

- [Accessibility](#)
- [Email alerts](#)
- [RSS feeds](#)
- [Contact us](#)



www.parliament.uk

- [Home](#)
- [Parliamentary business](#)
- [MPs, Lords & offices](#)
- [About Parliament](#)
- [Get involved](#)
- [Visiting](#)
- [Education](#)

- [House of Commons](#)
- [House of Lords](#)
- [What's on](#)
- [Bills & legislation](#)
- [Committees](#)
- [Publications & records](#)
- [Parliament TV](#)
- [News](#)
- [Topics](#)

You are here: [Parliament home page](#) > [Parliamentary business](#) > [Publications and Records](#) > [Committee Publications](#) > [All Select Committee Publications](#) > [Commons Select Committees](#) > [Environment, Food and Rural Affairs](#) > Environment, Food and Rural Affairs

Select Committee on Environment, Food and Rural Affairs [Written Evidence](#)

3. Memorandum submitted by Former Veterinary Officers, State Veterinary Service

Further to my letters of 16 October and 6 November^[3] I enclose a summary of what we would like to present to the Committee.

We are concerned that a number of the assertions put forward by the ISG whilst theoretically feasible are in fact implausible in the light of field experience of the realities of this problem.

In this context we would like to elaborate on three main areas namely:

1. The nature of TB in the badger.
2. Cattle to cattle transmission.
3. The culling efficiency of the RBCT.

THE NATURE OF TB IN BADGERS

1. Tuberculosis has a different manifestation in most species. In the badger it is fundamentally different from TB in cattle essentially due to the lack of development of a hypersensitivity response which is a prime feature of infection in cattle. Thus small numbers of organisms infecting cattle produce a vigorous cellular response which results in extensive cell death and the development of large cold abscesses in the affected tissues usually the lung and respiratory lymph nodes. This is in fact the host immune reaction to TB. Whilst causing disease and disruption to the

affected organs the changes inside these abscesses strongly inhibit the TB bacteria and kill many of them.

The badger does not show such a vigorous destructive reaction but rather a slowly progressive proliferative reaction which eventually results in cell death as numbers of bacteria increase markedly. TB lesions are thus relatively much smaller but contain relatively vastly more bacteria than those of cattle. TB bacteria do not produce toxins but rather cause lesions as a result of their highly antigenic cell walls to which different hosts may respond with greater or lesser aggression.

PROGRESSION OF INFECTION

2. Once a badger develops disease all the members of that social group are likely to become infected due to the confined living space in their underground tunnel systems, their highly gregarious nature and constant mutual grooming. But that seed of infection (the primary focus) will usually only progress to produce disease and eventually death in a minority of cases. Latency is a feature of TB in many species and this is so in badgers and cattle. The bulk of infections in badgers, usually 70% or more will become latent or dormant. A small number of badgers may resolve the infection completely and self cure. But the latent infections remain fully viable and may breakdown under stress which may be of nutritional origin, intercurrent disease, senile deterioration or social disturbance and disruption. Some badgers may develop fulminating disease (Gallagher et al 1998).

Badgers with terminal generalised tuberculosis can excrete vast numbers of bacteria particularly when the kidneys are infected. Counts of several million bacteria in a full urination have been recorded (Gallagher and Clifton-Hadley, 2000).

