

## **BOVINE TB – THE FACTS**

### **Question**

### **TB TESTING**

1. Is it true skin test positive animals that show no signs of diseased tissue at post-mortem examination (*known as NVL or no visible lesions*) have never had the infection?
2. Is it true the skin test only picks up half of all infections?
3. Should skin test positive animals be kept because they have mounted an immune response to the disease and are therefore protected?
4. Is it true the skin test is a good herd screening test but a poor individual animal test for bovine TB?
5. Does tuberculin from different sources give different results?
6. If animals test positive to bovine TB using the gamma interferon blood test but show no visible lesions, are they disease free?
7. Negative culture results from the lab must mean no infection is present?
8. Does the gamma interferon test give a large percentage of false positives?

### **COMPROMISING FACTORS FOR TB TESTING**

9. Do animals with fluke show a stronger reaction to the skin test and result in false positive reactions?
10. Does the use of flukicide reduce the reaction to the skin test?

11. Is TB testing compromised by the presence of Johne's disease?
12. What is the TB implication of BVD infection in herds?
13. If the skin test for bovine TB can be compromised by other mycobacteria (e.g. avium, microti), is the gamma interferon test compromised in the same way?
14. Is there is a large amount of undetected infection in cattle herds?

#### TB IN CATTLE

15. Is it true a large proportion of cattle are never tested?
16. Do cattle become infectious only in the late stages of TB - once they have developed "open" lesions?
17. Isn't it pointless to test calves for TB as this is a disease of adult cattle?
18. Why are the genotypes (strains) of *M. bovis* geographically clustered in GB if the movement of cattle is the major cause of spread of disease?  
  
Why not an even distribution of all spoligotypes or at the very least a spread in keeping with the major movements of cattle?

#### TRANSMISSION

19. Does cattle to cattle contact only account for 1 - 2% of all TB cases?
20. Can cattle become infected by badgers and their infected excreta only when out at pasture?

- 21. Do cattle regularly give TB to badgers?
- 22. Can cattle that stray into a herd for a day cause a TB breakdown in that herd?
- 23. Is the requirement for the isolation of reactors really necessary?

**INFECTED BADGERS/BADGER SETS**

- 24. Can the badgers in a sett be proven to have TB by testing the soil and faeces?
- 25. Isn't it relatively easy to identify TB infected badgers on the basis of appearance and behaviour?
- 26. Is it easy to identify TB infected setts?

**RESISTANCE/SUSCEPTIBILITY**

- 27. Are some cattle breeds more resistant to bovine TB than others?
- 28. Do family lines within the same breed have different levels of susceptibility?

**BADGERS AND BOVINE TB**

- 29. Are 60% of badgers in 'Hot Spot' areas infected with TB?
- 30. How much cattle TB is caused by badgers?
- 31. Do badgers infected with TB suffer?

- 32. Will TB in badgers die out if disease is controlled in cattle?
- 33. Are there many more badgers in England and Wales now than in the 1990s?

#### BADGER CULLING

- 34. Was the risk of perturbation and subsequent effects sustained after proactive culling in the RBCT had stopped?
- 35. Did the results of the RBCT demonstrate that reactive badger culling has no role in bovine TB control in GB?

#### VACCINES

- 36. Badger vaccine will not be ready for several years?
- 37. Isn't it pointless to start a badger vaccination programme before infected badgers are removed?
- 38. Will cattle vaccine ever be allowed, due to international trade regulations?
- 39. Is vaccination the 'magic bullet' for TB control?

#### OTHER SPECIES

- 40. Are other wild mammals a TB risk to cattle?
- 41. Are wild deer as much a risk to cattle as badgers?
- 42. Are pigs a dead-end host of *M. bovis*?

**TB CONTROL/ERADICATION**

- 43. **Can TB be eradicated from cattle through extra cattle measures without addressing the wildlife reservoir?**
- 44. **Can tuberculin testing and slaughter of cattle eradicate the disease in cattle?**
- 45. **Did the gamma interferon test make a significant contribution to the eradication of bovine TB in Australia?**
- 46. **Is pre-movement testing a waste of time and money?**
- 47. **Isn't TB in cattle just an economic problem - not an animal health one?**
- 48. **Does a badger vaccine against bovine TB offer the best prospect of eradicating TB in the UK?**
- 49. **Is the UK an OTF country?**

**PUBLIC HEALTH**

- 50. **What is the public health risk of TB in cattle and other species in the UK?**
- 51. **Does raw milk give you immunity against bovine TB?**
- 52. **Are TB infected camelids ( llamas and alpacas) a significant public health risk?**

**HUSBANDRY AND BIOSECURITY**

- 53. **Will supplementing cattle feed with trace elements and/or selenium prevent a TB outbreak?**

- 54. Do cattle only become infected by badgers through close contact? Close the barn doors, put up electric fencing around silage clamps and you will resolve the problem...**
- 55. Does growing maize increase the risk of a TB breakdown in your herd?**
- 56. Does ensiling kill the TB bacterium?**
- 57. Do iron rich soils cause bovine TB in cattle?**
- 58. Is there a risk from spreading slurry on land used by cattle?**
- 59. Is cleansing and disinfection (C&D) of buildings/yards used by reactor cattle a waste of time?**
- 60. Are newly calved cows more prone to give a false positive reaction to a TB test?**

## ***BOVINE TB – THE FACTS***

### **TB TESTING**

**Q1. Is it true skin test positive animals that show no signs of diseased tissue at post-mortem examination (*known as NVL or no visible lesions*) have never had the infection?**

**A1.** The false positive rate for the skin test is very low (1 in 1000, see also question 2) and so it is very likely that any animal that tests positive is infected, regardless of whether this is confirmed at post-mortem.

**Background:** The specificity of a test can be defined as the proportion of truly non-infected animals in a screened population that are correctly identified as non-infected (i.e. “negative”) by the test. The large percentage of reactors in which no visible lesions are detected at post-mortem examination (approximately 60%) is often cited as evidence of poor specificity of the comparative tuberculin skin test. However, this is not the case.

Admittedly, no screening and diagnostic test is perfect (i.e. 100% sensitive and 100% specific), but the comparative tuberculin skin test used in the UK and Ireland has a specificity in excess of 99.9%. This means that only 1 in every 1000 (or more) truly non-infected cattle that are correctly tested will be expected to be misclassified as reactors (i.e. false positives). These genuine false positive reactors may be caused by non-specific tuberculin responses to the environmental mycobacteria that cattle are sometimes exposed to.

The ease with which the typical lesions of bovine TB can be detected and the causative bacterium isolated from tissues depend upon the thoroughness of post-mortem inspection and the stage of *Mycobacterium bovis* (*M. bovis*) infection, being harder early in infection when most cattle are detected by the skin test.

It is important to remember that the skin (and gamma interferon blood) tests are designed to detect an immune response to TB *infection* rather than the signs of *disease*. Because immune responses in TB infected cattle usually develop before visible signs of disease are evident to the vet or meat inspector, the percentage of test reactors without visible tuberculous lesions or positive cultures are not valid indicators of the false positive fraction for this test.

**Q2. Is it true the skin test only picks**

**Background:** No diagnostic test, including the tuberculin skin test, is 100% accurate, but the current

<p><b>up half of all infections?</b></p>	<p>skin test is effective (and is the primary diagnostic test required under EU legislation). On the one hand, the comparative skin test used in the UK and Ireland can be expected to vary around approximately 80% detection rate of all the infected cattle in a herd at any one test (at standard interpretation, range 52-100%). On the other hand, reactions to the tuberculin test can sometimes be caused by exposure to other mycobacteria which do not cause bovine TB. When the skin test is applied to cattle without TB in Great Britain, there is a 1 in 1,000 chance that a non-infected animal will be wrongly classified as a reactor.</p> <p>The sensitivity of a test can be defined as the proportion of truly infected animals in a screened population (e.g. a herd) that are correctly identified as infected (i.e. “positive”) by that test. In the case of most tests, sensitivity and specificity (see also question 1) are inversely related. In other words, as sensitivity is increased, specificity will be reduced and vice-versa, A compromise between the two must therefore be selected. It is important to understand that neither sensitivity nor specificity are fixed and the compromise between the two is selected according to the job that we want the test to do and the population in which we want it to do it. An example of changing (enhancing) the sensitivity of a screening test is the application of the severe interpretation of the skin test in those circumstances where we are certain that <i>M. bovis</i> infection is present in the herd being tested.</p> <p>Several studies from various countries have reported estimates of sensitivity for the comparative and other variants of the tuberculin skin test. Test sensitivity (and specificity) is independent of the prevalence of infection in the population and is frequently assumed to be constant across different populations. In practice, however, it can be influenced by a host of other factors including the test procedure, cut-off point for a positive result, tuberculin potency, the stage of infection in the host, other inter-current infections and prevalence of cross-reacting organisms in the locality. It is thus very difficult to quote a single sensitivity estimate for the comparative skin test that would apply to all herds in GB at all times.</p> <p>Studies evaluating the sensitivity of the test suggest that its sensitivity lies between 52% and 100%, with median values of 80% and 93.5% for standard and severe interpretations, respectively. In those studies that used the same concentrations of bovine and avian tuberculin and interpreted the comparative skin test results as in the current bovine TB control programme in UK, the estimated sensitivity lay between 75% and 95.5% at standard interpretation. In other words, a thoroughly</p>
<p><b>A2.</b> No. The current skin test for bovine TB is an effective test. It is the accepted standard laid down in both national and international legislation for determining the existence of infection in a cattle herd. Studies have shown that the test is on average 80% sensitive at standard interpretation rising to 93.5% sensitive at severe interpretation.</p>	

	<p>performed comparative skin test can be expected to miss about 5 to 25 in every 100 infected cattle on a single round of testing, although this will vary from herd to herd. Furthermore, when infection has been detected in at least one animal in a herd, further rounds of testing will take place at 60 day intervals (and possibly using a severe interpretation) until no further reactors are identified, all of which will improve the initial sensitivity of the test.</p>
<p><b>Q3. Should skin test positive animals be kept because they have mounted an immune response to the disease and are therefore protected?</b></p>	<p><b>Background:</b> There is no scientific evidence to support this view which runs counter to one of the basic principles of control of bovine TB (and other contagious infectious diseases of animals). Where effective vaccination or treatment are not available (as in the case of bovine TB), early detection and removal of infected individuals before they become infectious to others is essential.</p> <p>The pathogeneses of bovine and human TB infections are not directly comparable. Nevertheless, the experimental findings suggest that a significant proportion of cattle infected with <i>M. bovis</i> could enter a state comparable to the latency defined for the majority of people infected with <i>M. tuberculosis</i>. This would explain, at least partially, why a large proportion of the skin (and gamma interferon) test reactors from herds with culture-confirmed bovine TB, are culture-negative and have no gross visible lesions. Many of these animals are likely to be carrying infection that is not detectable by the culture methods employed and that has the potential to re-emerge at a later date, especially when animals are stressed e.g. by movement to a new herd, high production demands etc. Such cattle, even if not infectious at the time of slaughter, might be so at a later stage if left in the herd. They should therefore be considered as potential disease transmitters that pose a threat to the disease security of the herd.</p> <p>Additionally, experimental models of drug-attenuated primary <i>M. bovis</i> infection in cattle have shown that simulated latent infection confers only a limited degree of protective immunity against subsequent re-challenge. Hence the need for regular herd testing and speedy removal of all skin (and gamma interferon) test reactors.</p>
<p><b>A3.</b> Very definitely not. Such animals are infected and can infect others.</p>	
<p><b>Q4. Is it true the skin test is a good herd screening test but a poor individual animal test for bovine TB?</b></p>	<p><b>Background:</b> Any diagnostic test (not just the skin test) that is applied to individual animals for disease screening will have a better chance of detecting infected groups of animals (herds) than individual infected animals, because one only needs to find a single infected animal to declare a whole herd as infected. Therefore, the sensitivity of a test at the herd level will always be at least as high as its sensitivity at the individual animal level. Herd-level sensitivity (the probability that an infected herd is detected by a screening test) is a function of within-herd prevalence, the number of</p>
<p><b>A4.</b> The skin test is best used as a herd test but has value in controlling</p>	

