Tuberculosis in badgers; a review of the disease and its significance for other animals

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Introduction

The discovery of tuberculosis in badgers (*Meles meles*) due to *Mycobacterium bovis* in Britain was made by Muirhead in 1971, whilst investigating an outbreak of tuberculosis in cattle on a farm in the Cotswold Hills in Gloucestershire (Muirhead et al.,1974). The ensuing investigations over the next 20 years revealed that this infection was present in 24 of the 61 counties in mainland Britain (Wilesmith, 1991). The first record of infection in Ireland was in Cork in 1975 (Noonan et al 1975) and subsequently infection was discovered in all 26 counties. In Northern Ireland infection was reported in 1978 (Report, 1978). In all these areas outbreaks of tuberculosis in cattle of obscure origin had been occurring and investigations gradually revealed a clear association of infection in the badger with that of cattle. Muirhead et al. (1974) initially found an association of cattle infection with disease in badgers in 17 of 30 herd outbreaks (56%) but with further investigations by Wilesmith et al. (1983), this association increased to 74.4% in Gloucester and Avon with 198 of 266 farms so affected. 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 (Clifton Hadley et al., 1995b; Report 1995). No such association was reported in the original report of the infection in the badger by Bouvier et al. (1951) from the Basle region of Switzerland. These workers had previously found tuberculosis in chamois (*Rupicarpa tragus*) and roe deer (*Capreolus capreolus*). These were considered the probable source of the badgers' infection.

The control of badgers, a statutorily protected species in Britain since 1973, has resulted in a political dilemma. A total of three independent reviews have been carried out to evaluate all the evidence as to the badger's role as a source of infection for cattle and propose an appropriate plan of control (Zuckerman, 1980, Dunnet et al., 1986, Krebs, 1997). All agreed that the badger was a significant source of infection for cattle but proposed differing approaches to control.

The purpose of this article is to review the current state of knowledge of tuberculosis in the badger according to the following aspects.

1. Gross pathology
2. Pathogenesis of disease
3. Immunity
4. Diagnosis of infection
5. Excretion and viability of infected products
6. Epidemiology in badgers
7. Overspill of infection to other wildlife
8. Overspill of infection to domestic animals
9. Control of infected badgers
10. Conclusions

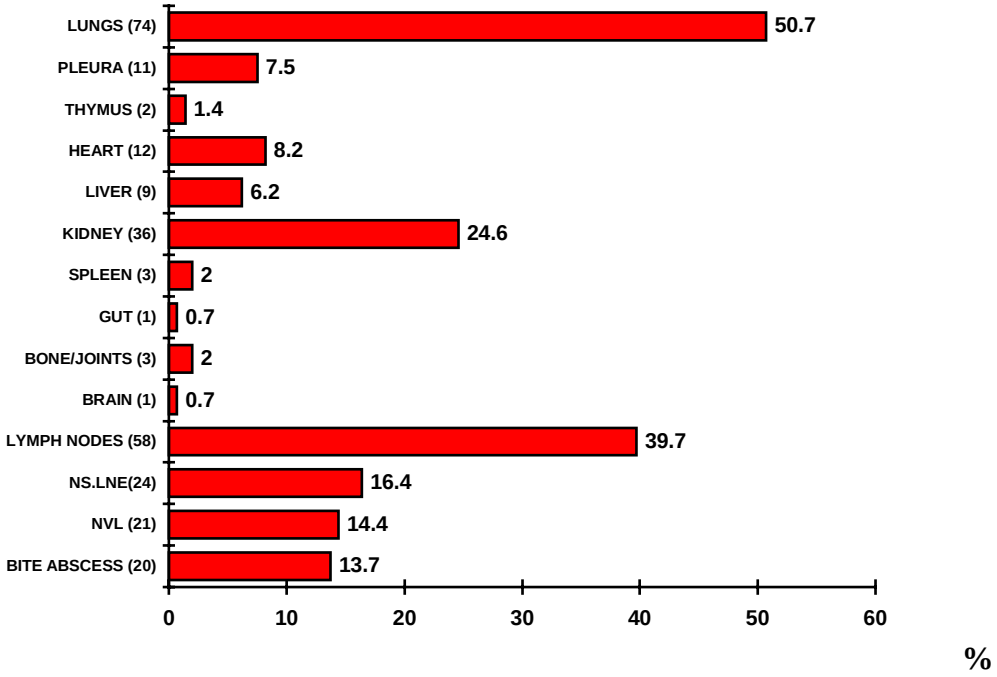
1. Gross pathology

As in most species, tuberculosis in the badger can affect virtually all organ systems. *Figure 1* shows the distribution of gross lesions found in a series of 146 badgers infected with *M.bovis* from Gloucestershire, Avon and Wiltshire and examined during 1973 to 1976 (Gallagher, 1998). No genital or mammary disease cases were seen in this series but two cubs were encountered with sub maxillary lymph node lesions consistent with milk borne infection. Also a single instance of seminal vesicular tuberculosis was reported earlier (Gallagher et al., 1976). However, as is usually the case in other species, tuberculosis, in the badger is essentially a respiratory disease.

In the series of 146 infected animals examined by Gallagher (1998), 74 (50.7%) showed lung lesions whilst a further 9 (6.2%) showed lesions in the broncho-mediastinal nodes thus implying a likely total of lung infections of 57%. Nolan (1991) found a similar frequency of lesions of the lung and its drainage nodes of 54% (39 of 72 cases) in badgers from southern England. In Ireland, Fagan (1993) found lesions at these sites in 46% of infected badgers (13 of 28 cases).

However, in contrast with cattle in which discrete gross lymph node lesions accompany lung lesions in the great majority of cases, (Francis, 1958), in the badger, they did so in a minority, being found in only 36% (27 of 74 cases) examined by Gallagher (1998). Clifton Hadley et al. (1993) found broncho-mediastinal node lesions in half the tuberculous pneumonia cases they examined (20 out of 40).

Figure 1 **A summary of % lesions seen in 146 badgers* infected with *M.bovis***



NS.LNE = *non specific lymph node enlargement.* NVL = *no visible lesions.* ( ) = *number of cases*
**Many badgers showed lesions at more than one site. M.bovis isolated from 134 cases (92%); remainder positive by smear/histopathology.*