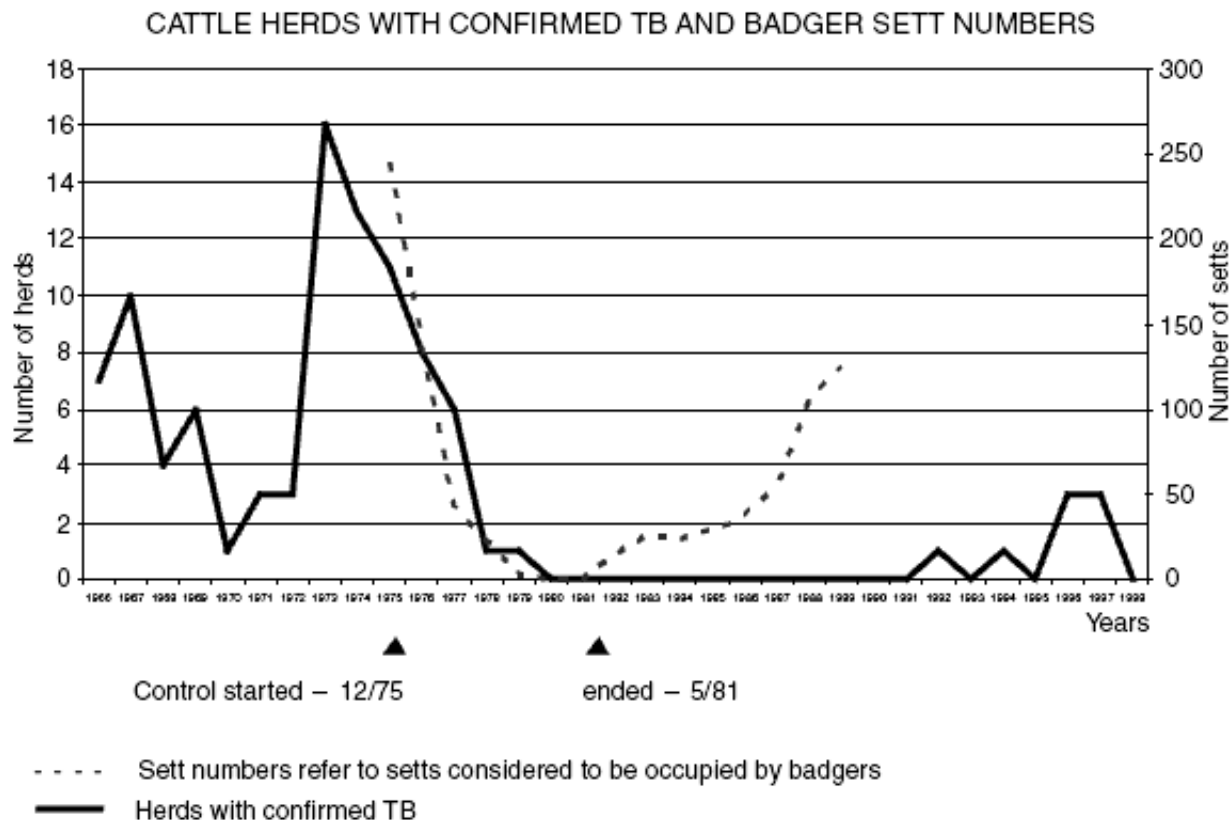
When infection is acquired by a bite wound from the contaminated mouth of another badger, the bacteria are inoculated either deeply subcutaneously or intramuscularly and rapid generalisation of infection usually occurs, causing progression to severe and often fatal tuberculosis which may develop in a matter of several months (Gallagher and Nelson, 1979). Respiratory origin infections have a longer duration and cases in an endemically infected population (Woodchester) have been monitored showing intermittent excretion of infection for a year, with the longest recorded case excreting for almost three years before death.

The above ground mortality due to TB is estimated as about 2% of the population per annum. Thus in the South West alone with its now extensive endemically infected areas the annual deaths due to TB will be of the order of at least 1000 to 2000.

Tuberculosis has an unfettered progress in the badger population and the cycle of infection and disease in the badger has long been known to be self sustaining (Zuckerman 1980). Over time the badger has become well adapted as a primary reservoir host of bovine TB infection.

TRANSMISSION OF INFECTION TO CATTLE

3. The Thornbury Trial, Gloucestershire was set up with the agreement of the then Nature Conservancy and MAFF to test the hypothesis that infected badgers were spreading TB to cattle. Complete social groups were removed from the trial area by gassing of setts whilst routine tuberculin testing of cattle herds continued. Results are shown below.

