Published in the Veterinary Times on 25th July 2005 Vol 35, No 27, pp 14 - 17

Tuberculosis: tracing the dilemma

Whilst tuberculosis was first found in badgers 34 years ago it is in fact quite understandable that the question is still asked as to whether badgers do spread TB to cattle. The political discomfort that surrounds this problem and the action necessary to address it is such that the politicians have done their best to wish it away. Misinformation about the subject now almost outweighs the truths. The purpose of this article is thus to set out the facts behind this remarkable dilemma.

The discovery of tuberculosis in badgers (*Meles meles*) due to *Mycobacterium bovis* in Britain was made by Muirhead in 1971. He found classic TB lesions in a badger found dead in the field, whilst investigating an outbreak of tuberculosis in yearling cattle on a farm in the Cotswold Hills in Gloucestershire (**1**). But examination of greater numbers of infected badgers found the lesions to be more subtle. The assertion by Robert Koch (1884) in his studies of disease in different animals "that the disease appears under a different aspect in each species" has proved fully apposite in the badger story.

Cattle like man react to the invasion by the tubercle bacillus by producing large purulent, caseous and mineralised lesions, the classic "cold abscesses" of tuberculosis. But the badger responds by producing small cellular nodules initially often difficult to detect. Only in the more advanced stages do the lesions become obviously purulent.

So why the difference in the badger ? Not surprisingly it is the consequence of a markedly different cellular response. In cattle after about 30 days post infection a strong hypersensitivity response develops associated with a developing cellular immunity. A vigorous, aggressive reaction ensues with the development of a granulomatous reaction with extensive necrosis becoming caseous, giant syncitial cells and a strong containment fibrosis reaction. In contrast, only a variable, transient, mild hypersensitivity occurs in badgers. Granulomata develop but no giant cells are formed, initially only slight central necrosis occurs and fibrosis is minimal. But whilst in cattle very few bacilli are found in these large destructive lesions the opposite occurs in badgers. As badger lesions mature vast numbers of bacilli can accumulate as necrosis gradually increases (*plates 1to 5*). Occasionally massive colonisation can be seen in the tubules of damaged nephrons giving the appearance of a broth culture growth as shown in plate 5 (**2**). Plates 6 and 7 show the cattle response.

Notable difference

In virtually all species TB is primarily a disease of the lungs and this is so in the badger. But the vigorous hypersensitivity in cattle strongly signals the presence of tubercle bacilli causing florid lesions in the tissues invaded. Thus TB abscesses in the lungs are almost consistently accompanied by abscessation of the drainage nodes. A notable difference in badgers is that lesions in the drainage lymph nodes are seen in the minority of cases. Also lymph node lesions in the badger are mostly relatively small whilst in cattle large granulomatous masses usually involve the entire node in an extensive caseous necrosis with mineralization. This reaction is rare in the badger. Another very significant difference is that in the badger the predilection site for

secondary spread, following generalisation with haematogenous dissemination, is the kidney. Very rarely is this organ affected in cattle, spread usually involving the liver or less often the spleen (**2**).

The significance of the high bacillary counts in badger lesions is all too apparent. With over half of the diseased animals showing lung lesions, heavily infected lung discharges will be coughed up with sputum to contaminate the environment as well as swallowed discharges contaminating the faeces. Kidney disease affords another portal of exit for infection but lesions are more variable with reports of involvement in 15% to 42.5% of diseased badgers (**3, 4**).

Just how this contamination affects other animals sharing the same habitat as the badger will be considered later but first the impact of this disease on the badger population should be considered. When infection is introduced to a sett, probably most often by immigration of an infected individual into the group, it is likely to spread to all occupants as soon as that individual starts excreting.