<p>the spread of disease when used as an individual animal test.</p>	<p>animals tested in the herd, the animal-level sensitivity of the test level and the minimum number of individual-animal positive test results required to designate a herd as infected (one, in the case of TB). Herd-level sensitivity will rapidly increase to its maximum level (100%) as the proportion of tested animals in the herd increases. This relationship holds true even if the within-herd disease prevalence is low to moderate. You are always going to be more likely to detect a herd with a 5% prevalence of infection than a herd in which only 1% of animals are infected. And that is regardless of whether you use skin testing, gamma interferon blood testing, post-mortem examination or culture to screen the herd.</p>
<p><b>Q5. Does tuberculin from different sources give different results?</b></p>	<p><b>Background:</b> A VLA report analysing tuberculins produced between 1 January 2005 and 31 March 2007 by VLA and Lelystad can be found on the Defra website at: <a href="http://www.defra.gov.uk/animalh/tb/pdf/tuberculin-report.pdf">www.defra.gov.uk/animalh/tb/pdf/tuberculin-report.pdf</a>.</p>
<p><b>A5.</b> Analysis has been carried out of the relative performance of both the VLA and Lelystad tuberculins and we are confident that both products are effective and reliable. Both are produced and assayed to the same standard, as part of the European Union licensing procedures.</p>	<p>In August 2006, a report by Defra's Chief Veterinary Officer which looked at the apparent reduction in TB statistics included an assessment of the performance characteristics of the two tuberculins. The report, <i>CVO Statement on the reduction in the number of new TB incidents in GB</i> is available at: <a href="http://www.defra.gov.uk/animalh/tb/pdf/cvo-tbstatement.pdf">www.defra.gov.uk/animalh/tb/pdf/cvo-tbstatement.pdf</a>.</p> <p><u>Sources of tuberculin</u></p> <p>The antigens currently used for the skin and gamma interferon tests are the so-called PPD tuberculins extracted from <i>M. bovis</i> and <i>M. avium</i>. In 1975, PPD production switched from cultures of <i>M. tuberculosis</i> to cultures of the AN5 strain of <i>M. bovis</i>, which was circulating in cattle in England around 1948. Among the 30,000 plus strains of <i>M. bovis</i> from GB that have been typed since the 1970s, none isolated have been recovered bearing the AN5 spoligotype pattern. This raises the possibility that AN5 might not be optimal for the detection of cattle infected by the <i>M. bovis</i> strains currently prevalent in GB. However, analysis by VLA researchers of the genome of <i>M. bovis</i> AN5 has shown that this strain has not suffered extensive gene deletions or lost any major antigens in the course of its extensive in vitro cultures, unlike the BCG vaccine strain. As part of a Defra-funded project (SE3220), VLA researchers identified through gene expression analyses 31 genes that were expressed at lower levels in <i>M. bovis</i> AN5, some of which encoded antigens. However, they concluded that it was unlikely that reduced expression of any of those genes would have significant effects on the potency of AN5 tuberculin. In conclusion, there is no significant evidence to suggest that the AN5 strain itself, its growth methods, or PPD production processes have impacted in an</p>

	<p>adverse way on the sensitivity of bovine tuberculin.</p> <p>The latest findings from project SE3220 (Garcia Pelayo <i>et al.</i>, 2009) indicate that the differences observed between AN5 and field strains are likely to have only a marginal effect on the diagnostic accuracy of bovine PPD and continue to support the use of the AN5 strain as the universal source of bovine PPD tuberculin.</p>
<p><b>Q6. If animals test positive to bovine TB using the gamma interferon blood test but show no visible lesions, are they disease free?</b></p>	<p><b>Background:</b> Failure to detect lesions of tuberculosis by post-mortem examination at the slaughterhouse, or to culture <i>M. bovis</i> in the laboratory, does not imply that a test reactor was not infected with bovine TB. Indeed, in the early stages of this disease it is not always possible to observe lesions during abattoir post-mortem examination, and, due to the fastidious nature of this organism, it is very difficult to isolate it from tissue samples without visible lesions.</p>
<p><b>A6.</b> A positive gamma interferon result indicates the presence of replicating <i>M. bovis</i> organisms. There is evidence that they are more likely to be in the early stages of infection. Therefore, failure to find post-mortem evidence of disease does not mean that the animal in question was free of the infection.</p>	
<p><b>Q7. Negative culture results from the lab must mean no infection is present?</b></p>	<p><b>Background:</b> The success of culture mainly depends on the presence or absence of visible lesions in the samples submitted to the culture laboratory. Due to the fastidious nature of this organism, it is very difficult to isolate it from tissue samples without visible lesions. The culture-positivity rate of samples collected from so-called NVL animals (without visible lesions) is very low (circa 5-6 %), whereas it is relatively high in VL (with visible lesions) animals where it is typically around 95-96%. Therefore, in the first instance, culture success depends on the quality of the abattoir inspection to detect lesioned animals. Whilst it is difficult to obtain precise figures on this aspect, particularly for GB, published data from Australia suggested that 'in a sample of cattle that were reactors to the tuberculin skin test, abattoir inspection failed to detect an estimated 47% of cattle with lesions' (Corner <i>et al.</i>, 1991). Whilst likely that the percentage of animals missed by meat inspection in GB may be lower, particularly when examining reactor animals, it is nevertheless indisputable that a substantial number of lesioned animals will be missed (because lesions are in organ systems that are not regularly examined, very small lesions, single lesions etc).</p>
<p><b>A7.</b> No. Detection of <i>M. bovis</i> by culture is affected by many factors including the sampling process, with visibly lesioned animals giving a greater chance of detecting infection. Animals at early stages of disease and latently infected animals do not present with visible lesions at post-mortem and will result in some animals escaping</p>	

detection.	
<b>Q8. Does the gamma interferon test give a large percentage of false positives?</b>	<p><b>Background:</b> Scientific research has shown that the average specificity (accurate identification of uninfected animals) of the gamma interferon test is 97% - which is only slightly lower than the 99% plus for the skin test. Performance evaluation carried out in a number of countries shows that at the laboratory cut-offs used in GB the gamma interferon test has a sensitivity comparable to or marginally better than the skin test – between 73 and 100%, with a median value of about 87%. Scientific research has also shown that the two tests (gamma interferon and skin test) identify different populations of infected cattle. The gamma interferon test can identify infected animals at an earlier stage in the infection as well as infected cattle that simply fail to react to the skin test. An animal that reacts positively to the gamma interferon test and negatively to the skin test will not, in the vast majority of cases, be a false positive.</p>
<p><b>A8.</b> No. The risk of the gamma interferon test identifying a false positive animal is 3 in 100, this risk is further reduced when the test is applied in a herd known to be TB infected. It is a common misconception that, as 82% of gamma interferon test positive animals do not show post-mortem evidence of TB in the slaughter house or laboratory, they were “false positives”. A failure to find post-mortem evidence of disease does not mean that the animal in question was free of infection.</p>	
<b>COMPROMISING FACTORS FOR TB TESTING</b>	
<b>Q9. Do animals with fluke show a stronger reaction to the skin test and result in false positive reactions?</b>	<p><b>Background:</b> A review of the veterinary literature on this topic provides somewhat conflicting evidence. On one hand, the cattle TB pathogenesis study conducted in GB (Defra project SE3013) reported that skin test reactors and contacts with antibodies to liver fluke (<i>Fasciola hepatica</i>) were less likely to show with evidence of <i>M. bovis</i> infection at post-mortem examination. The effect was most significant in dairy reactors. This finding could be explained by some husbandry practices associated with exposure to liver fluke that are also associated with a factor that retards pathogenesis of bovine TB. It is also possible that liver fluke infestation modulates the inflammatory response, reducing the positive predictive value of the skin test in infected animals. Liver fluke antigens are potent stimulators of T-helper (Th2) responses and prior or concurrent exposure to liver fluke antigens may modulate the cell-mediated response to tuberculin which is the basis for the skin test.</p>
<p><b>A9.</b> There is no conclusive evidence to support this. On the other hand fluke, through compromising immunity might make animals more susceptible to infection and/or might make infected</p>	

<p>animals less likely to react to the skin test (infected animals may therefore be missed).</p>	<p>On the other hand, <i>F. hepatica</i> infestations result in polarization of the host's immune response and generation of Th2 cell-mediated immune responses, which are known to inhibit the Th1 responses detected by the skin and gamma interferon test. In Ireland, Flynn <i>et al.</i> (2007) established an experimental model of co-infection of <i>F. hepatica</i> and <i>M. bovis</i> BCG to examine the impact of liver fluke infestation on correct diagnosis of TB in cattle. They found that the sensitivity of skin and gamma interferon tests was compromised in co-infected animals and that <i>F. hepatica</i> infection altered macrophage function. Their results raise the question of whether <i>F. hepatica</i> infection can affect the predictive capacity of tests for the diagnosis of bovine TB and possibly also influence susceptibility to bovine TB and other bacterial diseases. In summary, this is a hypothesis that merits further investigation.</p>
<p><b>Q10. Does the use of flukicide reduce the reaction to the skin test?</b></p>	<p><b>Background:</b> There is no known biological or pharmacological reason in principle why flukicides <i>per se</i> should interfere with the skin test. Farmers are advised not to give their animals <i>any</i> drugs (not just flukicides and other wormers) in the course a tuberculin skin test (see also question 9).</p>
<p><b>A10.</b> There is no evidence to support this.</p>	
<p><b>Q11. Is TB testing compromised by the presence of Johne's disease?</b></p>	<p><b>Background:</b> Johne's disease, caused by infection with the bacterium <i>Mycobacterium avium</i> subspecies <i>paratuberculosis</i> (abbreviated 'MAP') is a chronic and insidious disease of cattle and other ruminants which is believed to be endemic in the UK and many other countries worldwide. It is well known that exposure of cattle and other animals (including man) to MAP and environmental mycobacteria can cause cross reactivity to components of the bovine PPD tuberculin used in the skin and gamma interferon tests for bovine TB. In particular, this reduces the specificity of the single tuberculin skin test (in the neck or the caudal fold) in TB-free herds infected with (or vaccinated against) MAP.</p>
<p><b>A11.</b> Yes. Exposure to Johne's disease can cause cross reactivity when using the skin and gamma interferon tests for bovine TB.</p>	<p>In the UK and Ireland, however, responses to the test reagent avian-PPD are used alongside the bovine PPD tuberculin in the routine screening test for bovine TB to provide a comparative measure of cattle exposed to non-pathogenic environmental mycobacteria. Hence the higher specificity of the comparative skin test over the single test. The same principle applies to the Bovigam test, where optical density levels of gamma interferon released by white blood cells stimulated with avian tuberculin are subtracted from those measured in blood stimulated with bovine tuberculin.</p>