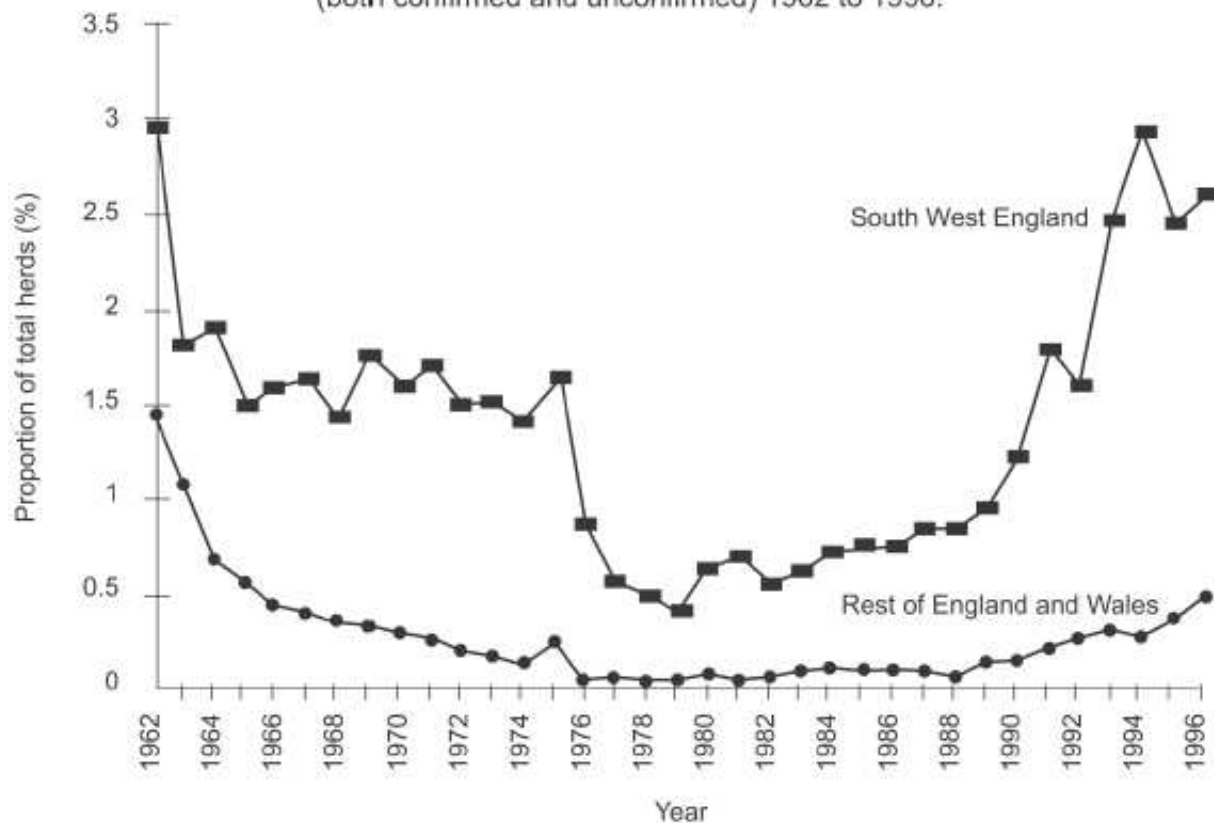


The trial area was 104 km² with 158 farms and over 12,000 cattle. Complete cessation of new cases of TB in cattle indicated that in this area all infections had been of badger origin.

This trial was replicated in Steeple Leaze, Dorset by staff at CVL (now VLA) where again complete cessation of new cases resulted, implying badgers were the sole source of infections there. This effect lasted seven years as thereafter the farms switched to arable production so whilst the duration of the effect may have been longer this could not be determined.

Whilst these trials were progressing strategic control of badgers by gassing was being carried out in the problem areas from 1975 until 1980. In that year a moratorium was introduced during the Zuckerman review. It was restarted later that year but halted in 1981 following the review. These control actions resulted in a more than fourfold reduction in new incidents of TB in cattle herds over this period of five years as shown in the graph below (Krebs 1997).

Figure 1.1 - Proportion of total herds with reactors (both confirmed and unconfirmed) 1962 to 1996.