Badgers are gregarious, fossorial animals and generally only emerge after dusk showing a nocturnal activity pattern. Their days are spent in the confines of underground setts which comprise tortuous tunnel systems expanded occasionally into chambers some used as latrines but most are bedding chambers. Often all the occupants will sleep together in a tangled mass in one of these. The confined airspace, close proximity and the very frequent mutual grooming activity, all encourage spread of TB infection throughout the group. A diseased dam may spread infection readily to her cubs. Like the very young of most species they appear considerably more susceptible and florid disease is more likely (**5**). But in adults the outcome following infection is variable.

TB infection elicits a primary focus, a very small granuloma 0.5 to 1mm diameter, sometimes several and usually in the lungs. In many healthy individuals this may progress no further as a cellular immune reaction completely resolves the infection. In a high proportion of others infection will spread to the drainage node ie. usually the bronchial or mediastinal and no further, forming a primary complex. Since lymph node lesions are relatively small compared to the huge destructive lesions seen in cattle, these badgers will appear as showing no visible lesions (NVL). Whilst variable, about 70% of badgers from which *M.bovis* is isolated may be NVL. These represent latent infections which may remain so for years. But the reverse has also been found with 70% showing lesions (**5**).

Latency

Latency is a feature of TB seen in many species. Partial resolution of active disease was frequently seen in man in the pre-antibiotic era, during rest and improved nutrition at a sanatorium. There is evidence that this phenomenon may also occasionally occur in badgers (**6**). But reversion and activation of latent infection will occur with stress conditions which in the case of the badger may be territorial pressures, social hierarchical pressures particularly in boars and nutritional stress.

Active disease mainly results in an insidious tuberculous pneumonia. Where the natural disease has been observed at a MAFF / DEFRA study site in Gloucestershire

faeces sampling for excretion of swallowed infected lung discharges indicated the time span of development of lung disease to be one year in several animals whilst the longest duration was two years (**7**)
Emaciation appears to develop over the last few months with generalisation and miliary lung disease probably for one to three months or so before death. Badgers are compulsive miners and when chronically sick will tend to cease these exertions. Thus emaciated cases frequently show obviously overgrown claws (*plates 8 to 12*). They will often stay above ground in the late stages of disease near an easy source of food and such animals have quite often been found in farm buildings (**8**).

Bite wounds
The most severe form of disease is frequently associated with bite wounding. Territorial defence particularly by the boars may involve mere skirmishes with neighbouring badgers but occasionally serious fighting with severe bite wounding occurs (*plates13 to 15*). If the attacker has lung disease this is likely to result in bite wound inoculation of infection, abscess development and frequently early generalisation with haematogenous spread to lungs and kidneys. Lesions are usually particularly extensive and more acute in this type of infection. Death may result in a matter of several weeks. Transmission by this method is relatively common varying from 13 to 32% of diseased badgers (**5**).

Deaths due to tuberculosis have been estimated to amount to 5 to 10% of infected populations annually (**9,10**). The main cause of death of badgers is road traffic accidents. But of the natural causes, whilst mis-mothering and starvation are the principal reasons for cub deaths, in the endemic affected areas tuberculosis has been shown to be the single greatest cause of death of adult animals killing about 40% (**9**).

Since it was made a protected species in 1973 badger numbers have increased enormously in most areas including the South West. The population in Britain was last estimated in the 1990 National Badger Survey and was considered to be 300,000 to 400,000. The Survey reported a 77% rise in numbers between the 1980 and 1990 Surveys. The rise since has been at least of this order and taking the lower estimate the current likely total is thus in excess of half a million.

Infected populations will also have expanded and in areas already heavily populated, such as many in the South West, territorial space will be under pressure. Territorial fights which can cause the worst manifestation of TB in badgers are likely to increase and probably have done so already.

Action
So what action was taken following the discovery of TB in badgers and the potentially severe problems that might occur if badgers were hosting this disease? Indeed whilst mainly a problem of the South West of England, investigations over the next 20 years revealed that this infection of badgers was present in 24 of the 61 counties in mainland Britain (**11**).