	<p>We have no direct data on the effect of (MAP) infection on the sensitivity of the comparative skin and gamma interferon tests for bovine TB in GB. Experimental studies in calves pre-sensitised with <i>M. avium</i> subspecies <i>avium</i> (a bacterium closely related to MAP) have shown that raised responses to avian tuberculin in the comparative skin and gamma interferon tests may mask the detection of <i>M. bovis</i> infection, even when the specific antigens (ESAT-6 and CFP-10) are employed (Howard et al. 2002, Hope et al. 2005). In Spain, Aranaz et al. (2006) studied a herd with both MAP and bovine TB infection that was followed up for 3.5 years. The comparative tuberculin skin test, gamma interferon assay and a serological test for MAP were used in parallel. Overall, the skin test detected 65.2% of all animals in the herd that were culture-positive for bovine TB and the gamma interferon test detected 69.6% of them. These percentages are in the lower part of the accepted normal range. Both the skin test and the gamma interferon test were able to detect bovine TB-infected animals in the first part of the trial, but the blood test was the only test able to detect such animals in the last three tests.</p>
<p><b>Q12. What is the TB implication of BVD infection in herds?</b></p>	<p><b>Background:</b> Bovine viral diarrhoea (BVD) is most common in young cattle (6-24 months old). Serologic surveys indicate that BVD virus is distributed worldwide and the virus is regarded as endemic in most parts of the world. A small experiment with five neonatal calves artificially infected with BVD virus and <i>M. bovis</i> BCG was carried out by Charleston <i>et al.</i> (2001). The results showed that infection of cattle with this virus could transiently reduce gamma interferon responses to <i>M. bovis</i> in the two weeks after BVD virus inoculation and resulted in a failure to identify tuberculous cattle. There is therefore some experimental proof of the principle that BVD virus infection could suppress the host's immune response against <i>M. bovis</i>, but it is far from clear that this is a significant issue in normal field conditions. BVD virus infection appears to be widespread in Australia, yet they have successfully eradicated bovine TB in their cattle.</p>
<p><b>A12.</b> It is likely that any infective agent that suppresses an animal's immune response mechanism such as occurs in cattle when infected with BVD virus, will increase the likelihood of establishment and progression of any additional disease such as TB. For instance, concurrent TB infection is frequently seen in people infected with human HIV (AIDS) infection, but there has been limited work to demonstrate a similar risk for cattle with BVD.</p>	
<p><b>Q13. If the skin test for bovine TB can be compromised by other mycobacteria (e.g. avium, microti), is the gamma interferon test compromised in the same way?</b></p>	<p><b>Background:</b> <i>M. avium</i> is widespread in the environment and voles are the natural host of <i>M. microti</i>. The use of comparative antigens increases the likelihood of a positive reaction being true (increased specificity). The gamma interferon test, like the skin test used in the UK and Ireland, is a comparative test and the risk of false positive reactors is reduced by the use, alongside bovine PPD, of avian PPD tuberculin which provides a measure of sensitisation by environmental mycobacteria (see also</p>

<p><b>A13.</b> Yes. In cattle, false positive reactions to the gamma interferon test can sometimes be caused by exposure to mycobacteria other than <i>M. bovis</i>. However, this is minimised by a comparison of the reaction to avian and bovine PPDs (tuberculin) to try to discriminate between reactions due to environmental mycobacteria and <i>M. bovis</i>.</p>	<p>question 11).</p>
<p><b>TB IN CATTLE</b></p>	
<p><b>Q14. Is there a large amount of undetected infection in cattle herds?</b></p>	
<p><b>A14.</b> There is undoubtedly some undetected infection - no test is 100% accurate and not all animals are tested. Despite this, test and slaughter regimes based on the skin test have been successfully used in other countries to control bovine TB where there is no wildlife reservoir.</p>	
<p><b>Q15. Is it true a large proportion of cattle are never tested?</b></p>	<p><b>Background:</b> Using CTS (Cattle Tracing System) data, a descriptive analysis of TB testing coverage in the cohort of the British cattle population that died in 2004 found that 71 to 85% of the cattle included in the analysis appeared not to have been TB tested in their lifetimes (Mitchell <i>et al.</i>, Proceedings of the SVEPM annual conference 2006). However, this study included cattle that had lived through the FMD outbreak of 2001, when the TB testing programme was severely disrupted. The proportion was lower (65%) when the same analysis was re-run in 2007 on a more recent</p>
<p><b>A15.</b> At present, 20% of parishes and 32% of herds in GB are tested every year (the proportion is higher in</p>	

<p>England and Wales). The frequency of TB herd tests (1-4 years) is determined by EU legislation, depending on the incidence of infected herds in a particular area. Herd testing frequencies are reviewed nationally on an annual basis and the proportion of herds and parishes annually tested has been increasing over the past few years.</p>	<p>sample (~100,000 cattle that had died in 2006).</p> <p>Recent policy changes (such as pre-movement testing and zero tolerance of overdue TB tests) reduce the opportunities for high TB risk animals to go untested during their lifetimes. In addition to screening of cattle on farms by skin testing, supplementary passive TB surveillance by the Meat Hygiene Service takes place during the commercial slaughter of cattle.</p> <p>A substantial proportion of the national herd may never be screened for TB before slaughter. Many of these animals are fattening cattle in 3 and 4 yearly testing herds which, by definition, will not be tested (they are unlikely to live long enough) and are unlikely to represent a significant TB transmission risk.</p>
<p><b>Q16. Do cattle become infectious only in the late stages of TB - once they have developed "open" lesions?</b></p>	<p><b>Background:</b> The concept of an "open" lung lesion is predominantly a term from human clinical practice, representing the situation where viable TB bacilli are demonstrated in respiratory secretions (i.e. sputum) during life. All cattle identified as TB reactors can pose an infection risk to other animals, regardless of whether or not lesions are found at post-mortem examination (PME). There is no way of knowing at PME of cattle whether lesions observed in any location were resulting in continuous or occasional shedding of bacteria in excretions or secretions while the animal was alive. Experimental data on pathogenesis of bovine TB indicate that shedding of <i>M. bovis</i> can occur at any stage of the infection process, but that there are phases of more frequent shedding during the early stages of infection, which are likely to be associated with an increased risk of transmission.</p>
<p><b>A16.</b> The evidence is that animals may become infectious – can pass on infection - very soon after they have themselves been infected (perhaps in days). This may be followed by periods when animals are less infectious with intermittent excretion of tubercle bacilli. These animals can eventually progress to clinical cases. Infected animals should be regarded as a risk to others.</p>	<p>The majority of TB lesions in cattle are located in the lymph nodes of the chest and head, with or without demonstrable lung tissue involvement. It is important to stress that the data collected at a cursory PME of reactors is not ideal to assess the status of a bovine animal as an <i>M. bovis</i> excretor. Any reactor with demonstrable signs of <i>M. bovis</i> infection is potentially infectious to other animals and any reactor, with or without TB lesions, is potentially infected, and may become infectious in due course. Whilst it is probably correct to say that all cattle with visible lesions in the lung parenchyma (with similar pathology perhaps to the so-called human "open cases") are a continuous or intermittent risk to other cattle and wildlife, it is not correct to imply that cattle without such lesions pose no such risk. The conditions under which an infected bovine becomes an effective disseminator of <i>M. bovis</i> are not well defined, although there is likely a gradation in the risk of excretion according to the distribution and severity of pathology.</p>

	<p>The possibility that nasal transmission of infection occurs during the early stages of infection cannot be excluded and it has been suggested that all cattle infected with <i>M. bovis</i> have the potential to shed bacilli at some stage during the infection (Neill <i>et al.</i>, 1992). This has been shown to occur sporadically shortly after experimental infection at 20-30 and 80-90 days post inoculation (McCorry <i>et al.</i>, 2005), but not yet in naturally infected field reactors (since the time of infection of these natural cases cannot be determined precisely).</p>
<p><b>Q17. Isn't it pointless to test calves for TB as this is a disease of adult cattle?</b></p>	<p><b>Background:</b> Cattle of any age, including newborn calves, can succumb to <i>M. bovis</i> infection by the respiratory (airborne) or oral (milkborne) route. Congenital infection of unborn calves <i>in utero</i>, although possible, is considered extremely rare in GB and other countries with long-established test and slaughter regimes. Therefore, there is no reason, in principle, why young calves could not be tested for TB and, if infected, identified as test reactors, as it happens on occasions. However, there are two main reasons why calves under 42 days are excluded from the majority of TB tests:</p>
<p><b>A17.</b> Cattle of all ages are susceptible to infection. TB has been successfully diagnosed by skin testing in animals less than 4 weeks of age. Young calves are also at risk through milk borne infection.</p>	<ol style="list-style-type: none"> <li>1. First, it is unlikely for such young animals to be infected with <i>M. bovis</i>. In GB, the rate of skin test reactors increases steadily with age of the animal until it stabilises at about 24-30 months of age. Age in itself does not affect the susceptibility to infection but opportunities for exposure to the bacterium accumulate with time and, once infected, cattle are believed to remain sensitised to bovine tuberculin for the rest of their lives. Therefore, there is an age-dependent risk of contracting the infection (and thus becoming a test reactor).</li> <li>2. Second, even if infected, not every calf undergoing skin testing in the first 42 days of life will be detected because it takes some time (usually a period of 3-6 weeks) to mount a detectable immune response to the skin test.</li> </ol> <p>So, whether or not an infected calf under 42 days of age is detected by the skin test will largely depend on how soon after birth it became infected and any individual variations in the ability to mount a delayed- type hypersensitivity response to tuberculin. Therefore, it is generally considered ineffective to TB test young calves, and this thinking is reflected in the rules for pre-movement and pre-export TB testing of cattle. However, in GB we have traditionally tested young calves in specific high risk situations, such as check tests of herds contiguous to a confirmed TB breakdown, or following disclosure of tuberculous cattle at routine meat inspection, or at short-interval tests of</p>