TRANSMISSION BETWEEN CATTLE

4. Tuberculosis of cattle can be a highly infectious disease resulting in spread within herds and movement of infected individuals has been implicated in spread of infection to other herds. But the frequency with which this occurs in the field situation remains a matter of debate. Cattle to cattle transmission is reported to be of low frequency in the field, in a review of this subject by Menzies and Neil (2000). Features of the current epidemic which are relevant to the assessment of the frequency of this mode of infection are:

- Over recent years from 40 to 55% of outbreaks involve a single reactor and 60% to 80% involve less than three reactors.
- In the majority of breakdown herds (65%) reactors are only found at the initial test
- Reactor herds are usually identified during the autumn round of testing as cattle are brought into the winter housing. Testing during the winter months usually clears the herd. But if between cattle spread occurred it would be most likely during this period of confinement in the buildings.
- Analysis of testing data during the Steeple Leaze clearance trial showed a peak of infections of the circa 600 reactor cattle which occurred in May-June whilst the cattle were at pasture (Wilesmith *et al* 1982).
- None of the 200 reactor animals removed from farms in the RBCT for bacteriological sampling were found to be shedding tubercle bacilli (Sainsbury and Gallagher, 2007).
- The distribution of different spoligotypes in the cattle population is highly clustered geographically. If cattle movements have been spreading the disease around the country then the types would be randomly distributed and this pattern would not be seen.

- The great majority of the TB isolates from cattle and badgers (and other mammalian species) in any area are the same spoligotype, indicating that infection is cycling between these species.

The ISG appear to assert that if an animal is infected it will be shedding bacteria. But this is in conflict with field experience where we have found that unless the animal has lung disease it is highly unlikely to be infectious and associated with multiple cases. This is in line with medical experience where routine tracing of contacts of patients diagnosed with TB is normally carried out only if they are sputum smear positive.

Compared with badger lesions there is a relative paucity of bacteria in cattle lesions other than in those with severe advanced disease.

Whilst the ISG consider cattle to cattle transmission the main mode of infection they do not elaborate how many of the outbreaks encountered during the RBCT were considered due to (1) movements into the herd of infected cattle, (2) to contiguous cattle contact or (3) to a badger source. Rather, they state they have assumed equal weighting to all three sources in their estimates. Yet analysis of on farm outbreak investigations prior to the start of the RBCT had shown less than 10% to be associated with cattle movements and approximately 90% considered due to a badger source (Report 1995, Clifton Hadley 1995). This also correlates more with the field trial findings at Thornbury and Steeple Leaze where all outbreaks there were associated with badgers.

The ISG note the importance of translocation of TB to other areas by movement of infected cattle. They cite the 30 outbreaks post FMD in the four year testing area of North East England where in five cases there was evidence of spread of infection to small numbers of cattle in the recipient herds. But of course this equates to no spread in 83% of these herds yet some of the moved cattle had been there for almost two years.

The ISG also express concern over the sensitivity of the tuberculin test and consider that it is not identifying significant numbers of what they assume to be infectious individuals which are fuelling the deteriorating TB epidemic. Two points need to be considered in relation to this assertion namely:

- All but two States in the EEC have either eradicated TB using this test or are in the final stages of eradication. Those two States are Britain and Ireland. Both States have a reservoir of bovine TB infection in wild badgers.

- Similar views to those of the ISG stimulated a draconian test and slaughter campaign in West Cornwall during the early 1970's using severe interpretation as standard and partial herd slaughters. After several years this was abandoned as it made no difference to the incidence of new outbreaks. A draconian approach was also adopted in Ireland by the CVO Downie in the late 1970's with the same outcome and was accordingly abandoned.

EFFICACY OF CULLING

5. Serious questions remain concerning the efficacy of the culling approach used in the RBCT which is of course fundamental to the proper conduct of a culling trial and significance of its results. Points to consider are:

- Minister's reply to a Written PQ that interference with 57% of traps had occurred and a further 12% had been stolen (*Hansard* 2003).
- Minister's reply to a Written PQ that trapping efficiency had been as low as 30% (*Hansard* 2004).
- Statement by DEFRA that culling efficacy in the RBCT was 20% to 60% (DEFRA,2005).
- Submission by a trapping team supervisor (P Caruana) to this committee that trapping approaches required by the ISG were seriously flawed (EFRACOM 2006).
- ISG Final Report data showing 5 of the crucial initial 10 proactive culls were carried out in midwinter which is well known to be the least successful time for trapping.
- Trapping was carried out for eight days on average and only once a year.
- The average annual rate for the removal of badgers was 1.8 badgers per km² with a variation from 0.7 to 2.91.

