Annex A

Bovine TB: the disease, its epidemiology & history of its control in England

1. Bovine TB, caused by the bacterium *Mycobacterium bovis* (*M. bovis*), is a chronic, primarily respiratory, disease that can take a variable amount of time (from a few weeks to a lifetime) to develop from infection to clinical disease and to become infectious to other animals. The transmission rate between animals is low and the epidemic only progresses slowly: on average any infected animal is thought to transmit infection to just over one other animal. Animals infected with TB tend to shed *M. bovis* intermittently in relatively low numbers of organisms (compared to acutely infectious and contagious viral diseases such as Foot and Mouth Disease).

2. *M. bovis* is an intracellular organism[1]. The dominant immune reaction in animals is therefore cellular and detectable using diagnostic tests such as skin tests and gamma interferon, from 2-4 weeks post infection. Only late in disease are there sufficient levels of *M. bovis* to stimulate an antibody reaction which can be detected in circulating blood. As a result of this cellular response, the pathology and clinical signs seen with the disease are associated with the body's immune response to the bacteria, and not solely with the pathogenicity of the bacteria itself. After initial infection the mycobacteria may lie dormant in the body, sometimes without multiplication or producing any visible lesions in tissues for variable amounts of time – this is known as the latent period. During this time the animal may still be infectious, or likely to become so, and is therefore a potential risk for spreading the disease. Therefore cattle that react to the diagnostic tests used but are not found to have gross TB lesions at post-mortem examination (so called non-visibly lesioned, or "NVL" animals) can still be infected and infectious.

3. ***M. bovis* multiplies quite slowly making it very hard to detect** either directly in clinical samples from living animals or by growing it in the laboratory from samples of tissues from dead animals. It also means that there are usually low numbers of organisms in clinical samples: therefore techniques looking directly for the organism or its DNA, such as culture or Polymerase Chain Reaction (PCR), are insensitive. Hence, primary diagnostic tests for *M. bovis* (and those for human *M. tuberculosis*) rely on detecting the immune response of the host (e.g. cow/human) to the organism by a skin reaction or in blood samples. The specificity (the ability of a test to correctly identify truly negative animals) of skin testing in cattle is high[2], therefore a positive result is likely to be a true positive.

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[1] de la Rua-Domenech, R., Goodchild, AT., Vordermeier, HM., Hewinson, RG., Christiansen, KH., Clifton-Hadley, RS., (2006). *Ante mortem diagnosis of tuberculosis in cattle: a review of the tuberculin tests, gamma-interferon assay and other ancillary diagnostic techniques*. Research in Veterinary Science. 81: 190-210.

[2] Goodchild, AV., Clifton-Hadley, RS., (2001). *Cattle-to-cattle transmission of* Mycobacterium bovis. Tuberculosis. 81: 23-41.

Epidemiology

4. This was reviewed by Morris *et al*. (1994)[3]. Although our knowledge of badger ecology and the role of badgers in the disease epidemic has progressed, many of the points raised in this paper are still valid and have been included here.

5. Transmission of *M. bovis* between hosts is thought to be mainly through the airborne route via aerosol droplets. Past studies have shown that low numbers of bacilli are needed to experimentally infect animals via the lung, whereas large doses are required to infect animals via ingestion. This is supported by the distribution of lesions found in cattle. Oral infection of man and calves was important when tuberculous milk was relatively common (when there was still clinical disease in the national dairy herd), although this is now much more rare with ongoing skin testing and removal of reactor cattle.

6. **Transmission can be either direct, through close contact, or indirect** from exposure to viable bacteria in a contaminated environment (for example pasture and feed). The relative contribution of each of these routes has not been quantified. However, we know that *M. bovis* only lives and multiplies in mammalian hosts (or in a laboratory incubator on specific media which mimic the host) and only small numbers of viable bacteria are likely to persist in the environment for any length of time. Outside of a host, survival of *M. bovis* is thought to be usually short (measured in weeks) as bacteria deteriorate quickly, especially if conditions are dry and sunny as they are killed by desiccation and UV light. However, there is published evidence of survival for up to 11 months in ideal conditions[4], i.e. moist and dark areas.

