic for triplets first culled in 2002
Number of badgers culled in badger removal operations	Number of badgers caught during the interim strategy (April 1 1986 to December 12 1998)
Number of badger removal operations	Number of culling operations conducted during the interim strategy (April 1 1986 to December 12 1998)
Badger <i>M. bovis</i> prevalence in badger removal operations	Percentage of <i>M. bovis</i> positive badgers caught during the interim strategy
2. Time related variables	
Triplet duration	Total number of years since the end of the initial proactive cull in the triplet
Calendar year of breakdown	Analysis conducted on yearly incidence by calendar year and adjusting for the number of days in the year
Year since enrolment	Analysis conducted on yearly incidence by the year since the end of the initial proactive cull
Year since initial cull	Analysis conducted on yearly incidence by culling year May 1 to April 30
3. Trial badger variables	
Number of active main setts	Number of active main setts identified during initial surveys before randomisation
Number of active setts	Number of active setts identified during initial surveys before randomisation
Number of badgers caught	Number of proactive badgers caught in initial and first follow-up proactive culls
Proactive badger <i>M. bovis</i> prevalence	Percentage of <i>M. bovis</i> positive badgers caught at initial and first follow-up proactive culls
4. Other trial variables	
Number of baseline herds	Baseline herds must have had a whole herd test in the five years before the initial proactive cull or during the trial and also have been in existence on VETNET
Trap opportunities	Percentage of all cage traps set to catch, which were available to catch badgers, i.e. that were not damaged, and did not catch non-target species etc.
Occupier compliance	Percentage of occupiers agreeing to cull and survey in August 2004
Wildlife Unit	The two Defra Wildlife Unit bases, which undertook trial fieldwork including culling operations.