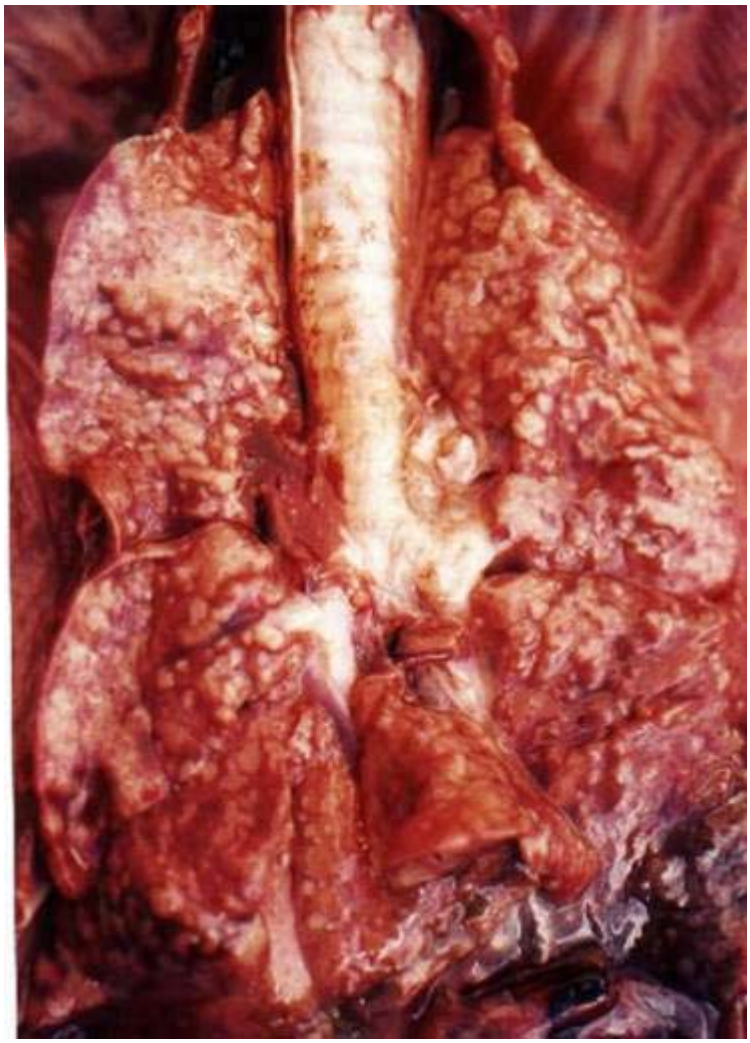


Plate 1: lungs of a badger with advanced fatal lesions of miliary tuberculosis. Note most of the 2 – 3 mm miliary tubercles are coalesced to form large masses. There is virtually no normal lung tissue remaining. This 3 – 4 years old female badger was emaciated weighing 5.4 kg, about half the normal body weight.

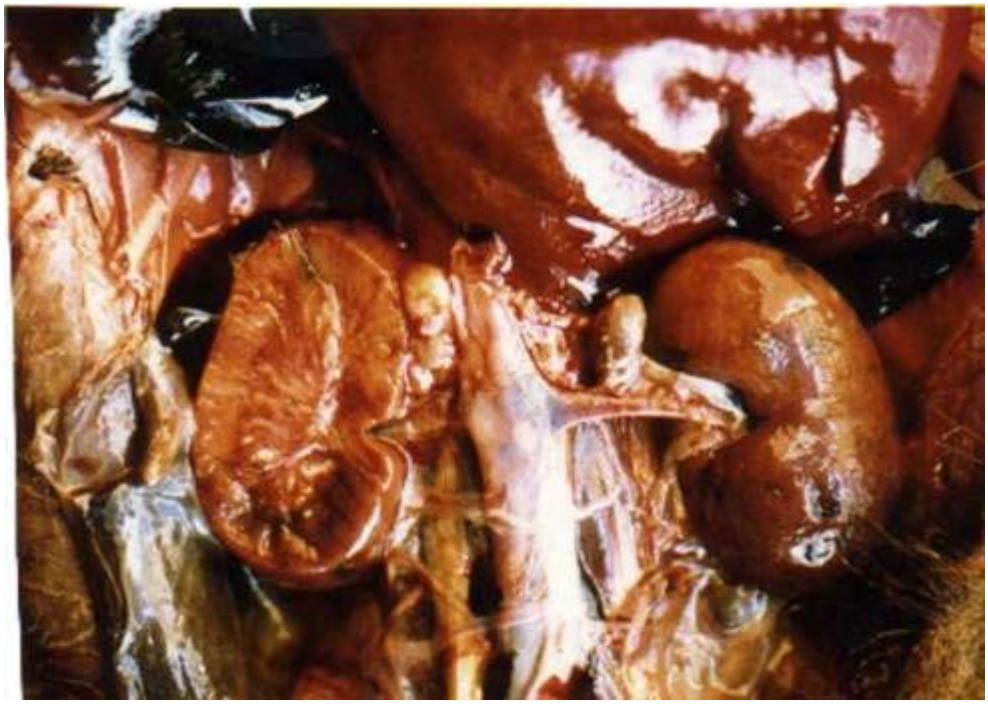


Plate 2: View of the kidneys of the same badger showing advanced disease. Note the radial disposition of necrotic medullary lesions on surface of the kidney which has been cut in half and numerous elongate cortical lesions. Depressed infarcts are present in the uncut kidneyand below it the renal lymph node is visible showing several 1 – 2 mm necrotic lesions. The adrenals are medial to the kidneys and are enlarged to twice normal size.

The spectrum of lung disease in the badger may vary from nodules of 1–3mm, to satellitism with clustering of numbers of lesions around a larger lesion, to widespread lobar disease and finally miliary disease as shown in *plate 1* (Gallagher et al., 1976). These authors also recorded a variant of miliary disease in which large coalescing tumescent lesions were present showing an appearance of a more acute nature which they termed acute tuberculous pneumonia.

Whilst advanced lesions are characteristic, smaller lesions can be confused with those due to adiospiromycosis, lungworms or focal oseous metaplasia (Gallagher et al., 1998).

As shown in *figure 1* pleurisy, either nodular or unilateral pyothorax was found on occasions as was nodular epicarditis and pericarditis. Haematogenous dissemination from primary lesions occasionally resulted in lesions with the appearance of sawdust in liver or spleen. But dissemination most frequently resulted in kidney disease producing characteristic elongate radial lesions as shown in *plate 2.*

In the series of 146 cases examined by Gallagher (1998) 24.6% (36) showed kidney lesions. However, in Ireland Fagan (1993) found a lower incidence of 11% (3 out of 28 cases). The summary national diagnostic records for Ireland for 453 tuberculous badgers found a similar figure of 15% with kidney disease (Dolan, 1993). Badgers with terminal tuberculosis frequently show kidney disease as an apparent sequel to tuberculous pneumonia which is likely to have exacerbated the condition and accelerated death. Clifton Hadley et al. (1993) found kidney disease in 20 of 47 (42.5%) of such cases.

Table 1 **Frequency and distribution of lymph node lesions according to Fagan 1993, Gallagher 1998, O'Boyle 1997 and 1998.**

Site of lesion *	**Fagan**	**Gallagher**	**O'Boyle '97**	**O'Boyle '98**
Submandibular	2	5	} 75	} 112
Parotid	2	0		
Retropharyngeal	4	21		
Prescapular	3	8	26	59
Axillary	0	5	0	0
Sternal	0	2	0	0
Bronchial	8	23	} 72	} 136
Mediastinal	3	12		
Hepatic	0	2	6	13
Gastric	0	1	0	0
Mesenteric	1	2	3	6
Renal	0	1	0	0
Rectal	0	0	0	0
Prefemoral	0	2	0	0
Illiac	0	6	0	0
Popliteal	8	0	26	60
Total carcases with lesions of TB examined	28	101	194	341

** Lesions were frequently found at more than one site*

Fagan (1993) found a high prevalence of lymph node lesions in Irish badgers; 24 of 28 cases (80%). Of this number, lesions in the nodes were the sole abnormalities found in 17 (61%). These figures contrast markedly with those found in badgers from south west England by Gallagher (1998) in which the total with lesions in lymph nodes was 58 of 146 cases (39.7%). Fagan's findings have been corroborated by the national diagnostic findings for Ireland reported by O'Boyle (1997, 1998). However, the latter are not fully comparable, as some lymph nodes were grouped thus individual lymph node results are lacking. The frequency of involvement of different nodes is shown in *table1*. From the composite results of Fagan (1993) and Gallagher (1998), which are comparable, the nodes with the highest percentage lesion frequencies were bronchial(24), retropharyngeal(19.4), mediastinal(11.6), prescapular(8.5) and popliteal(6.2).

The pattern of lesion involvement mainly differed in the far higher proportions of superficial node involvement seen in the Irish badgers. But the high frequency of popliteal node involvement seen in Irish badgers of 13.4% and 17.6% reported by O'Boyle (1997, 1998) was not found by Monies (personal communication) when examining 385 tuberculous badgers from Cornwall and Devon in which lesions in this node were found in 14 (3.5%). Whether these differences are observational or due to differing disease virulence is uncertain.