<p><b>Q18. Why are the genotypes (strains) of <i>M. bovis</i> geographically clustered in GB if the movement of cattle is the major cause of spread of disease?</b></p> <p><b>Why not an even distribution of all spoligotypes or at the very least a spread in keeping with the major movements of cattle?</b></p>	<p>reactor herds.</p> <p><b>Background:</b> The most common <i>M. bovis</i> genotypes in GB show a highly aggregated distribution that is stable over time. This observation provides very strong empirical evidence that, in the high TB incidence areas of the country, wildlife reservoirs of this bacterium are involved in the persistence of infection in the more mobile cattle host. Non-random distribution of cattle movements could also, in very unusual circumstances, generate geographical localisation of genotypes. However, preliminary research indicates that cattle movement patterns are not compatible with single-genotype localised TB "hotspots". Thus the epidemic of bovine tuberculosis in GB may be seen as a series of local epidemics caused by different strains emerging in different areas of the country. Further modelling work is proceeding on this particular subject.</p> <p>Of course, each genotype of <i>M. bovis</i> is, on occasions, isolated outside its traditional core area ("home range") and many of the new TB breakdowns occurring in regions of traditionally low TB incidence can be traced back to movements of cattle from herds in the relevant core area, as was indeed the case during the restocking of herds in the North of England following the FMD outbreak of 2001. However, there is little evidence that cattle breakdowns detected outside the endemic TB areas are generating new "hotspots" of disease.</p>
<p><b>A18.</b> Because most cattle movements are local. Only the main strains are quoted in figures and maps but clustering is shown and there is also some mixing which implies cattle movements are not the major cause of spread in endemic areas but are in low incidence areas.</p>	
<h2>TRANSMISSION</h2>	
<p><b>Q19. Does cattle to cattle contact only account for 1 - 2% of all TB cases?</b></p>	<p><b>Background:</b> It is often very difficult to conclusively determine the precise cause of a TB breakdown in a cattle herd. However, in low bovine TB incidence areas, there is evidence that cattle to cattle transmission could be responsible for around 80% or more of cases. However, the situation is quite different in the high incidence areas of the country where 85% - 90% of all confirmed breakdowns occur. Some herds in these areas are also infected by purchased cattle (several studies have shown around 7% - 16%: Green <i>et al.</i>, 2008 and ISG), but wildlife is a major source of new herd infection and in many counties wildlife may be a more important source than cattle. It is impossible to put precise figures on these possible sources.</p>
<p><b>A19.</b> No. The extent of cattle to cattle transmission varies depending on area and level of infection. There is no evidence to support this theory.</p>	

<p><b>Q20. Can cattle become infected by badgers and their infected excreta only when out at pasture?</b></p>	<p><b>Background:</b> Transmission as a result of direct contact has received relatively little attention in the scientific literature because field observations suggest that badgers avoid grazing cattle. However there is an increasing body of evidence (Garnett <i>et al.</i>, 2002 (a &amp;b); Daniels <i>et al</i> 2003; Roper <i>at al</i> 2003; CSL 2006) to suggest that badgers regularly forage in farm buildings such as feed stores and cattle sheds, where they consume and contaminate feed and may come into direct contact with cattle. A Defra funded study (project SE3029) aimed to investigate the extent of badger visits to farm buildings in TB hotspots in southwest England and to identify the reasons why these occur. The final report can be seen at <a href="http://www.defra.gov.uk/animalh/tb/research/projects.htm">www.defra.gov.uk/animalh/tb/research/projects.htm</a>.</p> <p>Further work has been commissioned (Defra project SE3119) to assess the cost-effectiveness of farm husbandry manipulations to reduce risks associated with farmyard contact between badgers and cattle. This work will complete in 2009 and report in early 2010.</p>
<p><b>A20.</b> No. There is an increasing body of evidence to suggest that badger visits to farmyards and buildings may pose a comparable disease transmission risk to that posed by contamination of grazing land.</p>	<p><b>Background:</b> TB is endemic in the badger population and there is much evidence that it is self-sustaining in the absence of cattle TB. Evidence from the Defra Road Traffic Accident surveys in the 1980s show there are pockets of infection in badgers that at that time were not being transmitted and identified in co-located cattle.</p>
<p><b>Q21. Do cattle regularly give TB to badgers?</b></p>	<p>Studies of bovine TB in badgers at Woodchester Park have shown TB is maintained long-term in a stable badger population without cattle contact i.e. they are a natural self maintaining reservoir (Project SE3032: The long-term intensive ecological and epidemiological investigation of badger populations naturally infected with <i>Mycobacterium bovis</i> – Final Report; and SE3035: estimating badger density in RBCT proactive control areas).</p> <p>ISG findings (Woodroffe <i>et al.</i>, 2006) demonstrate indirectly that cattle may have transmitted TB to badgers. A suspension of TB controls in cattle during the epidemic of Foot and Mouth Disease, which substantially delayed the removal of TB infected cattle, was associated with a widespread increase in the prevalence of bovine TB in badgers in RBCT areas only. However, with the normal cattle TB control programme in place (testing and removal of reactor animals) the transmission of TB from cattle to badgers is a low risk, as cattle are unlikely to be shedding large amounts of TB organisms into the environment.</p>
<p><b>A21.</b> With the routine testing of cattle and reactor removal the transmission of TB from cattle to badgers is a low risk, as cattle are unlikely to be shedding large amounts of TB organisms into the environment. This is only likely in uncontrolled cattle TB situations e.g. during FMD and pre-1930s when a dedicated testing and slaughter regime was not being carried out. The ISG reported an increase in prevalence in both cattle and badgers following the 2001 FMD epidemic.</p>	
<p><b>Q22. Can cattle that stray into a herd for a day cause a TB breakdown in</b></p>	

<b>that herd?</b>	
<b>A22.</b> Generally accepted principles of disease transmission indicate that it is possible that infected, infectious cattle that stray into the herd can infect others almost immediately. Infection in these circumstances is a chance process and while transmission on the first day is possible, it is more likely the longer an infected animal is in contact with other cattle and If this contact is close or in confined spaces (as TB is primarily a respiratory disease). However, it is very difficult to ascribe date or source of infection in a long latent period disease such as bovine TB.	
<b>Q23. Is the requirement for the isolation of reactors really necessary?</b>	
<b>A23.</b> Reactor cattle are infected with <i>M. bovis</i> and thus infectious to other cattle. Development of bovine TB disease may take many months or years but transmission of infection may be immediate (see also question 22). Therefore the strict and immediate isolation of reactors is extremely important.	

### INFECTED BADGERS / BADGER SETTS

**Q24. Can the badgers in a sett be proven to have TB by testing the soil and faeces?**

**A24.** No. Currently there is no validated test and even if one were available detection of *M. bovis* directly from badger excretions is difficult, largely because of the low levels and intermittent nature of excretion of *M. bovis* by infected animals.

**Background:** Polymerase chain reaction, or PCR, is a laboratory technique that can amplify an amount of genetic material (DNA) from a tiny sample to a large amount in just a few hours. The PCR technique can be used to detect the presence of DNA from the disease causing organism in animal tissues, cultures of the organism or the environment. In addition to the difficulties described above, detection of *M. bovis* using the PCR technique is also problematic because of the difficulty in extracting DNA from mycobacteria and the presence of components that slow down the PCR in clinical samples (so-called 'PCR inhibitors'). Between 2007 and 2010, £1.3 million will be invested in work to validate and optimise PCR assays that are aimed at allowing discrimination between *M. bovis* and other closely related species of mycobacteria in environmental samples including soil from badger setts.

Whilst it may be possible to identify areas, such as badger setts, where the organism is present it would not be possible to identify individual animals that were infected or know definitely whether the DNA detected was from *M. bovis* mycobacteria that were viable and infectious. A study by Courtenay *et al.* (2006) found 100% of the main setts in Woodchester Park to test positive for *M. bovis* by PCR on soil samples, and in 16 of the 22 social groups at least one culture-positive badger was detected during the 32 months before environmental sampling. In the other 6 social groups no excreting badgers were detected, despite the presence of environmental *M. bovis* at the sett. However, the only clinical sampling methods currently available to us are insensitive and until the PCR test is fully validated it is not known whether the results included false positives (Courtenay *et al* 2008).

Before any test can be considered for use in TB control policy it is essential that it is robust and fully validated, so that its sensitivity and specificity (i.e. its ability to detect true positive and negative results) are known. Careful consideration of how such a test could usefully be employed to replace, or be used in conjunction with, existing tests will also be needed once its performance has been assessed.

**Q25. Isn't it relatively easy to identify TB infected badgers on the basis of appearance and behaviour?**

**Background:** TB infection is currently confirmed by culture of *M. bovis* from clinical samples or tissues in the laboratory. However, *M. bovis* grows very slowly so culture results can take 6 weeks to several months to come through. Tests on clinical samples are insensitive compared to post-mortem

<p><b>A25.</b> No. It is quite impossible, as with cattle, to identify infected badgers on the basis of appearance and behaviour. Only in the very late stages of disease do animals show clinical signs and these are non-specific and may reflect diseases other than TB.</p>	<p>examination and this itself is only reasonably sensitive if carried out in detail and visible lesions are then cultured. A more rapid test is needed to detect <i>M. bovis</i> both in cattle tissues and in live badgers or badger setts.</p> <p>With the available limited blood test (the Brock test) having to be repeated three times at intervals on individual animals, it is impractical and verging on the impossible to confidently distinguish between healthy and bovine TB infected badgers. The sensitivity of the Brock test based on validated data is 54%, with the lower 95% confidence limit being 49%. If applied three times, the overall sensitivity is therefore 87-90%. So put simply, even if you repeat the test three consecutive times, there is still a 1 in 10 chance that you will be releasing a TB positive animal. There are several tests which are more sensitive but they are not trap-side. Currently there is no sensitive and reliable field diagnostic test for bovine TB in live badgers.</p> <p>Bovine TB is difficult to diagnose in individuals of any species. Most of the tests (clinical signs, blood tests, skin tests, culture, histopathology, PCR and post-mortem examination) are less sensitive and less specific when testing individual animals than is ideal and rely on testing large numbers of individuals as a group (cattle herd or badger social group) in order to increase the sensitivity to acceptable levels.</p>
<p><b>Q26. Is it easy to identify TB infected setts?</b></p>	<p><b>Background:</b> Whilst it may be possible to identify areas where the <i>M. bovis</i> organism is present (by testing soil or faeces around setts) it would not be possible to identify individual animals that were infected or know definitely whether the DNA detected was from <i>M. bovis</i> mycobacteria that were viable and infectious. There is currently no validated test for use in the field. A study by Courtenay et al (2006) found 100% of the main setts in Woodchester Park to test positive for <i>M. bovis</i> by PCR on soil samples, and in 16 of the 22 social groups at least one culture-positive badger was detected during the 32 months before environmental sampling. In the other 6 social groups no excreting badgers were detected, despite the presence of environmental <i>M. bovis</i> at the sett. However, the only clinical sampling methods currently available to us are insensitive and until the PCR test is fully validated it is not known whether the results included false positives (Courtenay et al 2008). See also question 24.</p>
<p><b>A26.</b> No. It is impossible to identify infected setts without the capture of animals from that sett and detailed diagnostic tests.</p>	
<p><b>RESISTANCE / SUSCEPTIBILITY</b></p>	