— Our local knowledge that trapping success was poor.

Poor culling efficiency would be expected to cause social disruption and dispersal (perturbation) of infected badgers most of which would have been in a state of latent infection. As discussed under 1. above stress caused by such disruption is likely to cause latent infections to become activated and may produce fulminating disease. Culling rationally must always aim to remove the entire social group, as the infected unit. It should avoid dispersing infection to make the situation worse both in the badger population as well as for cattle and of course all other species sharing the badger's habitat.

It is noteworthy that cage trapping was used in the Hartland, Devon control exercise in 1984 and resulted in a fall in confirmed herd outbreaks in cattle from 15% to 4% in 1985 (Krebs 1987). Thereafter annual incidence declined and held at around 1% for nearly 10 years. In excess of 80% to almost 90% of badgers were removed which required protracted trapping efforts in some of the area. In some difficult parts trapping continued for up to three months. The area involved was 62 km² and no so called edge effect was seen during or after this removal.

Dr J Gallagher, former Senior Veterinary Investigation Officer, Devon and Cornwall, former Independent Consultant to DEFRA TB Research Division.

R M Q Sainsbury, former Specialist TB Veterinary Officer, Truro.

A T Turnbull, former Head Notifiable Diseases Section, Tolworth, former Veterinary Advisor to Krebs TB Review Group.

November 2007

References

Clifton-Hadley, R S, Wilesmith, J W, Richards, M S, Upton, P and Johnston, S (1995) The occurrence of *Mycobacterium bovis* infection in cattle in and around an area subject to extensive badger (*Meles meles*) control. *Epidemiol Infect*, 114, 179-93.

DEFRA (2005) Controlling the Spread of Bovine Tuberculosis in Cattle in High Incidence Areas in England: Badger Culling. www.defra.gov.uk/corporate/consult/badgers-tbcontrols/consultation.pdf. Accessed 10 April 2006.

Gallagher, J and Clifton-Hadley, RS (2000) Tuberculosis in badgers; a review of the disease and its significance for other animals. *Res Vet Sci*, 69, 203-217.

Gallagher, J, Monies, R, Gavier-Widen, M and Rule, B (1998) The role of the infected non diseased animal in the pathogenesis of tuberculosis in the badger. *Vet Rec* 142, 710-714.

Gallagher, J and Nelson, J (1979) Causes of ill health and natural death in badgers in Gloucestershire. *Vet Rec*, 105, 546-551.

Hansard (2003) Bovine TB. 8 December 2003, column 218W. London, *Hansard*.

Hansard (2004) Bovine TB. 29 April 2004, column 1189. London, *Hansard*.

Menzies, FD and Neill, SD (2000) Cattle to cattle transmission of bovine tuberculosis.

Vet Journal, 160, 92-106.

Krebs, J R (1997) Bovine tuberculosis in cattle and badgers. Report to Rt Hon Dr Cunningham MP. London: HMSO.

Report (1995) Bovine Tuberculosis in Badgers, 18th report by The Ministry of Agriculture, Fisheries and Food, London, HMSO.

Sainsbury, RMQ and Gallagher, J (2007) TB policy and the ISG's findings. *Vet Rec* 161: 495-496

Wilesmith, J W, Little, T W A, Thompson, H V and Swan, C (1982) Bovine tuberculosis in domestic and wild mammals in an area of Dorset. 1. Tuberculosis in cattle. *J Hyg (Camb)*, 89, 195-210.

Zuckerman, Lord (1980) Badgers, Cattle and Tuberculosis. Report to the Rt Hon Peter Walker MP. London: HMSO.

3 Not printed. [Back](#)

Previous

Contents

Next

Commons

Parliament

Lords

Search

Enquiries

Index

- [A-Z index](#)
- [Glossary](#)
- [Contact us](#)
- [Freedom of Information](#)
- [Jobs](#)
- [Using this website](#)
- [Copyright](#)