The finding of significant numbers of badgers with tuberculous bite wounds was originally reported by Gallagher et al. (1976). Such lesions were typically superficial abscesses associated with two puncture wounds c.3cms apart. Badgers with severe pulmonary disease may have highly contaminated mouths and subcutaneous or intramuscular inoculation of infection arising from bites by such animals is likely to result in more rapid generalisation and extensive disease. Of 16 cases showing bite abscesses, miliary or acute tuberculous pneumonias were seen in 12 (65%) but in only 12 (18%) of 57 cases of respiratory origin infection (Gallagher, 1998). Also acute tuberculous pneumonias were only observed in animals with tuberculous bite wounds.

In his series of necropsies Fagan (1993) found only a single case with a tuberculous bite wound abscess. But he considered that the finding of other animals with sole lesions of the prescapular or popliteal lymph nodes, implied bite wounding to be the likely origin of these, indicating bites as the probable route of infection for 9 of 28 cases (32%). O'Boyle (1997, 1998) subsequently reported incidences of tuberculous bite wounds of 5.7% and 13% in 194 and 341 infected badgers in 1997 and 1998 respectively. But when solitary carcase lymph node lesions consistent with bite wounding, (mostly prescapular and popliteal nodes) were also considered, the likely total of bite wounding cases rose to 13.4% and 24.3% respectively. In Gloucestershire, bite wounding was identified as the origin of infection in 26 (17.8%) of 146 cases, 20 of which showed bite abscesses (Gallagher, 1998).

The pattern of disease in cubs and adults appears to be different. Nolan (1991) examined a series of 72 infected badgers and noted gross lesions in only 18 of 52 adults (35%) whilst 13 of the 20 cubs showed lesions (65%). Only 11 infected cubs were examined by Gallagher (1998) but 9 (82%) showed gross lesions whilst lesions were seen in 92 of 135 adults (68%) thus giving further support to the contention that infection of cubs is more often associated with progressive disease

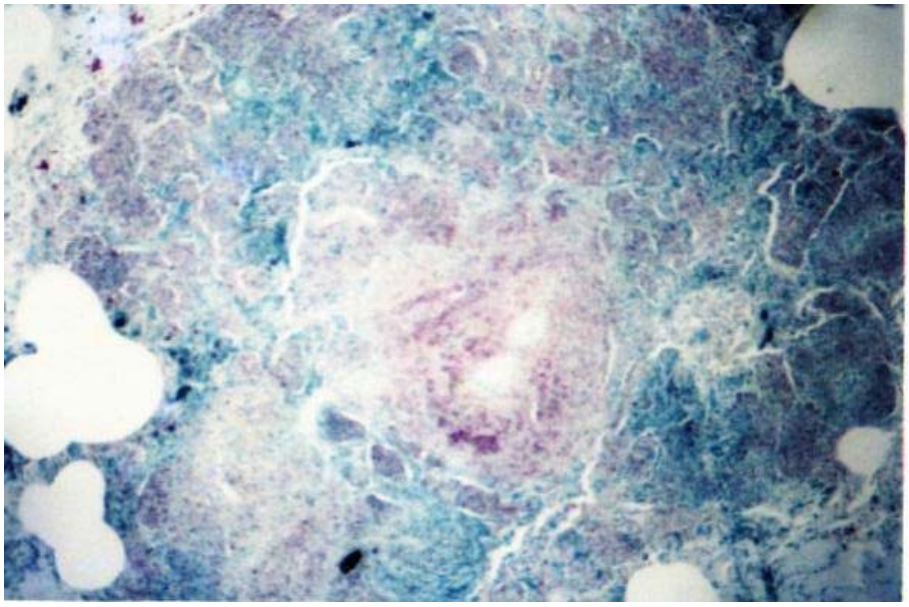
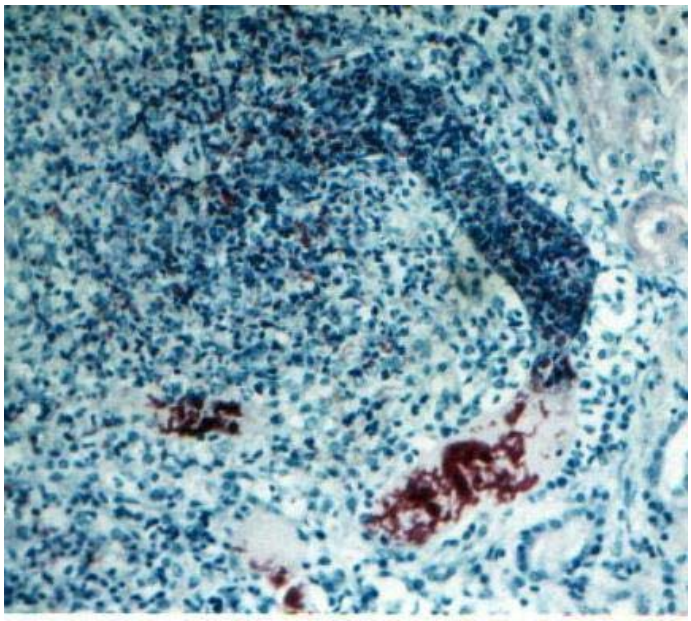


Plate 3 : Lung tubercle from a badger showing advanced miliary pneumonia. Large aggregations of acid fast bacilli are present in the central lesion as well as in the satellite lesions surrounding it.
(stain ZN x40)

Plate 4 : Kidney cortex of badger with advanced generalised tuberculosis showing a proximal tubule, associated with an advanced renal cortical tubercle, containing a cast of cellular debris and numerous acid fast bacilli. Distal to the cast is a tangled mass of acid fast bacilli apparently multiplying in cord like manner typical of broth cultural growths.
(stain ZN x100)



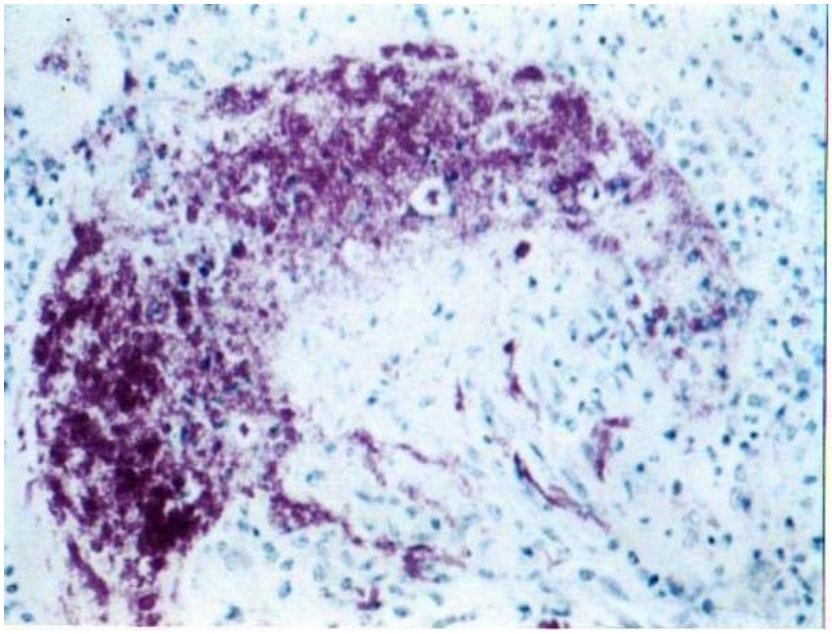


Plate 5: Kidney cortex of a badger with advanced generalised tuberculosis showing a glomerulus, associated with an advanced tubercle, containing vast numbers of acid fasts in large aggregations and outlining the entire glomerular tuft. Tangled masses of acid fasts were apparently multiplying in the glomerular filtrate of the damaged nephron.
(stain ZN x 400)

In man infantile tuberculosis similarly results in a more progressive syndrome, the child appearing to have a lower resistance to disease (Rich, 1951), which may well be the case with cubs.