<p><b>Q27. Are some cattle breeds more resistant to bovine TB than others?</b></p>	<p><b>Background:</b> Genetic variation may be expressed in resistance to infection, in the response to the diagnostic tests, or both. Defra funded a study (SE:3040) to test these hypotheses and the findings are due to be published shortly. Benham (1985) found no evidence of breed differences in susceptibility to <i>M. bovis</i> infection in the UK.</p>
<p><b>A27.</b> There is anecdotal evidence pointing to genetic variation for resistance of cattle to infection of <i>M. bovis</i>. However this has not been properly quantified in the cattle population in the UK and it remains a possibility that such genetic variation exists.</p>	
<p><b>Q28. Do family lines within the same breed have different levels of susceptibility?</b></p>	<p><b>Background:</b> There is anecdotal evidence that certain familial lines of cattle show particular susceptibility to bovine tuberculosis. Petukhov (1981) investigated two cattle farms with 2742 animals in Latvia, where 23% were infected, and noted that some families had 80% of its members infected, whereas others had none. If significant variation exists between familial lines this would not be surprising. In experimental animals, strains of disease resistant and susceptible mice and rabbits have long been recognised and utilised for research purposes. In humans both racial and ethnic variation in susceptibility to tuberculosis has been recognised (O'Reilly and Daborn, 1995).</p> <p>Hypothetically, many mechanisms of non-specific immunity may be effective in eliminating a low dose <i>M. bovis</i> challenge. Mechanisms under genetic influence might be the chemical nature of the bronchial mucus, the efficiency of the muco-ciliary escalator, the number of active non-specific macrophages in the lungs and the destructive efficiency of those macrophages' lysosomal enzymes. Other genetically controlled factors influencing susceptibility to bovine tuberculosis may be behavioural. For example, the animals grazing habits with respect to avoidance of excretory products may be under genetic influence. The amount of social behaviour that might facilitate cattle-to-cattle transmission, or investigatory behaviour towards badgers or their excreta, may also be under genetic influence. Specific mechanisms of immunity will almost certainly be genetically influenced (Phillips 2000).</p>
<p><b>A28.</b> There is no evidence to either support or dismiss this theory.</p>	
<p><b>BADGERS AND BOVINE TB</b></p>	

<p><b>Q29. Are 60% of badgers in 'Hot Spot' areas infected with TB?</b></p>	<p><b>Background:</b> The results of Defra's Road Traffic Accident survey, carried out in Cornwall, Devon, Dorset, Gloucestershire, Herefordshire, Shropshire and Worcestershire between 2002 and 2005, showed badger populations in all of the counties sampled were affected by bovine TB to some degree. On average <i>M. bovis</i> was detected in 15% of badger carcasses i.e. around one in seven. This is similar to that recorded in proactively culled badgers in the RBCT during the same time period (16.6%). An extended post-mortem examination carried out on a sample of 205 RBCT badgers revealed substantially more infected animals (Crawshaw <i>et al.</i>, 2008), approximately double (33%), than did standard post-mortem examination. Therefore, these prevalence values are likely to be under-estimates. This represents a high prevalence of infection.</p>
<p><b>A29.</b> It is not known for certain. Not all badger populations in GB have been tested for bovine TB. However, evidence of <i>Mycobacterium bovis</i> infection was found in all Randomised Badger Culling Trial (RBCT) areas.</p>	
<p><b>Q30. How much cattle TB is caused by badgers?</b></p>	<p><b>Background:</b> The RBCT has shown that culling badgers leads to a decrease of about 23% in cattle herd breakdowns in the culled areas, with a trend to a stronger effect (about 40%) in the central areas (Donnelly <i>et al.</i>, 2007). The question of how much bovine TB in cattle is caused by badgers has not been answered accurately through the RBCT as culling could not be conducted with 100% efficacy. It is unlikely that it will be possible to quantify the relative contribution each species make.</p>
<p><b>A30.</b> One of the conclusions the ISG reached at an early stage was that it was not possible to quantify the relative importance of badgers (and cattle) in transmitting infection. However, it was reported by the iSG at their final open meeting that results from the RBCT showed at least 40% was due to badgers.</p>	
<p><b>Q31. Do badgers infected with TB suffer?</b></p>	
<p><b>A31.</b> Infected badgers are able to reproduce and raise young successfully and live for several years. However, based on knowledge of the pathology and extrapolation from the disease in other species, there is evidence that indicates that the disease</p>	

<p>will have a progressively increasing negative effect on the physical well-being of the badger. This has been documented at Woodchester Park (Clifton-Hadley <i>et al.</i>, 1993).</p>	
<p><b>Q32. Will TB in badgers die out if disease is controlled in cattle?</b></p>	<p><b>Background:</b> Understanding host status is important for determining the role badgers play in perpetuating the disease amongst their own population. A spillover host is one in which the disease agent can persist in the population for a time (i.e. there is some transmission, but it is not self-sustaining), but will die out without an external source of infection. Extensive research has shown that badgers are capable of maintaining infection in the absence of outside infection (e.g. cattle) and therefore act as maintenance hosts.</p>
<p><b>A32.</b> We don't know for certain. Modelling suggests that if disease in cattle is reduced then disease in badgers will also be reduced. On the other hand, there is evidence that TB is a self-sustaining infection within the badger population and once introduced, the infection persists within that species without the need for input from other infected species such as cattle.</p>	<p>It is not known for certain but evidence from the Defra Road Traffic Accident surveys in the 1980s show there are pockets of infection in badgers that at that time were not being transmitted and identified in co-located cattle.</p> <p>During the outbreak of Foot and Mouth Disease in 2001, the majority of cattle TB testing was halted. This provided an opportunity for infected cattle to spread TB to other cattle and, potentially, to badgers. The prevalence of infection in adult badgers increased substantially and a weaker trend was observed in badger cubs across all seven proactive trial areas. A similar pattern in road-killed badgers from the seven counties in which the trial areas were situated confirms that this was not driven by culling itself (Woodroffe <i>et al.</i>, 2006). As the ISG noted, this suggests that cattle to badger transmission may be an important factor in TB dynamics and that cattle controls may influence the chances of reinfection of badgers through their effect on cattle-to-badger transmission.</p>
<p><b>Q33. Are there many more badgers in England and Wales now than in the 1990s?</b></p>	<p><b>Background:</b> Between November 2005 and December 2006 research (Defra Project WM0310 &amp; WM0311) was undertaken on behalf of Defra to provide an estimate of population densities of badgers in selected habitats in regions of south-west England which have a high incidence of bovine TB. These estimates, representing indices of overall abundance, serve as a baseline against which any future changes in population densities can be assessed.</p>
<p><b>A33.</b> This is not known for certain. A national survey in the 1980s estimated that the overall badger population was about 250,000 (Cresswell <i>et al.</i>, 1990).</p>	<p>- <u>WM0311</u>: Using distance analysis, the mean densities of badgers foraging in open pasture (autumn 2006) were estimated to be: Cornwall 2.9 badgers per km<sup>2</sup> (95% confidence limits:</p>

<p>Following repeated surveys in the 1990s estimates of the national badger population were published (Wilson <i>et al.</i>, 1997) indicating a likely increase between the two main studies, when it was estimated at around 300,000. It should be noted that the data are more than ten years out of date. Additionally, the methods used in these surveys differed so it is not possible to directly compare the results of these surveys.</p>	<p>2.1–4.0); Devon 4.3 per km<sup>-2</sup> (3.2–5.7); Gloucestershire 3.3 per km<sup>-2</sup> (2.4–4.6); Herefordshire 1.5 per km<sup>-2</sup> (1.0–2.4).</p> <ul style="list-style-type: none"> <li>- <u>WM0310</u>: Using distance analysis, the mean densities of badgers foraging in open pasture (spring 2006) were estimated to be: Cornwall 4.5 badgers per km<sup>-2</sup> (95% confidence limits: 3.2 – 6.5); Devon 4.1 badgers km<sup>-2</sup> (3.0 – 5.6); Gloucestershire 4.4 badgers km<sup>-2</sup> (3.2 – 6.1); Herefordshire 3.9 badgers km<sup>-2</sup> (2.9 – 5.4).</li> </ul> <p>The densities recorded in the hotspot surveys illustrate the inherent variability of badger abundance across regions within GB - a variability which is equally applicable at a local level – and the difficulty in estimating overall numbers of badgers.</p>
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## BADGER CULLING

<p><b>Q34. Was the risk of perturbation and subsequent effects sustained after proactive culling in the RBCT had stopped?</b></p>	<p><b>Background:</b> The ongoing analysis on the impact of proactive badger culling following the cessation of annual culls was updated in January and August 2008. The analysis concludes that the effect on reducing cattle herd incidence inside the proactively culled areas has continued more than one year after culling stopped (i.e. from summer and autumn 2006 and reports a reduction of 54% (95% CI: 39% to 66% lower) in confirmed herd incidence during this time period. This is based on sufficient data to make it statistically robust. The deleterious effect on cattle herd incidence initially seen after culling in the 2 km ring outside the culled area is reported to be no longer apparent over this timescale. The authors conclude that the borderline significant trend for the beneficial effect to increase over time from the start of culling that was reported in the ISG final report is thus shown to continue and appears to be increasing for at least the two years since proactive culling in the RBCT stopped (Jenkins <i>et al.</i>, 2008).</p> <p>These results affect the main figures in the ISG's Final Report by increasing the overall beneficial effect on cattle herd incidence since culling started from the 23% in the ISG Final Report to about 30% and is statistically significant. The overall deleterious effect is estimated to have fallen from the 24.5% reported by the ISG in June 2007 to around 12% and is now statistically non-significant (subject to further studies commissioned by Defra). The cost benefit analysis of culling will change</p>
<p><b>A34.</b> Initially this appears to be true - the borderline significant trend for the beneficial effect to increase over time from the start of culling that was reported in the ISG final report is shown to continue and appears to be increasing for at least the two years since proactive culling in the RBCT stopped. Further studies are underway to monitor if this effect continues.</p>	

	<p>once more data becomes available, however the issues around practicality of coordinated sustained culling over a wide area would remain. This evidence was taken into account when the Minister made the decision on culling in July 2008.</p>
<p><b>Q35. Did the results of the RBCT demonstrate that reactive badger culling has no role in bovine TB control in GB?</b></p>	<p><b>Background:</b> There has been some debate around the biological plausibility of timing and locations of culls and association with herd breakdowns (Godfray 2004; King 2007). Further examination of the spatial and temporal trends in cattle data associated with the RBCT was the subject of a research call advertised at the end of 2007. Five research projects were commissioned this year to further analyse the RBCT dataset to examine this issue.</p>
<p><b>A35.</b> Reactive, localised culling was stopped in November 2003 as results from the reactively culled areas showed an associated increase in new TB incidents of 22% (95% CI 2.5-45.3% higher) measured from the start of the proactive cull (or 18.9%, 95% CI 5.4% lower – 49.5% higher if measured from the start of the reactive cull) throughout the whole of the reactively culled areas. This led the ISG to conclude that it is highly unlikely that reactive culling, as carried out in the RBCT, could contribute other than negatively to future TB control strategies. The ISG hypothesised that the increase in disease was caused by perturbation of the badger population - culling disturbed territorial behaviour (increased ranging) which thereby increases contact rates between badgers and between badgers and cattle (ISG 2007).</p>	