7. **Badgers are known to be a reservoir of *M. bovis* in parts of the UK**, i.e. they are a maintenance host of the disease, and are able to live for several years while infected, breed successfully and transmit disease to other badgers. TB was first identified in a badger in GB in 1971 on a cattle farm undergoing a prolonged breakdown. Transmission between badgers is primarily via the respiratory route, but some will occur through bite-wounding and fighting, particularly in males. Transmission from the mother to her cubs is also thought to occur in the ideal environment of a sett where there is close direct contact. However, detected infection levels were found to be lower in cubs than adults in the Randomised Badger Culling Trial (RBCT).

8. Various estimates have been made of average disease prevalence in badgers in areas where TB is endemic, ranging from 6.9 % to 34.5 % in previous badger removal operations up to 1982, with 33-80 % of social groups found to be infected[5]. During the RBCT, an average of 16.6 % (within a range of 1.6 % to 37.2 %) of badgers in proactively culled areas were found to be infected[6]. The

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[3] Morris, RS., Pfeiffer, DU & Jackson, R (1994). *The epidemiology of* Mycobacterium bovis *infections*. Veterinary Microbiology 40 (1-2): 153-177.

[4] Gallager, J., and Clifton-Hadley, RS., (2000). *Survival of Mycobacterium bovis in defined environmental conditions*. Veterinary Microbiology. 10: 193-197.

[5] Krebs, J.R., Anderson, R., Clutton-Brock, T., Morrison, I., Young, D., Donnelly, C., Frost, S., & Woodroffe, R. (1997). *Bovine tuberculosis in cattle and badgers.* H.M.S.O., London.

[6] Bourne, J., Donnelly, C.A., Cox, D.R., Gettinby, G., McInerney, J.P., Morrison, W.I., & Woodroffe, R. 2007. *Bovine TB: the scientific evidence* Defra www.defra.gov.uk/animalh/tb/isg/pdf/final_report.pdf, London.

estimates are affected by the population studied and the level of detail of the post-mortem examination.

9. Badgers typically live in social groups of 4-7 animals, with defined territorial boundaries. There is evidence from the RBCT, Badger Vaccine field Study (BVS) and longitudinal studies of the badger population at Woodchester Park, Stroud that TB infection is clustered within badger populations and does not spread rapidly between social groups in a stable population. Disruption to a badger population, e.g. by incomplete culling, can lead to “perturbation” – an abnormal increase in ranging behaviour and breakdown of territorial boundaries[7]. The ISG concluded this lead to increased TB prevalence and ranging in badgers, and an increased risk of disease transmission in badgers and from badgers to cattle known as the “perturbation effect”.

10. *M. bovis* can affect a wide range of domesticated mammalian species including cats, dogs, sheep, pigs, camelids (llamas and alpacas), goats and deer as well as a range of wild species. At present these species seem to be spill-over hosts in the UK, acquiring infection from the significant maintenance hosts which are badgers and cattle. However the risk posed by other infected species will depend on the level of disease in the population and the environment, such as location, density, behaviour etc. For instance wild boar are a maintenance host in Spain[8]. In England, a recent wild and park deer prevalence survey and risk assessment[9] concluded that although they can be infected with TB, it is unlikely they currently play a significant role in the epidemiology of TB in cattle, other than in some isolated cases.

11. **Evidence from countries with a wildlife reservoir shows that TB control measures in cattle populations alone are not successful in eradicating the disease**. However, a programme of badger control will at best reduce but not eradicate TB infection within the remaining badger population. Control and eradication of TB in England therefore requires addressing the disease in both badgers and cattle with a package of control measures aimed at reducing transmission and the overall weight of TB infection.