In all reports on necropsy examination of badgers a significant proportion of cases, whilst yielding cultures of *M.bovis* from lymph node pools, fail to show obvious gross lesions of disease. Fagan (1993) reported 33% of such cases with no visible lesions at necropsy (NVL) and Gallagher (1998) found a similar figure of 35% in Gloucestershire during 1973 to 1976. However, during the ten-year period from 1984 to 1995 the NVL rate in Gloucestershire and Cornwall was approximately 80% (E.Krolik and S.Cushing cit Gallagher et al., 1998). This aspect of the disease is discussed further in the following section

2. Pathogenesis of disease

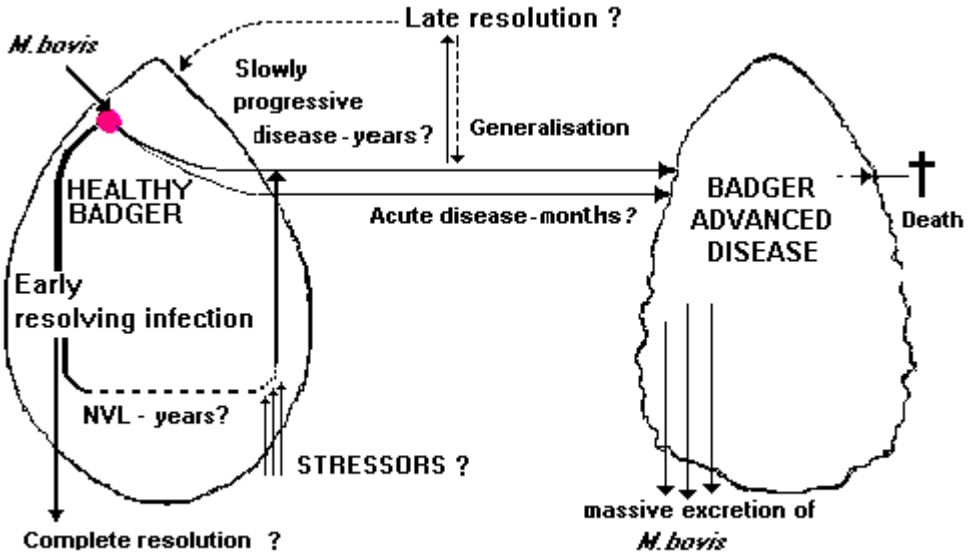
By examining serial sections of lung of 1-2 mm thickness under magnification, a single small lesion of tuberculosis, or occasionally several, was visualised in 7 of 15 badgers found NVL at necropsy but positive for *M.bovis* on culture (Gallagher et al., 1998). These minute lesions usually measuring 0.5 mm or less, were considered to be the primary foci of infection and comprised a fibrous capsule surrounding a core of coagulative necrosis with variable mineralisation. In 2/7 foci no acid fast bacilli(AFBs) were seen and in a further 1/7 only scant numbers of pale Ziehl Neelsen staining AFBs were apparent which were presumably dead or dying. Some pale staining AFBs mainly in the calcified areas, as well as vivid AFBs were seen in the remaining foci. Viable infection with *M.bovis* had been demonstrated by culture of a pool of retropharyngeal and broncho-mediastinal lymph nodes from all these animals and thus the AFB negative foci may not have been sterilised but merely contained very few AFBs.

From these findings it is apparent that the badger shows a containment phase early in the pathogenesis of this disease. The often very high proportions of NVLs found in infected populations of up to 80% imply this phase to occur in a high proportion of infections and probably results in a lengthy period of latency. The duration of this is speculative but is likely to be measured in years. With such a high proportion of latent cases many may never progress further but in some this state may be triggered by stressors to florid disease. Nutritional stress, territorial pressures, assertion of sexual dominance, lactation, intercurrent disease and senile deterioration may all act as stressors. In some cases early containment may result in partial or complete resolution. This situation has similarities to that in man where the majority of primary infections resolve completely whilst a small number may undergo recrudescence occasionally up to 60 years later (Sjogren and Sutherland 1975). Resolution late in disease is also well recorded in man (Cotran et al, 1989) and Clifton Hadley (cit Gallagher et al., 1998) found evidence that this may also occur in the badger. Four badgers which had yielded positive faecal samples, implying established lung disease, were necropsied 14, 22, 38 and 42 months later. None showed any gross lesions of tuberculosis and cultures from lymph node pools were negative. A further case was observed, which 7 months previously had yielded both positive faeces and urine samples implying generalised disease but when seen at necropsy the lesions appeared to have resolved completely and no *M.bovis* was cultured from its tissues. However, resolution in late disease probably occurs infrequently. A summary of the likely

pathogenesis and progression of disease which has been proposed is shown in *figure 2* (Gallagher et al., 1998).

The early tubercle is a round cellular nodule comprised mainly of epithelioid cells with a small central necrotic focus of cellular debris and pyknotic nuclei. Occasional polymorphonuclear leukocytes may be present in a central necrotic focus but only scant numbers of AFBs are usually seen. Peripherally lymphocytes and macrophages are present and the whole lesion is loosely encased by only a mild fibroblastic reaction. Giant cells of Langhan's type are not seen. A proliferative response to the presence of tubercle bacilli is apparent with no indication of the highly destructive reaction seen in man or cattle associated with the development of hypersensitivity (Francis, 1958; Gallagher et al., 1976).

Figure 2 . **Proposed pathogenesis and progression of tuberculosis in the badger.**



Pulmonary disease appears to be slowly progressive. From an initial infection in the lungs, endobronchial spread and centrifugal expansion or satellite development from infected migrant macrophages, seems likely to progress the disease gradually, metastasising and eventually spreading haematogenously to produce a variety of gross lesions. Infection of the pulmonary arterial system is likely to result in disseminated lesions confined to the lungs. But infection of the pulmonary venous system will result in infected blood being pumped via the left ventricle throughout the body. If the seeding of infection is only light, isolated organ tuberculosis is the likely sequel where bacilli may be killed or not established in some sites but colonise others (Cotran et al., 1989). In acute tuberculous pneumonias, which are usually asociated with tuberculous bite wound infection, early generalisation appears to result in accelerated pathological changes of a more acute nature (Gallagher et al., 1976)

As tubercles develop further, central necrosis becomes more pronounced and slightly greater peripheral fibroblastic activity may be apparent. Coagulative necrosis develops, bordered by a wide zone of pyknotic and karyorrhectic nuclei and epithelioid cells. Calcification of necrotic debris is not a feature but in some cases a number of minute granules of mineralisation may be found in such debris. Advanced

disease is associated with the presence of numerous AFBs. Sometimes clumps and tangled masses of bacilli may be seen in such lesions in lungs, kidneys and elsewhere (*plates 3 to 5*) giving a picture similar to that described for the anergic state of leprosy in man known as lepromatous leprosy (Jopling, 1982).

The likely time-span for the development of chronic progressive tuberculous pneumonia has been broadly elaborated by serial sampling of the naturally infected badger population at the badger ecology study site, Woodchester Park, Gloucestershire. The time from the first positive faeces sampling, indicating established lung disease, to death was calculated for those with obvious bite origin infection and those with infection of apparent respiratory origin. The mean survival of those with bite infection was 117 days (95%CI –180 to +341 days) compared with 491 days (95%CI +253 to +729 days) for those with apparent respiratory origin infection (Clifton Hadley et al., 1993), further supporting the contention that bite wound infection results in a far more rapid demise. Thus disease in such cases may have a course lasting months whilst respiratory origin disease generally persists for a year with some cases lasting two years (Cheeseman et al., 1989).