## VACCINES

<b>Q36. Badger vaccine will not be ready for several years?</b>	<b>Background:</b> Vaccine development has been a priority for a number of years in line with the recommendations in the 1997 Krebs Report. There are currently six Defra research projects underway. Details of all on-going and completed research projects are available on the Defra website at <a href="http://www.defra.gov.uk/science/default.htm">http://www.defra.gov.uk/science/default.htm</a> and <a href="http://www.defra.gov.uk/animalh/tb/research/projects.htm">http://www.defra.gov.uk/animalh/tb/research/projects.htm</a> respectively.
<b>A36.</b> An injectable badger vaccine is expected to be fully licensed in spring 2010. The earliest projected date for the availability of an oral badger vaccine is 2014.	Badger vaccines are further progressed than those for cattle - a three and a half year vaccine field trial to gather safety data and assess efficacy of injectable Bacille Calmette-Guerin (BCG - the human TB vaccine) in badgers, and a project developing oral bait formulations of BCG are underway. An injectable badger vaccine will be the first product from the vaccine research programme. Whilst an injectable vaccine is not regarded as suitable for widespread use, stakeholders have agreed that a small scale project to demonstrate the principle of vaccination could be beneficial. An injectable badger vaccine will be used in a Defra funded vaccine deployment project to assess the viability of injectable vaccination and to support the long-term goal of oral vaccination.
<b>Q37. Isn't it pointless to start a badger vaccination programme before infected badgers are removed?</b>	<b>Background:</b> Although vaccination of infected animals is unlikely to have an effect on these, nor will it be harmful. It is impractical and verging on the impossible to confidently separate healthy badgers from bovine TB infected badgers (see also questions 24 - 26). The BCG vaccine will reduce the risk of uninfected badgers becoming infected but would not offer protection to already infected badgers, nor will it harm them. Even if infected badgers were present in the population at time of vaccination, one would still expect the disease pressure on cattle to reduce over time as infected badgers die off naturally. The typical life-span of a badger is between 3-5 years. Other advantages of starting a badger vaccination programme now are to build farmer confidence in the long term contribution badger vaccination can make to tackling bovine TB and to provide valuable information which can help us move towards the long term goal of an oral badger vaccine, before it is available.
<b>A37.</b> No, there is a good case for starting a vaccination programme even though a proportion of animals are infected. The key objective is to reduce transmission risks – between badgers and from badgers to cattle. Although desirable, there is no need to vaccinate all badgers or stop them becoming infected to have an impact on transmission.	
<b>Q38. Will cattle vaccine ever be</b>	<b>Background:</b> Vaccines based on BCG will potentially make cattle react to the current tuberculin skin

<b>allowed, due to international trade regulations?</b>	test as if they were infected with <i>M. bovis</i> . Without a test to differentiate infected from vaccinated animals (a 'DIVA') cattle from vaccinated herds would be indistinguishable from infected animals and would lose their Officially Tuberculosis Free (OTF) status and would be required to be slaughtered as reactors. Significant changes are required to EU legislation to allow the use of a DIVA test. A cattle vaccine in conjunction with a reliable, EU accepted DIVA test is not expected to be available for at least 8 years. The work by the Veterinary Laboratories Agency on developing a DIVA test has shown some initial promise based on experimentally infected animals. Work is ongoing to validate the test in the field.
<b>A38.</b> The Government is currently investigating the scope and potential timetable for making changes to EU trade regulations which would allow vaccination of cattle against bovine TB.	There is evidence to suggest the Commission would be open to persuasion in the use of vaccines. In particular, the Commission has agreed funding under Framework Agreement 7 for diagnostic and cattle and badger vaccine research. Also the EU Animal Health Strategy for the European Union (2007-2013) mentions the EU moving to a more flexible approach to vaccination. This is in the context of controlling exotic disease outbreaks but again demonstrates the Commission's changing views on vaccination.
<b>Q39. Is vaccination the 'magic bullet' for TB control?</b>	<b>Background:</b> Vaccines for a chronic granulomatous disease such as TB do not work as well as for more acute infections such as leptospirosis due to the nature of the immune response and course of disease in the host. Vaccines will not provide a single answer to the problem of bovine TB. However modelling suggests that they may make an important contribution when used as part of a raft of control measures. The lead candidate vaccines in both cattle and badgers are based on BCG. While BCG vaccination has shown promise in both cattle and badgers, efficacy is unlikely to exceed 80% and may be substantially lower. This does not mean that vaccines are of no use for the control of bovine TB - for badgers in particular as any level of efficacy in reducing transmission will have a positive benefit.
<b>A39.</b> No. Vaccines can only ever contribute to the control of bovine TB where, as for many other disease control strategies, it is a combination of control measures that is most likely to be successful.	
<b>OTHER SPECIES</b>	
<b>Q40. Are other wild mammals a TB risk to cattle?</b>	<b>Background:</b> While small numbers of many mammalian species such as rats have been shown to be able to be infected with bovine TB (Krebs 1997, Defra project SE:3010) most are spillover hosts and there is no evidence that they can transmit the infection to other species or even maintain infection in their own populations. The reasons for this are usually immunological or behavioural (e.g. they do not
<b>A40.</b> The greatest TB risk to cattle in	

<p>wild mammals is from badgers which are the main wildlife host.</p>	<p>develop progressive disease, are solitary species or not in contact with other susceptible species). Previous research undertaken by the Central Science Laboratory and Oxford University (2005) has shown that the only wild mammalian species which act as reservoirs of bovine tuberculosis and thus are a risk to cattle are badgers and some species of deer (see also question 41). Other species may be infected with TB but are end hosts (i.e. do not transmit the disease further).</p>
<p><b>Q41. Are wild deer as much a risk to cattle as badgers?</b></p>	<p><b>Background:</b> Quantitative Risk Assessments commissioned by Defra (CSL 2005) demonstrated that the risk of cattle infection from deer is only likely to be significant if the prevalence of TB infection in deer is high. The indication from research is that the overall prevalence of TB infection in deer (wild, park and farmed) is not high and is estimated to be generally less than 5%. The ecology and behaviour of wild deer makes it unlikely that they would have any close direct contact with cattle. More information about this research can be found on Defra's website at: <a href="http://www.defra.gov.uk/animalh/tb/research/projects.htm">www.defra.gov.uk/animalh/tb/research/projects.htm</a></p> <p>Defra subsequently commissioned a wild deer density and disease prevalence study the results of which were published in November 2008. The study shows that on public forest estate land in the Southwest Peninsula, bovine TB is present at a very low level (less than 1%, except in one area where it is present at 3.8% in fallow deer); in the Cotswolds high prevalences were found in two of the three areas sampled (15.9% and 8.1%), particularly in fallow deer; and in all areas surveyed, fallow deer were the species most likely to have the highest level of infection with <i>M. bovis</i> (Defra 2008). On their own, these data cannot predict the role that deer may play in the current epidemic of bovine TB in cattle; however, it does provide essential, previously missing data for use in ecological disease models for this purpose.</p> <p>Results from the density and prevalence surveys were subsequently used to inform a Quantitative Risk Assessment (CSL 2008) to determine the risk of <i>M. bovis</i> infection posed to cattle from wild deer. The findings of the QRA indicate that wild deer do not currently pose a significant TB risk to cattle. Under current conditions of low to moderate density and bovine TB prevalence the majority of infected wild deer populations in Southwest England and Wales are most likely to act as spillover hosts of <i>M. bovis</i>. More detailed information about this research can be found on Defra's website at: <a href="http://www.defra.gov.uk/animalh/tb/index.htm">www.defra.gov.uk/animalh/tb/index.htm</a></p> <p>It is known that close contact of animals can help spread bovine TB, so any measures which help</p>
<p><b>A41.</b> Wild deer in GB are generally considered a sentinel or 'spillover' host of infection in cattle rather than the source of disease in cattle. Overall TB prevalence in wild deer is low and the ecology and behaviour of wild deer makes it unlikely that they would have any close direct contact with cattle. The key results of a Quantitative Risk Assessment (CSL 2008) indicate that deer are likely to pose less of a TB risk to cattle than badgers throughout most of Southwest England and Wales.</p>	

	<p>avoid this could be beneficial. Maintaining lower deer densities is one option of avoiding close contact. Whilst culling is one option that could be considered, it is not the only possible way of avoiding close contact in high numbers.</p> <p>Wild deer do pose a significant risk to cattle in other countries, especially when the deer in question occur at high densities (for references see Wilsmore &amp; Taylor, 2008). Since 1994, the state of Michigan, USA has recognized a problem with <i>M. bovis</i> in wild white-tailed deer. Strategies for eradication of bovine TB from Michigan wildlife focus on reducing deer population densities to biological carrying capacity and reducing artificial congregation of deer by restriction or elimination of baiting and feeding. While much work remains, substantial progress has been made towards eradication of TB from Michigan wildlife.</p>
<p><b>Q42. Are pigs a dead-end host of <i>M. bovis</i>?</b></p>	<p><b>Background:</b> Historically, and in most countries including GB, domestic and feral pigs are regarded as incidental spillover hosts of <i>M. bovis</i>, which become infected through direct or indirect contact with infected cattle, badgers or deer, their carcasses and excreta. The evidence from Australia, New Zealand and the USA indicates that pigs become infected only when the prevalence of infection in the natural hosts is relatively high and pig populations cannot sustain the infection in the absence of infected cattle or a wildlife maintenance host i.e. the incidence of infection in pigs wanes as it is eradicated from the cattle population. However, more recent pathological and molecular epidemiological evidence has emerged in Spain suggesting that wild boar and semi-feral pigs could be acting as maintenance hosts of <i>M. bovis</i> in parts of that country, particularly where the population densities are kept at artificially high levels.</p>
<p><b>A42.</b> Currently pigs are considered spillover hosts in Great Britain.</p>	<p>In GB we are likely to continue to observe sporadic incidents of porcine TB due to <i>M. bovis</i> infection, on farms where pigs and cattle are raised together and in outdoor breeding-fattening units in those regions where bovine TB is endemic. <i>M. bovis</i> has not yet been reported in the very small British feral pig population.</p>
<p><b>TB CONTROL / ERADICATION</b></p>	
<p><b>Q43. Can TB be eradicated from cattle through extra cattle measures</b></p>	<p><b>Background:</b> EU member states and Australia, that have successfully eradicated bovine TB in their cattle have done so without the presence of a wildlife host of the disease. Other countries where the</p>