History of TB controls

12. Interest in the control of TB in cattle in the UK began shortly after Robert Koch’s characterisation of the “TB bacillus” in 1882, followed by his development of the first tuberculin in 1890, which was initially tried as a vaccine but shown to have more diagnostic potential. At this time many people were infected and died from milk-borne *M. bovis*. Initial control relied on the clinical examination of cattle

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[7] Carter, SP., Delahay, RJ., Smith, GC., Macdonald, DW., Riordan, P., Etherington, TR., Pimley, ER., Walker, NJ., Cheeseman, CL. (2007). *Culling-induced social perturbation in Eurasian badgers Meles meles and the management of TB in cattle: an analysis of a critical problem in applied ecology*. Proceedings of the Royal Society B: Biological Sciences. 274: 2769 – 2777.

[8] Vicente, J., Höfle, H., Garrido, JM., Fernández-De-Mera, IG., Juste, R., Barral, M., Gortazar, C. (2006). *Wild boar and red deer display high prevalences of tuberculosis-like lesions in Spain.* Veterinary Research 37 (1) 107-119.

[9] The final report of the deer risk assessment is available at http://randd.defra.gov.uk/Default.aspx?Menu=Menu&Module=More&Location=None&ProjectID=13622 accessed 9th September 2010.

herds and bacteriological examination of milk, with voluntary slaughter of any tuberculous dairy cows, which did little to limit *M. bovis* prevalence in the dairy herd.

13. It was not until 1935 that the then Ministry of Agriculture and Food (MAF) launched the first voluntary testing scheme in England and Wales. This introduced the basic principles of regular skin testing and compulsory slaughter (with compensation) of clinical cases and test reactors, with additional monetary incentives for attested herd owners (bonuses per gallon of milk or head of cattle).

14. A national compulsory TB eradication scheme using the tuberculin skin test and slaughter of reactor cattle was launched on 1st October 1950. This took the form of an Area Eradication Plan which began in those counties of GB with the lowest bovine TB incidence (and those with the highest proportion of herds enrolled in the voluntary attestation schemes) and was gradually rolled out to the rest of the country. The whole of GB became "attested" on 1st October 1960 (i.e. each cattle herd was certified as being subject to regular tuberculin testing with immediate slaughter of any reactors). Once a county had remained TB attested for some time, with a very low incidence of reactor herds, the testing interval became extended to 2, 3 and eventually 4 years. Progress was maintained throughout the 1960s and 1970s, with the incidence of bovine TB reaching a historical minimum in the late 1970s and early 1980s. This highly successful test and slaughter scheme reduced the annual number and rate of test reactors from nearly 15,000 (16.2 reactors per 10,000 cattle tests) in 1961 to 569 (2.3 reactors per 10,000 tests) in 1982. As the testing coverage of the national herd improved year on year, the contribution of clinical examinations to the detection of infected cattle steadily declined and by the mid-1960s less than 10 cattle with clinical TB were being removed each year. Over the same period, the proportion of all tested herds with reactors fell from 3.5 % to 0.49 %.

15. **Unfortunately, the progressive reduction in TB incidence stalled in the mid-1980s**. TB herd incidence in parts of the Southwest of England had remained about three times higher than in the rest of GB, despite the retention of an annual (and occasionally more frequent) tuberculin testing regime in those areas. The difficulties in resolving these final TB hotspots, and the identification in 1971 on a Gloucestershire farm of a wild Eurasian badger (*Meles meles*) infected with *M. bovis*, meant that attention began to turn to the badger as a possible wildlife reservoir of infection. Subsequent studies (such as the RBCT) have demonstrated that the badger is a maintenance host to *M. bovis* and a major impediment to the eradication of TB in large tracts of GB, notably the Southwest and parts of the Midlands in England and in Wales. From 1973 to 1998, the cattle test-and-slaughter regime was complemented with a succession of culling strategies, aimed at reducing badger populations in the areas where bovine TB remained endemic (see **Table 1**), but in the absence of experimental controls it is not possible to know whether the observed fall in breakdowns was due to badger removal or some other factor.