3. Immunity

Ranke (1916) first described the three phases of tuberculosis in man, of infection, containment and disease, to which the badger also appears to broadly conform from the above histopathological studies. Immunological responses were examined by Mahmood et al., (1987) in four badgers experimentally infected intradermally with *M.bovis*. They identified three phases which appear to conform to the histopathological stages of initial establishment of infection, containment and latency and finally florid disease. An early phase was found where the lymphocyte transformation assay (LTA) to intradermally injected BCG steadily and significantly increased and limited although not consistent tuberculin sensitivity could be demonstrated from 3 months post inoculation. Little antibody was detected during this stage. After 6 months post inoculation an intermediate stage followed where these reactions were highly variable. The final stage, seen in 2/4 cases was characterised by extensive disease, associated with changing, usually negligible skin sensitivity, a falling LTA response and a sudden increase in antibody levels.

Attempts to enhance the natural immune reaction in the badger by vaccination have been carried out in a pilot trial using BCG (Stuart et al., 1988). Intradermal inoculation of BCG stimulated an LTA response but no skin test reactivity or antibody responses in the 12 vaccinates. When 7 of these were challenged intradermally with a pathogenic *M.bovis* strain, between 5 and 25 months post vaccination, the LTA response fell, the antibody response rose and a positive skin test response developed. The vaccinates were shown to live longer, shed fewer bacilli and their inoculation sites healed more rapidly, than the group of 3 unvaccinated controls. Also the vaccine was found not to be excreted by the badgers.

Although only small numbers of animals were used in this trial, vaccination with BCG appeared to enhance cell mediated immunity and possibly ameliorate the effects of disease resulting in reduced excretion of bacilli. Southay and Gormley (1998) further examined the LTA response using 18 wild badgers, 13 of which were vaccinated with a low dose of BCG Pasteur (10^5 cfu/ml) whilst the remainder were injected only with saline. Some evidence of possible seasonal increase in LTA

activity was found from January to March but booster vaccination in March resulted in a significant increase in LTA to bovine PPD compared with controls, implying the development of a cell mediated immune response to *M.bovis* antigen.

In man silicosis is associated with a greatly increased susceptibility to tuberculosis (Snider, 1978). Higgins et al. (1985) demonstrated the presence of silicates and silica in macrophages in badger lungs. They postulated that a similar susceptibility was thus likely in the badger. However, the cellular reaction identified in the 3 badgers examined was relatively mild and they did not find the destructive fibrotic silicosis nodules associated with macrophage leaching and consequent immunosuppression as seen in man (Snider, 1978). Unlike man the badger, well adapted to an underground existence, appears to be considerably more resilient to the excesses of silica and it may have little or no role in its susceptibility to tuberculosis.

4. Diagnosis of infection

Pritchard et al. (1986) examined the efficacy of clinical sampling of 47 badgers of which 10 were subsequently found to be infected following necropsy. The samples taken were faeces, urine, tracheal aspirates and bite wound swabs. Only 2 of the 10 were detected, one showing a positive tracheal aspirate and faeces and the other a positive bite wound swab. Using the same techniques, similar results were obtained by Cheeseman et al. (1985) in which only 2 of 8 infected individuals were detected. However, at Woodchester Park, repeated clinical sampling identified 11 of 23 infected individuals (Clifton Hadley et al., 1993). The same sampling approach was used but additionally laryngeal swabs were taken. Bite wound samples initially identified 8 of the positive cases, the remaining individuals being identified by one or more of the other samples. An intermittent pattern of excretion was apparent from these findings with no isolations being made at some of the subsequent samplings.

These techniques of clinical sampling are insensitive and suffer the disadvantages of only detecting animals which are excreting and probably with well established disease. Also there is a time lag of up to 6 weeks for culture. Faeces sampling of badger latrines was found to be a useful tool for identification of infected social groups detecting 4 out of 5 such groups in a study by Cheeseman et al. (1985). However, in another study repeat sampling was carried out at monthly intervals over a period of 6 months collecting a total of 263 samples which yielded 13 positives (5%). Collection of latrine samples at weeky intervals during springtime, over a period of from 5 to 8 weeks, identified infection respectively in 6, 8, 4 and 2 of 8 social groups during the 4 years of a field study by Wilesmith et al.(1986a). The maximum isolation rate was 18.5% and the minimum 1%. They collected a total of 727 samples yielding 48 isolates (6.6%). Only fresh faeces were selected in this exercise but collection of a further 1,064 latrine samples irrespective of freshness gave a much lower isolation rate of 27 positives (2.5%).

Intestinal lesions of *M.bovis* infection in badgers are extremely rare. Faeces sampling thus seeks to detect cases of lung disease in which *M.bovis* will be excreted in faeces via swallowed lung discharges.

The expected detection rate of infection by faeces sampling was modelled by Gallagher (1998) using culture of rectal faeces taken at necropsy. A series of 30 badgers with lung lesions categorised according to severity were sampled and 19 were

positive for *M.bovis*. Of these only 6 isolates were from those 16 with few to fairly numerous lesions whilst almost consistent excretion was found with 13 isolates being recovered from the remaining 14 cases of miliary or advanced acute tuberculous pneumonia. Adjustment for the frequency of cases according to these categories in the larger series of 146 infected badgers examined gave an expected overall isolation rate from tuberculous pneumonia cases of 54.6%. The prevalence of infection in the population from which these badgers were taken was approximately 20% and 50% of the infected animals showed lesions of tuberculous pneumonia. The likely isolation rate of faeces sampling of this severely diseased population would thus have been 5.5%. This figure approximates to those reported but when taking serial latrine samples, an animal with advanced disease may be re-sampled, thus giving an apparently higher detection rate.

A technique of needle biopsy of the retropharyngeal lymph nodes has been tested on 153 carcasses of which 12 were later found infected at necropsy (Gallagher, unpublished findings). The results of culture of the needle aspirates onto modified Middlebrook 7H11 medium (Gallagher and Horwill, 1977) are shown in *table 2.* Six of the 12 were identified by this technique including 2 cases NVL at necropsy. But unless great care is taken the technique has the potential to result in haemorrhage complications. Haemorrhages of 3 to 6 cms along the fascial planes of the neck muscles were found in 3 of 25 anaesthetised animals sampled prior to euthanasia. However, this approach could have limited value.

Table 2: **Results of culture for *M.bovis* from needle biopsy aspirates of retropharyngeal lymph nodes and necropsy of 12 infected badgers**.

SAMPLE REF	BIOPSY CULTURE	NECROPSY FINDINGS
199	-	Mediastinal LN calcified lesion
209	+	Miliary pneumonia
226	+	Retropharyngeal LN caseous lesion
235	+	NVL
255	-	NVL
256	-	NVL
258	+	NVL
262	+	Miliary pneumonia
298	+	Miliary pneumonia
339	-	NVL
369	-	Broncho-mediastinal LN calcified lesion
387	-	Broncho-mediastinal LN calcified lesion

As discussed earlier the retropharyngeal nodes were the most frequently affected after the broncho-mediastinals. It is noteworthy that Pritchard et al. (1987) demonstrated the presence of infection at necropsy in a variety of lymph nodes, including the head nodes, in badgers experimentally infected from 17 to 22 months previously. However, clinical sampling techniques suffer the disadvantage of an in-built delay of up to 6 weeks for cultural isolation. Development of a specific PCR for rapid antigen detection would be a valuable asset for all such approaches.

Tuberculin testing of badgers as a means of diagnosis was first attempted by Little et al. (1982a). Every 2 months for 2 years 13 badgers, subsequently found to be infected, were tested intradermaly in the thigh with bovine and avian tuberculins. No reactions were elicited. Using whole killed cells of *M.bovis* and bovine and human tuberculins injected intradermally in the flank, Pritchard et al. (1986) managed to correctly identify only 3 of 45 naturally infected animals. Histological examination of the skin inflamatory response to tuberculin showed that changes indicative of a mild hypersensitivity reaction were apparent after 2–3 days post inoculation associated with a small increase in skinfold thickness although no erythema, palpable oedema or induration was apparent (Higgins, 1985).