<p><b>without addressing the wildlife reservoir?</b></p>	<p>disease is present in wildlife have succeeded in controlling the disease in cattle with varying success by tackling the wildlife population. In New Zealand, a deteriorating bovine TB problem in cattle and deer has been halted and then reversed over the last decade. <i>M. bovis</i> infection in both wild and domestic animal populations has been controlled. This has been achieved by applying a concerted, resource intensive multi-faceted science-based programme including the wildlife reservoir. Reducing that reservoir of infection by removal of possums dramatically reduced the incidence of cattle TB (Tweddle &amp; Livingstone, 1994).</p>
<p><b>A43.</b> In September 2005, the Wilsmore review concluded that the international evidence shows clearly that bovine TB in cattle cannot be eradicated by cattle controls alone when there is a secondary reservoir of infection from wildlife. Thus, on the basis of this evidence, some form of intervention in the wildlife domain is necessary if bovine TB in cattle is to be eradicated. The ISG concluded that the elimination of infection in high risk areas can only be achieved in the very long-term and that this problem is a consequence not only of the failure to remove all infected cattle on some farms, but also reintroduction of infection from wildlife (see also question 32).</p>	<p>The ISG used a simple model (Cox et al, 2005) to summarise the TB epidemic in two species, cattle and badgers, either being capable of infecting the other. The implication from this model is that the current TB epidemic can be controlled by either increasing testing frequency, by using a diagnostic method which increases effective testing sensitivity, or by a combination of both. However the epidemic will be reduced by these means only where it is driven by infection from cattle to cattle.</p> <p>On the other hand, the Government's former Chief Scientific Advisor, Sir David King noted in his report that badgers are a clear source of infection for cattle and that TB control will require interventions that reduce the prevalence of disease in both cattle and wildlife. It is likely that the value for each transmission route (cattle / badger) varies from one region of GB to another, in which case the contribution of badger removal to TB control will also vary. However, it is clear that any badger measures must be applied alongside continued cattle controls if the best results are to be achieved</p>
<p><b>Q44. Can tuberculin testing and slaughter of cattle eradicate the disease in cattle?</b></p>	<p><b>Background:</b> The systematic application of tuberculin skin testing and slaughter programmes over extended periods, along with other cattle controls, has eradicated bovine TB from most industrialised countries where cattle are the sole maintenance host of infection. Examples of this are: Australia, the majority of the 50 states in the USA, most provinces and territories of Canada and 11 of 27 EU Member States.</p>
<p><b>A44.</b> Yes - where there is no transmission from wildlife to cattle.</p>	
<p><b>Q45. Did the gamma interferon test make a significant contribution to the eradication of bovine TB in Australia?</b></p>	<p><b>Background:</b> The gamma interferon assay was not routinely applied in the eradication of bovine TB from Australia (Eradication of Bovine TB from Australia; key management and technical aspects. CSL Veterinary Ltd, Cousins <i>et al.</i>, 1998) The gamma interferon test was used in the latter stages only and by the time the test was developed most of the residual infection was located in extensive herds in Northern Australia remote from diagnostic labs. The main control measures used in the</p>

<p><b>A45.</b> The gamma interferon test was introduced into the programme at a late stage and did not make a significant contribution.</p>	<p>eradication scheme were the tuberculin skin test (single caudal fold test) used in repeat herd testing and in most cases in the later stages, disease was eradicated by depopulation. Movement and trade restrictions were imposed on infected herds and areas, slaughter out of the feral buffalo reservoir and use of radio tracked Judas cows to locate stragglers in extensive grazing areas. Incentivised slaughterhouse monitoring for TB lesions was also used. The eradication scheme was run by Government in partnership with the cattle industry with clear strategic aims signed up to by all involved.</p>
<p><b>Q46. Is pre-movement testing a waste of time and money?</b></p>	<p><b>Background:</b> Cattle to cattle transmission is a serious cause of disease spread which is substantiated by scientific evidence (see <a href="http://www.defra.gov.uk/animalh/tb/pdf/prmt-litreview.pdf">www.defra.gov.uk/animalh/tb/pdf/prmt-litreview.pdf</a> ). Ascertaining the disease status of an animal prior to movement using the tuberculin skin test and only permitting movement of those that test clear (i.e. disease is not detected) will reduce the number of cattle with bovine TB that are moved within the country and in turn the risk of disease spread. New TB incidents are being prevented by pre-movement tests and infection is being picked up earlier in high risk herds Furthermore, the obligation to carry out pre-movement tests discourages what was common practice of moving cattle prior to a routine herd surveillance test, so fewer cattle should be escaping Government funded routine surveillance tests.</p>
<p><b>A46.</b> No. Pre-movement testing helps to reduce the risk of spreading bovine TB through cattle movements, especially to areas that are currently free of disease.</p>	
<p><b>Q47. Isn't TB in cattle just an economic problem - not an animal health one?</b></p>	<p><b>Background:</b> Bovine TB is GB's biggest endemic animal health issue, costing the taxpayer around £80 million in 2007/08 (surveillance, research, testing and compensation). Despite recent increases in cattle herd breakdowns this has not been mirrored by an increase of bovine TB infection in humans. The introduction of milk pasteurisation (1930s) and systematic culling of cattle that react to a skin test has virtually eliminated <i>M. bovis</i> infection in humans in the UK. Cases of human TB caused by <i>M. bovis</i> do occur occasionally in the UK and elsewhere, but the majority are attributable to reactivation of latent infection in older people or infection contracted abroad. The current risk posed by bovine TB to human health in the UK is very low. The overwhelming cost of bovine TB to society is directly attributable to the cost of controlling the disease in cattle and associated research.</p> <p>As long as infection is detected at an early stage - as it is almost always under the current testing regime - very few animals are affected by the clinical disease. In the absence of significant transmission to humans the question has been raised whether controlling bovine TB should be justified only in economic terms of reducing losses in animal productivity (Torgerson &amp; Torgerson, 2008).</p>
<p><b>Q48. Does a badger vaccine against</b></p>	<p><b>Background:</b> Although the development of vaccines would provide a significant contribution to the</p>

<b>bovine TB offer the best prospect of eradicating TB in the UK?</b>	control of bovine TB, it must be noted that it will not provide a single answer to the problem but would need to be used in conjunction with other control measures. This is because vaccination is not 100% effective in terms of protection so would need to form part of a package of measures. The use of badger vaccination as another tool is supported by the ISG (and Godfray) which concluded that use of a vaccine for badgers that might reduce transmission of infection and the risk of infection of cattle, thus providing another control option.
<b>A48.</b> Yes – when used in conjunction with cattle control measures. Bovine TB is unlikely to be eradicated from the UK unless the secondary wildlife reservoir is addressed and badger vaccines currently offer the best prospect for tackling this (see also question 43).	
<b>Q49. Is the UK an OTF country?</b>	<p><b>Background:</b> An OTF herd is one where, i) all animals (over 6 weeks old) are being routinely tested in accordance with the correct intervals for the herd; and ii) in infected herds where all the bovine animals have reacted negatively to at least two consecutive routine tests. Where a positive reaction is detected or suspicion of TB is found at routine meat inspection of slaughtered cattle, the herd will cease to be regarded as TB free for a period and will have to undertake a series of herd tests.</p> <p>EU Council Directive 64/432/EEC defines an officially TB free (OTF) country or region as one in which the percentage of herds with confirmed TB breakdowns has not exceeded 0.1% per year and at least 99.9% of its herds have achieved OTF status each year for six consecutive years. Because of the herd incidence of bovine TB, no country/region of the UK (England, Northern Ireland, Scotland or Wales) is currently (or has ever been) designated as OTF by the European Commission. However, most herds in UK are considered OTF at any particular time and so are able to trade freely and export live cattle to other EU Member States, provided that those animals have received a tuberculin skin test with negative results in the 30 days before the date of export.</p>
<b>PUBLIC HEALTH</b>	
<b>Q50. What is the public health risk of TB in cattle and other species in the UK?</b>	<b>Background:</b> In developed countries TB in humans arises principally from infection with <i>M. tuberculosis</i> , which is generally transmitted from person to person through the air by sneezing or coughing. <i>M. bovis</i> infections in humans are rare. In the 1930/1940's large numbers of people were infected with TB. At that time it is estimated that approximately 2,500 deaths a year and 50,000 cases

<p><b>A50.</b> For the majority of the population, the risk of people contracting TB from cattle in Great Britain is considered very low. At present, less than 1% of all confirmed cases of TB in humans are due to infection with <i>M. bovis</i>. The majority of these cases are considered to be due to reactivation of latent disease contracted before widespread milk pasteurisation or from infection contracted abroad. Somewhat greater risk in some occupations where there is direct exposure to infected animals.</p>	<p>of illness in humans were due to <i>M. bovis</i> infections.</p> <p><i>M. bovis</i> infections in animals are transmissible to humans through inhalation of infectious aerosols, ingestion of unpasteurised dairy products or, less commonly, by contact with broken skin. The risk to the general public has decreased significantly due to an extensive cattle testing and slaughter programme, almost universal pasteurisation of the drinking milk supply and veterinary inspection of cattle carcasses at slaughterhouses.</p> <p>The HPA (through the local CCDC) closely monitor human cases of TB caused by the <i>M. bovis</i> infection in the UK (and related bodies in Wales, Scotland and Northern Ireland). The numbers of cases identified remain consistently low, at less than 50 new cases a year. This represents between 0.5% and 1% of the approximately 8,000 culture positive cases of human TB diagnosed in the UK every year. This relative incidence of human <i>M. bovis</i> infection in the UK is in line with that of other industrialised countries with long standing bovine TB eradication schemes. The disease can in most cases be successfully treated with antibiotics. Provisional data show 31 cases, in Great Britain, of bovine TB in humans in 2006, and 27 cases in 2007. This is similar to the situation reported in the vast majority of developed countries.</p>
<p><b>Q51. Does raw milk give you immunity against bovine TB?</b></p>	<p><b>Background:</b> Zoonotic TB was formerly a far more common disease in the UK human population, usually transmitted to man by consumption of raw cows' milk. <i>M. bovis</i>, the bacterium that causes bovine TB, is killed by normal pasteurisation. Disease due to human <i>M. bovis</i> infection usually occurs as a result of reactivation of previously acquired infection in older patients, in whom drinking unpasteurised milk in the past is the probable source of infection, or as a result of infections acquired overseas by immigrants to GB (SE3017). Since 1990, only one case has been documented in the UK of confirmed, indigenous human <i>M. bovis</i> infection recently acquired from an animal source.</p> <p>Cattle herds that produce cow's milk for sale raw in England and Wales i.e. unpasteurised, are subject to more regular bovine TB tests than other herds, which should reduce the risk that infectious cattle are present in the herd. No unpasteurised milk is sold in Scotland. The EU's consolidated Food Hygiene Regulations (in effect from 2006) consolidate legislation relating to milk. Milk (raw or pasteurised) from any animal showing a positive reaction to a TB test cannot be used for human consumption. Milk from the rest of the herd may continue to be sold for human consumption in England and Wales but only if it is heat-treated.</p>
<p><b>A51.</b> No. Unless milk is pasteurised it is possible that it could be a source of infection.</p>	