In all these areas outbreaks of tuberculosis in cattle of obscure origin had been occurring often involving out wintered young stock, under three years, at grass and a high proportion involved stock in closed herds. Open lesions in reactors were rare

with spread within herds being exceptional. About 40 - 50% of outbreaks involved single reactors.

Clear association

Investigations gradually revealed a clear association of infection in the badger with that of cattle. Initially an association of cattle infection with disease in badgers was found in 17 of 30 herd outbreaks (56%) (**8**) but with further investigations this association increased to 74.4% in Gloucester and Avon with 198 of 266 farms so affected (**12**). From 1986 to 1995 it was considered that infected badgers were incriminated annually as the origin of infection in 90% of new outbreaks in South West England (**13,14**).

Whilst an association was established further evidence was needed to determine how significant infected badgers were in the cattle disease.

The Thornbury trial

This was the question that the Thornbury Trial, South Gloucestershire set out to answer in December1975 and which was set up with the agreement of the then Nature Conservancy and MAFF. It took place in a TB hotspot area of 104 Km^2 in which there were 12,000 cattle in 158 herds, in 13 of which (8.2%)TB had been diagnosed at the start of the trial. Prior to the start 130 badgers had been trapped of which 24 (18.6%) were infected with *M.bovis*. Badgers were completely removed using anhydrous sodium cyanate fogging of setts, most during 1976.

Sett activity was used to monitor for badger presence. No activity was seen anywhere in the area by 1980 and in that year for the first time for many years no reactors were detected. Badgers re-colonised from the margins and by 1988 many setts showed activity. But it was not until 1991 that TB was diagnosed in any of the cattle and that in a herd just inside the trial area. Thus there had been a period of complete freedom from TB in cattle of 10 years following the removal of badgers and infection re emerged subsequent to recolonisation of the area with badgers (*figure 1).*

This exercise though draconian demonstrated unequivocally that the badger was the maintenance host in this area. The same exercise was repeated in 1976 in a much smaller area of 12 Km^2 at Steeple Leaze, Dorset with exactly the same results with complete cessation of cases of TB in cattle. But seven years later stock were no longer kept on the group of farms involved as they all went over to arable. Thus the full duration of TB freedom could not be determined.

However, very detailed investigations were carried out into the epidemiology of these outbreaks. Severe disease was present in many of the badgers which during the summer had been dining on grain much of which passed out whole in the faeces. Rats were also found infected probably through eating such faeces. But more significantly as cattle had been tested at two monthly intervals through the years a seasonality to occurrence of reactors was detected in May – June correlating with a peak in cases of disease in badgers some 30 days earlier in Apr – May (**15**).

Gassing, was a technique used to control rabbits for many years, and when used strategically on badgers in hotspot areas in the South West it very effectively more than halved the total of confirmed outbreaks of TB to 90 herds nationally by 1980 over a period of less than five years.

However, use of gassing with cyanide with its distressing connotations resulted in an understandably unpleasant postbag for the then Minister. He set up the first of a total of three independent reviews which have been carried out to evaluate all the evidence as to the badger's role as a source of infection for cattle and propose appropriate plans of control (**16**).

Lord Zuckerman acknowledged badgers were acting as a maintenance host for bovine tuberculosis but queried the use of cyanide gassing. As a consequence of his report the use of this was stopped in 1981.

This policy was then replaced with the "clean ring strategy". On the basis that where present, TB in badgers was at a prevalence of 20% or more, then two or more badgers were trapped at setts using farms where TB outbreaks were confirmed to establish infection status. Those social groups from infected setts were trapped out and the trap and test approach extended centrifugally until clear setts were encountered. Trapping was less efficient than the gassing approach, also lactating females were released but control was maintained and when this policy was stopped in 1986 the total of outbreaks was 88. The last large scale exercise in control was carried out using this technique, the Hartland Trial, North Devon and resulted in a fall in herd outbreaks from 15% in 1984 to 4% in 1985 and improvement was sustained for ten years.