Serological tests for tuberculosis are renowned for poor sensitivity and specificity due to the presence of common antigens from ubiquitous mycobacteria and the dominance of cell mediated responses in other than the later stages of disease. In the badger this is no different. The complement fixation test (Little et al., 1982a) and the enzyme linked immuno-sorbant assay (ELISA) using mycobacterial extracts or tuberculins as antigen were both found too insensitive to be of practical value (Morris et al., 1979; Mahmood et al., 1987). However, Nolan (1991) identified an immuno-dominant component of *M.bovis* which was present in all isolates examined and absent from other mycobacteria. Using this she developed a blocking ELISA. This was later developed into an indirect ELISA using the highly specific 25 kDa antigen (Goodger et al., 1994). Field trials of this test showed a very high specificity of 94.3% but its sensitivity was low at 40.7%. However, the sensitivity was significantly higher, 62.3%, in animals found to have gross lesions (Clifton Hadley et al., 1995a). Whilst of limited use for diagnosis in individuals, the test could be useful for screening social groups where diseased animals may be present.

Work by Mahmood et al. (1987) and Stuart et al. (1988) determined the conditions for performance of the LTA. However it is only recently that a comparative LTA for badgers has been developed (Dalley et al., 1999). Preliminary results indicate an increased sensitivity (87.5%) but a decreased specificity (84.6%) compared with the indirect ELISA. Further development and validation of this test is indicated, since, used in conjunction, these two tests may provide a high diagnostic performance.

Haematological parameters were examined by Mahmood et al., (1988) who found a picture typical of disseminated tuberculin negative tuberculosis in man of a rise then a fall in red cell and white cell counts, an increase in the proportion of polymorphs and monocytes and then a fall in lymphocytes late in the disease.

Radiography is unlikely to be of any diagnostic value as lesions rarely calcify and are generally small. In a small series of carcases examined only advanced miliary disease could be distinguished as well as a case of calcification of the maxillary nodes (Gallagher – unpublished findings). Radio opaque dirt particles complicated interpretation.

5. Excretion and viability of infected products

Animals with advanced miliary disease can excrete enormous numbers of bacteria. Using direct culture onto selective modified 7H11 medium (Gallagher and Horwill, 1977) with no prior decontamination, Gallagher (1998) counted $200x10^3$ and $75x10^3$ cfu per ml of bronchial pus which was aspirated at necropsy from two animals with

miliary disease. The counts from urine were $250x10^3$ and $217x10^3$ per ml and from faeces $75x10^3$ and 68 per g. A full urination, which may amount to 20–30 ml, could thus contain several million organisms.

Nolan (1991) examined the bacterial load in badger lesions and found a significant direct correlation of markedly increased numbers of bacteria in cases showing more advanced pathological changes. Using animals with advanced as well as minimal lesions Gallagher, Monies and Rule (in preparation) attempted to determine the proportion of cases excreting bacteria. Lung lavage and urine samples were taken at necropsy from 43 badgers subsequently found to be infected with *M.bovis*. The results are shown in *table 3*.

The miliary lung and kidney cases were all positive for *M.bovis*, almost all yielding heavy growths. Where less advanced lung lesions were found only 4 of 9 yielded positive lavage samples but one of these also had kidney lesions not apparent grossly but yielding a positive urine sample. Solitary kidney lesions were found in a further 3 cases and produced positive urine samples. Histopathology of the lungs of one of these cases, as well as the only NVL case yielding a positive lavage sample, revealed early lung lesions which were not apparent grossly. This was also so for the 2 cases showing only lymph node lesions at necropsy. These findings imply that excretion from rupture of lung lesions, is not unexpectedly intermittent but once disease has advanced to produce miliary lesions excretions are likely to be continuous and heavy. Whenever gross lesions were found in the kidneys, the urine was consistently positive and growths of bacilli heavy.

Table 3: **Results of culture of lung lavage and urine samples from badgers found to be infected with *M.bovis***

Necrospy lesions	Totals	Positive lung lavage	Positive urine
Miliary lung & kidney	8	8	8
Lung only	9	4	1*
Kidney only	3	1*	3
Lymph node(s) only	8	2*	nil
NVL	15	1*	nil
Totals	43	16	12

* *Histological examination revealed several small AFB +ve lesions in the tissues yielding these culture positive samples in each case, which were not grossly apparent at necropsy.*

The duration of infection on pasture is fortunately fairly brief. On unshaded pasture during the winter two heavily infected urine samples, taken at necropsy, showed an apparent decay of 99% and 99.92% against pre-exposure counts, of $250x10^3$ and $217x10^3$ cfu.per ml respectively, by the first sampling at one week post-exposure. Scant numbers of *M.bovis*, of uncertain epidemiological significance, were recovered up 5 weeks post- exposure. Using bronchial pus samples the decay rate was 99.4% and 96.6% from counts of $200x10^3$ and $75x10^3$ cfu per ml, at the first sampling after 2 weeks. Again scant numbers could be recovered and in the more protective substrate afforded by mucous these were found up to 10 weeks. Deterioration in faeces was, not surprisingly, less rapid, 88% dying after 2 weeks and 99.7 by 4 weeks from the initial counts of $75x10^3$ and 68 per g. These experiments were repeated in summer and the first samplings for urine were at 3 days, for bronchial pus at one week and faeces 2 weeks. All were negative. (Gallagher, 1998).

Whilst tubercle bacilli are reputed to be resistant organisms surviving for long periods of environmental exposure, much of the work elaborating this reputation was carried out under highly artificial conditions (Wray, 1975). Cultures or tissues were often used rather than excreta from diseased animals and estimations were mostly qualitative, measurements being made using the highly sensitive guinea pig test. In nature it is likely that sunlight, rain dilution, temperature and the effects of other microrganisms, particularly antibiotic producing fungi, greatly modify the normal resilience of tubercle bacilli.

Viability of infection in the dead badger has also been examined using carcases showing miliary pneumonia. In such cases lesions are fairly evenly distributed throughout the lungs. After removal of a standard sized portion of lung, the viscera disturbed during necropsy were replaced and the carcases were then sutured back together again. This was done to facilitate the normal putrefactive process. Cultural counts from the initial sample were compared with those taken serially after burial or exposure on the surface. Three of the 5 carcases used yielded no isolations at 2 weeks post-exposure. By 4 weeks only one was still positive and by 6 weeks all were apparently sterilised by putrefaction (Gallagher, 1998).

The duration of infection within setts is still uncertain. Setts can be very extensive but the bedding would seem the most likely source of persisting infection from saliva drools, urine and faecal contamination as well as discharges from bites. Mindful of this bedding samples have been examined from a sett known to be infected which had been sealed for six months after the remaining residents had been gassed. Samples from an adjacent sett belonging to the same social group were tested for infection a week after gassing and one of four samples of bedding contained an estimated 3,000 to 6,000 cfu. per 100g. But the remainder were negative as were all 9 samples from the sett sealed for 6 months (Gallagher, 1998). These results imply that infection is possibly not as widespread or persistent within the sett as might be thought. Badgers regularly replace bedding and probably keep levels of infection relatively low.

6. Epidemiology in badgers

Wilesmith (1983) demonstrated a direct association between increased sett density and the occurrence of tuberculosis in cattle in the south west of England as well as in other affected areas in the rest of England and Wales. Using data from the national survey of road traffic casualties Cheeseman et al. (1989) confirmed a higher prevalence in the counties of the south and west in areas designated as having a very high or high sett density. The highest prevalences were seen respectively in Gloucestershire, Avon, Cornwall and Wiltshire of 11.5, 8.2, 6.9 and 3.8% carcases infected with *M.bovis* . These prevalences were much the same up to 1993 (Report, 1994). However, these authors (Cheeseman et al.,1989) observed that at social group level there was not a simple relationship between prevalence and social group numbers. Adjacent social groups in a relatively small area can show marked variations in prevalence with infection being undemonstrable in some (Cheeseman et al., 1981). Both temporal and spatial variations have been observed in the Woodchester Park study area with spread of infection being slow and restricted (Cheeseman et al., 1988).