<p><b>Q52. Are TB infected camelids ( llamas and alpacas) a significant public health risk?</b></p>	<p><b>Background:</b> Bovine TB is not a major health problem with camelids in comparison to cattle, but these species do occasionally develop the disease. Although reports of infection in their natural habitat in South America are few, cases have been diagnosed in llamas and alpacas in New Zealand, the USA and in Great Britain. <i>M. bovis</i> infections in camelids, as in other mammals, are zoonoses (i.e. infections that are naturally transmissible between animals and humans). One of the potential mechanisms of transmission between camelids and man could be through aerosols generated if an infectious camelid “spits” while being handled by a person. Owners and keepers of these animals need to be aware of the associated public health risks (which are not negligible). Camelids are spillover hosts to <i>M. bovis</i> and the prevalence of infection in these species is low compared to cattle and badgers in the traditionally endemic TB areas of GB.</p>
<p><b>A52.</b> There is a low risk to the public in general but many owners of these animals are not aware of the zoonotic risks associated. Camelids are not regularly tested for TB compared to cattle. Educating owners and making them aware is something that needs to be taken forward by both industry and Government.</p>	
<p><b>HUSBANDRY AND BIOSECURITY</b></p>	
<p><b>Q53. Will supplementing cattle feed with trace elements and/or selenium prevent a TB outbreak?</b></p>	<p><b>Background:</b> Much of the soil in the UK is deficient in one or more minerals, and deficiencies of copper, selenium, cobalt and iodine can occur in farmed animals. Mineral supplements for cattle are desirable to help alleviate this, where it occurs. Some evidence also exists that trace element deficiencies can result in impaired immune responses.</p> <p>The association between <i>Mycobacterium bovis</i> infection and trace elements such as selenium, copper and vitamin B12 status of cattle was investigated as part of the Defra funded project “Pathogenesis and diagnosis of tuberculosis in cattle – complementary field studies” (project SE3013). The report concluded that lower selenium status might increase susceptibility to <i>M. bovis</i> infection and there might be an association with copper. However, given the design of the study and the evidence that the action of some micro-nutrients can be substantially influenced by the levels of others it was not possible to conclude that the associations observed were factors in the incidence of bovine TB in cattle. The full report can be downloaded from Defra's TB web pages at; <a href="http://randd.defra.gov.uk/Default.aspx?Menu=Menu&amp;Module=More&amp;Location=None&amp;ProjectID=9317">http://randd.defra.gov.uk/Default.aspx?Menu=Menu&amp;Module=More&amp;Location=None&amp;ProjectID=9317</a>.</p>
<p><b>A53.</b> No. Whether or not there is a possible relationship between trace element supplementation and decreased susceptibility to infectious diseases such as bovine TB has yet to be proved. Deficiencies of trace elements should be corrected as a matter of good husbandry practice.</p>	

	<p>Case-control studies TB99 and CCS2005, carried out as part of the Randomised Badger Culling Trial which ran from 1998 to 2006, also attempted to identify risk factors associated with TB herd breakdowns. Whilst it was not possible to identify specific risk factors which if addressed would confidently result in reduced transmission of disease to and from cattle, the study did support the application of broad principles of biosecurity which includes taking greater care with feeding practices and providing cattle with a balanced nutritional diet.</p> <p>Whilst occasionally a clear cause and effect relationship can be demonstrated by epidemiological studies, in most cases the situation is more complex and the research tells us what factors are important concerning a specific question or a theoretical level of risk associated with a particular event, behaviour or contact. This said, the need for further research to investigate the relationship between trace element deficiency and susceptibility to TB is being considered by the Defra TB Science Advisory Body.</p>
<p><b>Q54. Do cattle only become infected by badgers through close contact? Close the barn doors, put up electric fencing around silage clamps and you will resolve the problem...</b></p>	<p><b>Background:</b> We know transmission of bovine TB occurs from cattle to cattle; from badgers to cattle and cattle to badgers; and badger to badger. There are practical steps farmers can take to reduce the risk of transmission from badgers to cattle. Adopting husbandry best practice on farm to minimise, as far as possible, the risk of contact between cattle and badgers is advisable to reduce the risk of experiencing a herd breakdown. Defra has produced advice on husbandry and biosecurity best practice in partnership with the Bovine TB Husbandry Working Group. The advice includes details of low cost measures: <a href="http://www.defra.gov.uk/animalh/tb/abouttb/protect.htm">www.defra.gov.uk/animalh/tb/abouttb/protect.htm</a> The Husbandry Group did not think it would be practical to fence off entire farms, however fencing off specific fields or buildings could be useful in some cases. Research is currently being carried out into the cost and practicality of husbandry measures to reduce both indirect and direct contact of badgers with cattle (SE3119).</p>
<p><b>A54.</b> TB is mainly a respiratory disease, caught by breathing in the bacteria and direct transmission can occur through, for example, nose to nose contact. However, there is also evidence that indirect transmission is possible, for example through contact with infected saliva, urine, droppings, pus from TB abscesses etc. It is difficult to identify the relative importance of each route of transmission of the disease and for this reason emphasis should be put on</p>	

<p>efforts to reduce the risk of cattle and badgers coming into both direct and indirect contact.</p>	
<p><b>Q55. Does growing maize increase the risk of a TB breakdown in your herd?</b></p>	
<p><b>A55.</b> There is anecdotal evidence that badgers are attracted to maize and maize silage and in areas where maize is grown it often forms a major part of their diet, but there is no evidence to suggest that reducing the amount of maize / changing from maize to grass silage can reduce bovine TB to an extent that would justify what would be significant changes to farm management practices.</p>	
<p><b>Q56. Does ensiling kill the TB bacterium?</b></p>	<p><b>Background:</b> The objective of project SE3022 was to investigate the ability of <i>M. bovis</i> to survive the ensiling process undergone when grass is conserved for winter feeding to cattle. The increasing incidence of <i>M. bovis</i> infection in cattle has resulted in considerable debate over the routes by which cattle may be infected. It has been suggested that contamination of grass with <i>M. bovis</i>, for example by badgers urinating on the pasture, and subsequent ensiling and feeding to cattle is a possible route of infection. Due to the pathogenic nature of <i>M. bovis</i> and the risk of infection to man and animals it is not possible to carry out such studies using farm scale silage making. Therefore, a laboratory scale version was developed to replicate the farm process and investigate whether this was a possible source of mycobacteria for cattle. An experiment was designed in which a laboratory scale version of the ensiling process was developed to take account of the safety issues arising from the use of a human pathogen, at the same time allowing investigation of the ability of <i>M. bovis</i> to survive the ensiling process. <i>M. bovis</i> was recovered for twenty four hours from inoculated grass undergoing the ensiling process. <i>M. bovis</i> was not recovered from grass that had undergone the ensiling process for periods of 6 and 12 weeks. However, the results must be qualified by finding that recovery of <i>M. bovis</i></p>
<p><b>A56.</b> Research by VLA from 1999-2000 (Project SE3022) has shown that the ensiling process does, with time (6-12 weeks), kill the <i>M. bovis</i> bacterium. As with the effect of all such processes on bacterial survival, the longer that the organism is exposed to the hostile acidic conditions in silage, the higher the proportion that will be killed or rendered non-viable (McCaskey and Wang, 1985).</p>	

	from the 24 hour control was at a very low level compared to that of the inoculate used to prepare the grass sample. This indicates that the sensitivity of the recovery process requires further investigation to determine if the findings are real or a result of the low sensitivity of the recovery method.
<b>Q57. Do iron rich soils cause bovine TB in cattle?</b>	<b>Background:</b> A theory has been proposed that <i>M. bovis</i> bacteria survives and proliferates in iron rich soils thereby causing TB outbreaks in hotspot areas of the country where old mine spoils are known to exist. It is claimed that previous scientific research (Johnson-lfearulundu and Kaneene, 1997) supports this theory. The results of these studies where lime was spread on farms in Michigan suffering from high rates of mycobacterium infection, concluded that lime treatment (which reduces iron availability) had reduced infection of cattle after a three year period had passed. However, the studies were designed to look at the paratuberculosis strain of mycobacterium, not bovine TB, and as such are not scientifically rigorous enough to support this theory for bovine TB.
<b>A57.</b> No. Iron rich soils have not been shown to have a causal role in bovine TB.	
<b>Q58. Is there a risk from spreading slurry on land used by cattle?</b>	<b>Background:</b> Slurry has the potential to spread bovine TB via two routes: ingestion (via the pharynx and gut) and respiratory (via the lungs); however, in order to do this, the slurry must first contain viable <i>M. bovis</i> organisms in sufficient quantity and/or be presented as an infectious aerosol. For slurry to be a source of infection for bovine tuberculosis, at least one animal in the herd must be shedding <i>M. bovis</i> in faeces, urine or coughed-up sputum. In the areas where bovine TB is most prevalent, annual testing reduces the likelihood of cattle having time to develop these lesions before they are detected and slaughtered.
<b>A58.</b> Yes - slurry has the potential to spread bovine TB but this is highly unlikely under the conditions existing in the UK as a result of current cattle controls. The risk is mitigated by the dilution effect of slurry , the pH and the storage process, plus spreading on land and exposure of organisms to the environment.	The infected slurry must contain (i) an <u>infectious</u> dose of (ii) <u>viable</u> <i>M. bovis</i> and these must (iii) <u>come into effective contact</u> with at least one (iv) <u>susceptible</u> animal via the respiratory system or the gut. In order to do this, it must survive storage and the environment, either on or in the ground, or in the air as an aerosol – slurry spreading techniques commonly used produce small droplets rather than aerosols and these fall out of suspension in the air within a few hundred metres. These droplets are not small enough to enter bronchi and would have to be swallowed to reach a site of infection, which requires a far higher dose. Very small droplets (true aerosols) which are small enough to enter bronchi are very hostile to bacterial survival and bacteria are less likely to survive to transmit disease.
<b>Q59. Is cleansing and disinfection (C&amp;D) of buildings/yards used by reactor cattle a waste of time?</b>	
<b>A59.</b> TB infected cattle can shed <i>M.</i>	

<p><i>bovis</i> bacteria in faeces, urine and in coughed-up sputum. C&amp;D is a key part of TB risk reduction and in the control of other infectious diseases.</p>	
<p><b>Q60. Are newly calved cows more prone to give a false positive reaction to a TB test?</b></p>	<p><b>Background:</b> A paper by Buddle <i>et al.</i> (1994) looked at TB in pregnant cows after experimental, intratracheal, <i>M. bovis</i> infection. To quote the abstract directly: 'Pregnancy did not appear to affect the susceptibility to <i>M. bovis</i> infection, and immune responses of the cattle in this group at the end of the study were similar to those in the high dose non-pregnant group '. In short, in this research project, the test responses were not significantly different between pregnant and non-pregnant cows (certainly not higher in the pregnant ones shortly due to calve).</p>
<p><b>A.60.</b> The suggestion that heavily pregnant or newly calved animals are prone to react positively to the gamma interferon test is not supported by scientific evidence.</p>	<p>It is important that all potentially affected animals are removed from the herd once TB is diagnosed. Any other approach would risk leaving infected animals in the herd to spread the disease, thus perpetuating the problem and, ultimately, resulting in the need to remove a greater number of animals at a later stage. Positive reactors to a gamma interferon test are infected and therefore must be removed from the herd for disease control reasons.</p>