The Minister requested a second independent review which was carried out by Prof. Dunnet, Exeter University and mainly addressed the escalating cost of control and which looked to the promised likelihood of an accurate live test for TB in badgers being available within the next year or two. A new policy termed "the interim strategy" was introduced which downscaled trapping activity to the area of the breakdown farm or simply the field where the reactors were thought to have acquired infection (**17**). This compromise policy was introduced against field veterinary advice and following its introduction the number of herds with breakdowns showed a marked increase. Also the reoccurrence of breakdowns following control measures showed a marked increase.

In the meantime a pilot trial on the use of BCG was carried out at the Central Veterinary Laboratory, Weybridge (now VLA). When challenged vaccinates showed encouraging findings of enhancement of cell mediated immunity and amelioration of challenge infection with virulent *M.bovis* compared with controls. The vaccinates lived longer, shed fewer bacilli and their inoculation sites headed more rapidly (**18**).

But these findings were not followed up and no further work was carried out on this subject in this country. However, in Ireland baited vaccine trials were successfully completed and currently larger vaccine experiments are in progress with a view to extensive field trials.

A live test for badgers based on detection of antibody was eventually developed and trialled in 1994 but proved too insensitive for diagnosis. The trial was stopped in 1996.

The number of outbreaks had reached alarming proportions by 1996 and the interim strategy was stopped as the Minister requested a further review which was carried out by Prof Krebs, Oxford University (**19**). Whilst noting the "compelling evidence" for the transmission of infection from badgers to cattle he considered no action should be taken against infected badgers other than in proposed extensive field trials. These trials were to quantify the impact of killing badgers.

The Krebs trials comprised ten triplets of areas, each of 100 Km^2 of cattle farmland, where differing action against badgers was to be taken. Each triplet had a control area of no action, a proactive area where all badgers were removed by trapping and a reactive area where strategic removals of badgers were triggered by the disclosure of a cattle TB breakdown. The trial was started in 1998 and was scheduled to run for five years. An Independent Scientific Group (ISG) headed by Prof Bourne, formerly IAH, Compton was appointed to oversee the trials.

The start was piecemeal with only one triplet started in December of 1998. The last triplet was started in 2002. Problems of non compliance by landowners and farmers and interference by animal rights campaigners were considerable. Trapping efficiency was reported as being from 30 to 80% efficient (Hansard, MPs Questions). Then in 2001 the outbreak of Foot and Mouth Disease (FMD) halted all action in trials areas. In 2003, after only two full years for some of the triplets, Prof Bourne announced a halt of any further action in the reactive component of the triplets after his group noted a marked increase in herd breakdowns in those areas.

This announcement was badly received and the Minister invited Prof Godfray, Imperial College to examine the ISG data. He concluded the statistical basis for this decision was unsound and the case for stopping action was not valid. But the trials continued with only the control and proactively culled areas (**20**).

In January 2005 the results of the Four Counties Trials in Ireland were published and showed a highly significant effect of proactive culling, over large areas, in greatly reducing cases of TB in cattle compared with the control areas. A 60 to 96% decrease in TB breakdowns was found in the different counties (**21**). These results were in accord with the earlier successful Irish trial in East Offaly (**22**).

With no action taken to control diseased communities of badgers in most of the problem areas the disease base in cattle has naturally increased. This was especially apparent after 2001 when all TB testing was stopped as a result of FMD controls.

Unfortunately the SVS did not insist on permitting restocking only from TB tested herds or post movement testing and the inevitable spread of infection by cattle occurred to hitherto TB free areas.

One such was Cumbria. Purchased stock were the source in 23 affected herds in which reactors involved both the purchased animals and small numbers of the

indigenous stock on five occasions. But in the other 18 only the purchased animals were reactors, thus no spread occurred in about 80% of these cases.