Social groups are likely to be mainly genetically related family groups and varying susceptibility at this level is not unexpected. In man and in cattle familial genetic susceptibility is well recognised, although it is often difficult to separate the real effect of close familial contact in some cases (Hutt, 1958).

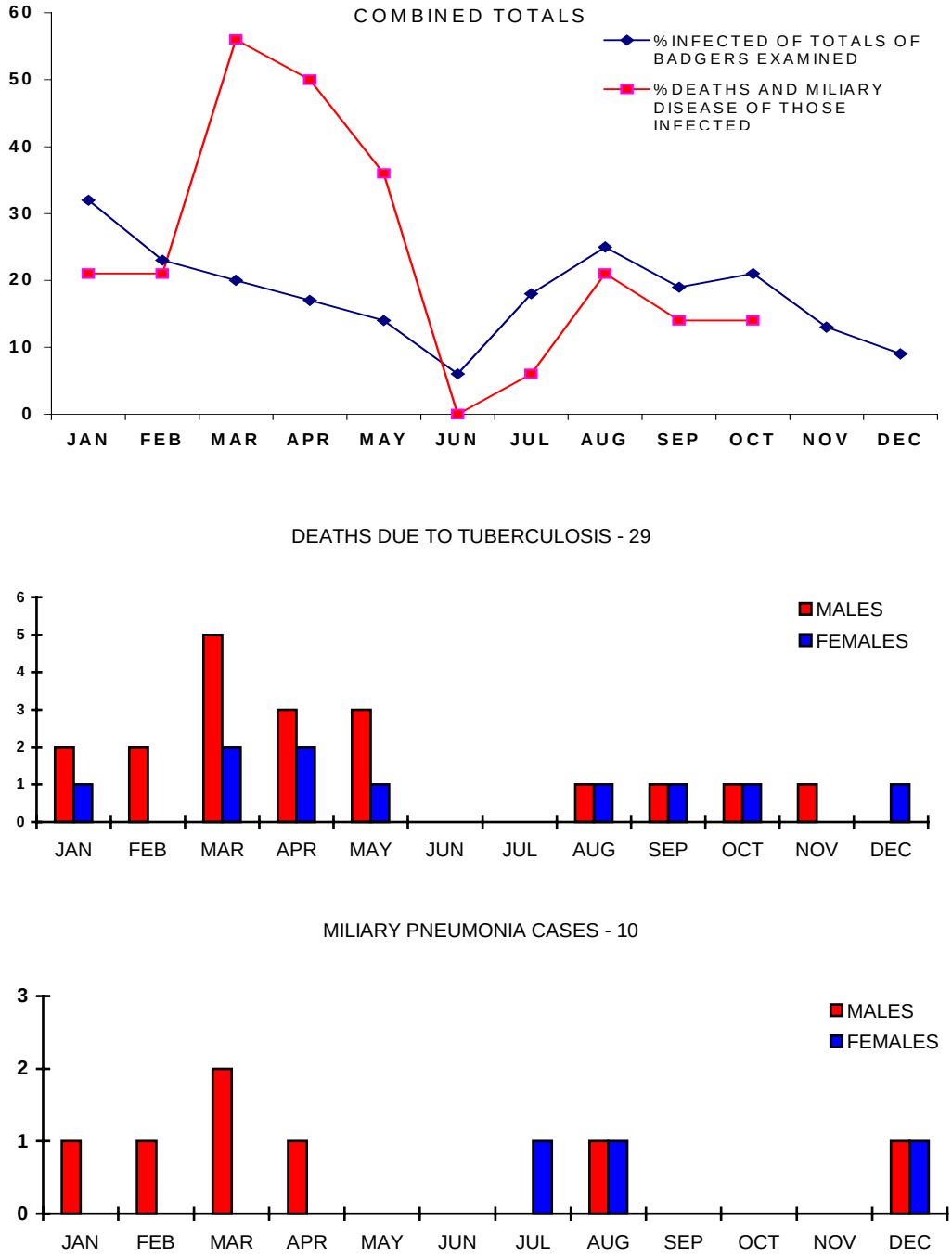
The sex associated prevalence of infection has been examined in several surveys in which males have shown a greater prevalence than females (Cheeseman et al., 1988; Gallagher and Nelson, 1979; Nolan, 1991). There are behavioural reasons for this difference. Males are far more concerned with territorial defence and ensuring territorial integrity by faecal or urine marking, which causes them to range more widely and thus have more contact with others. In Woodchester Park, Cheeseman (1979) using radio telemetry, found two males in spring ranged across a mean area of 49.3 hectares (range 41-57.3), whilst five females covered only 14.7 hectares (range 7.5 – 19).

Territorial aggression is essentially the province of males, although it is not exclusive to this sex. A hierarchical social system is thought to occur in badgers (Neal, 1977) and there is some evidence that large powerful boars assert sexual dominance over lesser males. Kruuk (1978b) and Cheeseman (1979) both record invasion of large boars into neighbouring social groups during the main mating season in early spring. Skirmishes and more vicious fights generally result from territorial defence and mostly occur in springtime, (Kruuk, 1978a; Gallagher and Nelson, 1979). The effects of these activities may result in physiological stresses on males which together with the likely increased contacts, may make them more likely to acquire infection and develop disease.

Bite wounding frequency seems likely be a direct reflection of population density. However, Rogers et al. (in press) observed a significant reduction in bite wounding frequency in the Woodchester Park study area where territories had long been stabilised whilst the social group sizes had increased significantly. Thus whilst density would logically appear to be important, disruption of stability seems also likely to be an important factor in territorial aggression.

There is a seasonality of severe disease and deaths due to tuberculosis with the peak occurring in March/April in the south west of England. A lesser peak in August/September is apparent but this seems likely to be an artefact (*see figure 3*). During March/April these cases represented over half the infected badgers examined in this series. A total of 26 of 39 such cases involved males and 19/39 cases occurred in the spring during March to May. These findings were based on the identification of 146 infected badgers out of 757 examined over a four year period from 1973 to 1976 at the Veterinary Investigation Centre, Gloucester (Gallagher, 1998). Of this sample 56% were killed for diagnosis, 43% were killed in road traffic accidents and 6% found dead. Whilst the prevalence of infection was higher in the killed badgers (killed 20%, RTA 14%) there was no difference in the pattern of infection and disease in these groups. In the natural death sample the prevalence was 43%. At Woodchester Park, the finding that 5 of 12 badgers positive on clinical sampling were found from February to March and a further 5 were disclosed up to July is consistent with this seasonality (Cheeseman et al., 1988).

Figure 3 **Seasonal occurrence of infected badgers and miliary pneumonia cases and deaths* due to tuberculosis in a total of 146 infected badgers out of 757 examined from 1973 to 1976.**