16. There are several other countries in which bovine TB is present in the wildlife population. A smaller number have a true wildlife reservoir, in which the wildlife population can sustain TB infection on its own, regardless of TB levels in cattle.

17. Of those countries which have successfully eradicated TB from cattle, only Australia is known to have had a longstanding wildlife reservoir. Australia achieved TB eradication through stringent cattle controls, combined with a control programme targeting wildlife. In other countries, such as the USA and Canada, a significant wildlife reservoir only became evident when TB was nearing eradication, making it necessary to introduce further control measures in certain regions[10],[11]. In France, TB eradication was officially achieved in 2000, but localised wildlife reservoirs have since emerged[12].

18. Other countries outside the UK with a known wildlife reservoir include the Republic of Ireland, Spain[8], and New Zealand. These countries have not been able to eradicate the disease solely through use of cattle control measures and wildlife controls have been implemented. New Zealand has made substantial progress towards this goal[13], helped by the attitude of farmers and the public towards possums in this country.

[10] O'Brien, D. J., Schmitt, S. M., Fitzgerald, S. D., Berry, D.E., Hickling, G.J. (2006). *Managing the wildlife reservoir of Mycobacterium bovis: the Michigan, USA, experience*. Veterinary Microbiology. *112:* 313–323.
[11] Nishi, J. S., Shury, T., Elkin, B. T. (2006). *Wildlife reservoirs for bovine tuberculosis (Mycobacterium* bovis*) in Canada: strategies for management and research*. Veterinary Microbiology. 112: 325–338.
[12] Zanella, G., Durand, B., Hars, J., Moutou, F., Garin-Bastuji, B., Ducauchelle, A., Ferme, M., Karoui, C., Boschiroli, M. L. (2008). Mycobacterium *bovis in wildlife in France.* Journal of Wildlife Diseases. 44: 99-108.
[13] Ryan, T. J., Livingstone, P. G., Ramsey, D. S. L., de Lisle, G. W., Nugent, G., Collins, D. M., Buddle, B. M. (2006). *Advances in understanding disease epidemiology and implications for control and eradication of tuberculosis in livestock: The experience from New Zealand*. Veterinary Microbiology. 112: 211–219.

Table 1. Previous culling control strategies in England (adapted from the Krebs Report[5])

Strategy	**Gassing**	**Clean ring**	**Live test**	**Interim strategy**
Dates	1975-1982	1982-1986	1994-1996	1986-1997
Badger sampling	Yes - 2 badgers per social group were sampled (culture)	Yes - 2 badgers per social group were sampled (post mortem and culture)	Yes - BROCK ELISA antibody test	No
Culling technique	Gassing with hydrogen cyanide	Cage trap and shoot	Cage trap and shoot	Cage trap and shoot
Prevention of recolonisation	12 months	6 months	3 months	None
Size of removal area	Up to $10km^2$	Mean $9km^2$	Mean $1km^2$	Mean $12km^2$
Effectiveness (number of badgers taken)	High	Med-high (70-80% of social group removed)	Variable	Low (due to low sensitivity)
Lactating sow policy (akin to a closed season)	None	Released	Released	Released
Effect on badgers	No post mortem of badgers.	Recolonising badgers had similar prevalence.	Unknown	Recolonising badgers had similar prevalence.
Effect on cattle	TB breakdowns fell (for up to 10 years in the Thornbury area)	In large areas, breakdowns were fewer.	Unknown	More larger breakdowns, more NVL breakdowns
Notes	Setts identified by field worker experience.	Social groups identified by bait marking. Samples taken from carcases from social groups to identify infected groups, which were subsequently removed– extended out to successive social groups until a clean ring of uninfected social groups was found.	This was a trial in which badger groups were randomly allocated to test or interim strategies.	Limited badger removal operations in response to confirmed herd breakdowns attributed to badgers, as recommended in the Dunnet Review until there were sufficient further data on the role of badger culling in controlling bTB and a reliable and effective diagnostic test for TB in badgers.