Aftermath

In the aftermath of FMD purchased stock have seeded infection fairly widely but where no badger maintenance is involved TB testing has readily cleared many of the herds. However, the pattern of infection in the endemically infected areas has remained unchanged.

Lateral spread within annually tested herds can occur but usually only at a low level. Still between 40 to 50% of herds have only one reactor. Whilst prior to FMD almost 75% of herds had three or less reactors the situation has since deteriorated considerably with multiple reactors in the ascendancy. But whilst some of these may well be the result of lateral spread more often they appear the result from infection from a single point source and are quite likely to be the consequence of infection by a seriously diseased badger.

Badgers in the final stages of disease are highly infectious becoming "super excretors". These have previously been found frequently associated with multiple reactor outbreaks. Unfortunately now such cases can no longer be investigated as SVS staff have been disallowed to catch and examine badgers for evidence of tuberculosis since 1998. Yet the last data published by DEFRA gave results of examination of 3414 badgers examined during 1997 and 1998 which showed 910 to be infected, an incidence of 26.6%.

Projects to examine other wildlife species for evidence of TB have been sanctioned and alarmingly all five species of feral deer in this country have been found in endemically infected badger areas to have contracted TB. Infection is pocketed with high levels being found in some sites such as in the red deer on parts of Exmoor More alarming are the reports of cases of TB in rural cats, 16 now confirmed. A correlation with badgers has been noted in several, classically with them jointly using outside feeding bowls (**23**). Infection of a dog has now also been recorded and is to be published shortly.

Also alarming is the spill over to pigs with a number of outbreaks now confirmed in the endemically infected badger areas and with several clearly circumstantially linked to badger presence. But investigation of the possible badger link by SVS staff has been disallowed. The first case in a sheep has been confirmed as well as TB in alpacas and a lama. It might seem incredible but cases of *M.bovis* infection have not been seen here in other wildlife, cats and dogs or any of these other species since the early part of the last century.

DEFRA has released the data on outbreaks of TB during 2004 which show a total of 3,343 outbreaks of which TB was confirmed in 1,737 in line with the annual rise of 18% per annum since 1986 which was, incidentally, the last year their policy was based on informed field SVS advice. The TB "eradication" policy has now deteriorated to a point it was at almost forty years ago.

Sadly there has been remarkable inaction about what needs to be done to control this serious notifiable disease which is affecting our native wildlife as well as companion

animals and our farmstock. Great concern appears to be given to what is perceived might upset the voters but if they are presented with the facts would those voters really plump for condemnation of our animals to a worsening eternity of TB ? Possibly when the almost inevitable first case of infection of a household from their cat or dog occurs we will find out. But that will of course be a rural household !

It is because of the above and driven to exasperation by the remarkable ineptitude over the handling of this worsening disaster, that a current total of 420 vets have signed a letter of no confidence to The Secretary of State for DEFRA.

The letter requests her to urgently address the problem by strategic culling of diseased badger communities and widespread vaccination trials of healthy badgers with BCG.

If the fountain head of infection in badgers is stemmed conventional control measures will resolve the cattle problem although infection of other species may be more problematic.

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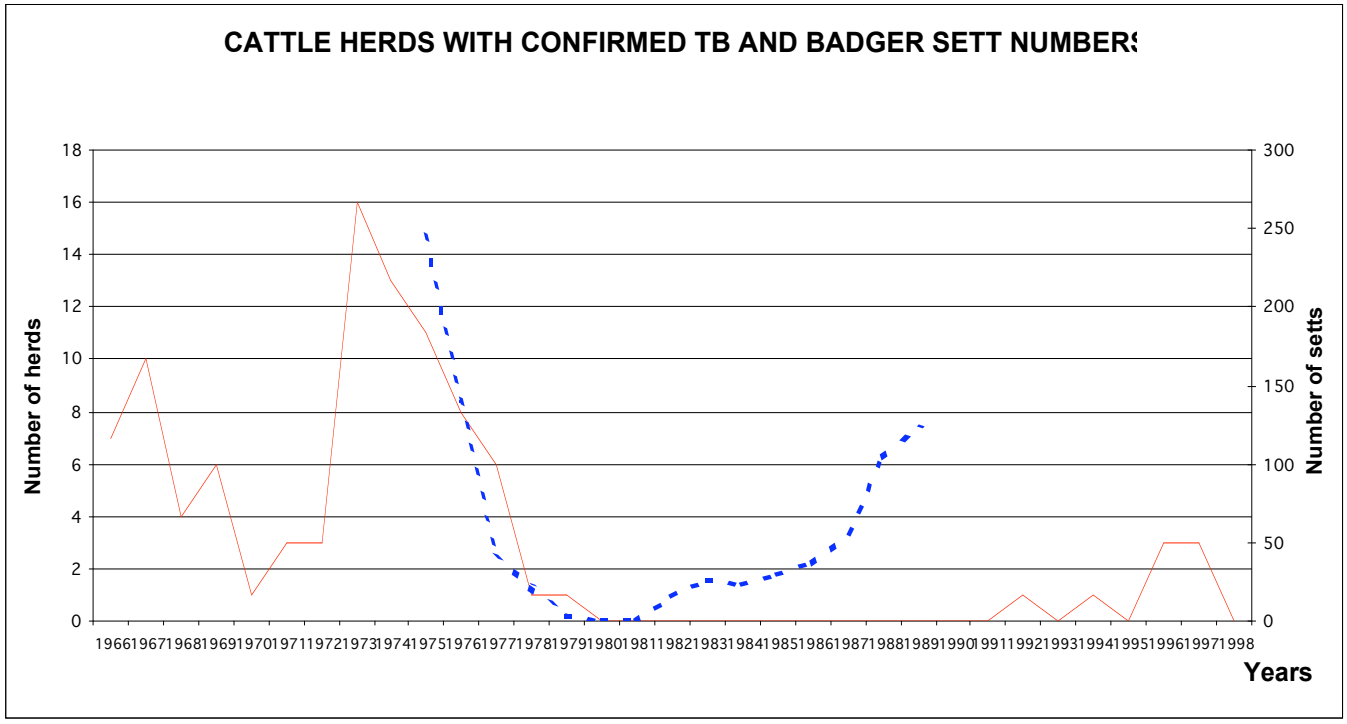
Legends for plates

1.Badger, macrophages packed with AFBs. Stain ZN x1000
2. Badger,mature lung tubercle,note numerous AFB masses throughout, minimal necrosis. Stain ZN x 100
3. Badger, advanced lung tubercle in centre, marked necrosis, surrounded by sattelites, vast numbers of AFBs. Stain ZN x 40
4. Badger, kidney showing complete colonisation of glomerulus with vast numbers of AFBs. Stain ZN x 400

5. Badger, kidney showing broth like growth of AFBs in tubular filtrate. Stain ZN x 400
6. Cattle, section through tubercle showing fibrous capsule, giant cell and inner extensive necrosis. Stain ZN x 200
7. Cattle, inner necrotic area of 6. showing total find of 2 AFBs. Stain ZN x 1000
8. Badger, advanced miliary pneumonia.
9. Badger, tuberculous nephritis, note radiate lesions of necrosis.
10. Emaciated tuberculous badger, 5 Kgs.
11. Emaciated tuberculous badger showing overgrowth of fore claws.
12. Emaciated tuberculous badger showing loss of periorbtal fat
13. Badger showing severe bite wounding neck and rump.
14. Badger showing canine tooth penetrations from a bite.
15. Tuberculous abscess associated with the bite shown in 14.

Figure 1 The Thornbury Trial



△ △
Control started – 12/75 **ended** – 5/81

- - - - *Sett numbers refer to setts considered to be occupied by badgers*