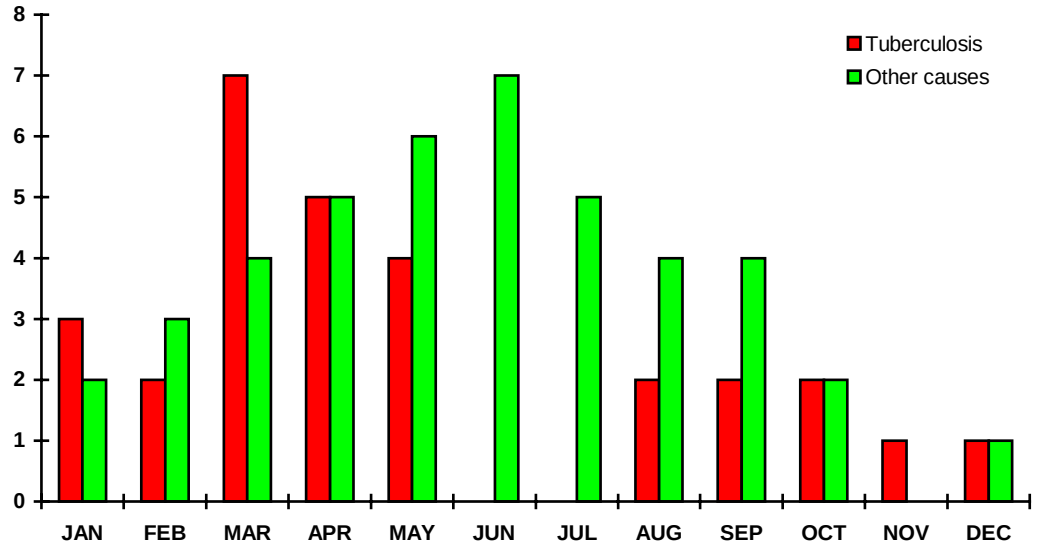


** Two animals with advanced tuberculosis killed in extremis are also included in 'deaths'. Miliary includes acute tuberculous pneumonias. In Nov 4 of 31 badgers were infected and 1 died of TB; in Dec 3 of 32 were infected all with miliary disease. These data were considered too small to % for the combined totals graph.*

Spring is the zenith of the badger's year with births in south west England occurring mainly in late February but extending from mid January through to mid March, followed by the start of the main mating season. Territorial demarcation activity is maximal at this time peaking in February/March with a lesser secondary peak in late

summer (Neal and Cheeseman, 1996). Cases of tuberculous bite wounding were found to mirror this pattern of territorial activity (Gallagher and Nelson, 1979). It is thus not unexpected that a chronic disease such as tuberculosis may fulminate in springtime. This seems apparent from a study of causes of natural deaths (*see figure 4*). Deaths due to tuberculosis peaked in spring and late summer showing a different pattern to those of other causes which, in ranked order of frequency were, non specific bite wound infections(11), starvation(10), pneumonia(3), enteritis(2), arteriosclerosis(2), metritis(1), nephritis(1), lymphosarcoma(1), polyarthritis(1) and undiagnosed causes (Gallagher, 1998). Whilst advanced arteriosclerosis lesions appeared the cause of death in 2 cases this condition was observed as a possible exacerbating cause in a number of other conditions (Gallagher and Nelson, 1979). Tuberculous bite wound cases contribute a large component of the diseased males seen in the spring but when these were disregarded there was still a greater tendency for disease in males although it was then not statistically significant probably due to reduced sample size (Gallagher, 1998).

Figure 4 **Seasonal occurrence of deaths due to tuberculosis and other causes of 72 badgers found dead from 1973 - 1976**



** Two animals with advanced tuberculosis killed in extremis are also included*

Bite wounding appears a common means of transmission of infection between social groups. Emigration of infected individuals is also likely to spread infection. Within social groups, because of the gregarious nature of badgers, aerosol transmission from close contact during communal sleeping and grooming, would be expected to result in a high level of intra-group infection. Pseudo-vertical transmission is also considered to be an important mode of transmission from sow to cubs (Cheeseman et al., 1981; Nolan and Wilesmith, 1994). But this may be variable.

Nolan (1991) found a higher prevalence of infection in cubs than in adults in a series of 177 badgers in which 52 (39.1%) of the adults were infected and 20 (45.5%) of the cubs were so. Whereas Gallagher (1998), examined 757 badgers and found infection in only 11 (13.4%) of the cubs but 135 (20%) of the adults. Considerable variation is probably to be expected as was found in an investigation by Cheeseman et al. (1981) of 4 sites, comprising 24 social groups, 11 of which were infected. From these

infected groups, cubs were found in 7 and in 3 of these the cubs were infected. But when a sow with cubs is diseased it is likely that all the offspring become infected and this did appear to be so. In the 3 groups 3 out of 3, 6 out of 7 and 4 out of 6 cubs were infected.

7. Overspill of infection to other wildlife

Several surveys have been carried out at sites where tuberculosis had been confirmed in cattle but the origin of infection was obscure, in order to determine the tuberculosis status of the resident wildlife species and what role they may have in the spread of infection to cattle.

Little et al. (1982b) carried out a comprehensive survey of wildlife species in an area of South Dorset where there was a severe outbreak of tuberculosis in cattle. Representative samples were taken from total of 19 wild mammal species which were using the farms involved covering 12 km^2. Infection with *M.bovis* was found in badgers, rats (*Rattus norvegicus*) and a red fox (*Vulpes vulpes*). Of 25 badgers trapped 14 were positive, 12 with lesions of tuberculosis. Ninety rats were caught and 2 were infected but showed no gross lesions. Some of the badgers had advanced disease and it was surmised that the rats may have acquired their infections from eating undigested whole grains of maize which were noted in a number of the badger faeces samples. They may also have become infected from eating cadavers of diseased badgers. The non-progressive nature of the infection in the rats was considered by the authors to make them unlikely to be other than a dead end host. The fox, one of seven examined, showed no lesions and was similarly considered a dead end host having probably acquired its infection from scavenging badger carcases.

Barrow and Gallagher (1981) carried out a similar intensive survey at 2 locations in the Cotswold Hills, Gloucestershire. Wilesmith et al. (1986b) intensively investigated an area of East Sussex used by 3 cattle herds over a 3 year period. In both investigations the only species found infected was the badger

More extensive surveying was carried out in Thornbury, Avon and again only badgers were found to be infected. Extensive surveying over the Cotswold Hills disclosed widespread infection in badgers but also 4 of 80 foxes were found infected although showing no lesions. These were found on 2 farms where diseased badgers were present and scavenging of badger carcases was thought the origin of their infections (Gallagher, 1980).

Few ferrets were found in the areas surveyed and no evidence of infection was found in them (Report 1981). However ferrets belong to the family mustelidae as do badgers. Free living ferrets (*Mustela putoris*) have been found to be infected in several locations in New Zealand where their infections were considered to have arisen from cattle or deer (de Lisle et al., 1993). Subsequent studies have implicated ferrets as the possible source of infection for some outbreaks of tuberculosis in cattle in New Zealand (Ragg et al., 1995)

M.bovis was cultured from 2 moles (*Talpa europaeus*) from a farm in Cornwall (Report, 1976). No lesions were found and because of the solitary nature of moles infection was considered to have come from diseased badgers which had been found

in the area. Neal (1977) notes that badgers have been known to excavate mole runs and devour nests of young.

Wild deer are highly mobile and it can be difficult to ascertain what areas they have been using and whether they are infected. The first indication of *M.bovis* infection of free living deer in Britain was the finding in North Wiltshire of a roe deer (*Capreolus capreolus*) showing lung abscesses (Gunning, 1985). Badgers with discharging tuberculosis lesions had been found in the locality and these were thought the likely origin. Subsequently further sporadic cases have been found in roe and fallow deer (*Dama dama*) in Wiltshire and Dorset (Report, 1985 and 1986). During 1998 a further 6 cases were found. Nearly all were in areas where tuberculosis had been found in cattle and badgers in the counties of Hereford, Gloucester, Somerset and Devon (Report 1998). Prior to these findings large numbers of free living and park land deer have been examined for mycobacteria. The only organisms isolated were either *M.avium* or *M.avium paratuberculosis* (Matthews et al., 1981).

Tuberculosis has also been found in deer in Ireland. Generalised tuberculosis due to *M.bovis* was identified as the cause of death in an emaciated fallow deer in the Wicklow mountains (Wilson and Harrington, 1976). Dodd (1984) attempted to determine the prevalence of tuberculosis amongst the deer in these mountains and found 5 (3.8%) with lesions of 130 deer which were shot. The dominant species were sika (*Cervus nippon*) and all the infected animals were sika or sika red deer (*Cervus elaphus*) hybrids. Dodd was uncertain as to whether the deer contracted infection from infected cattle in that area or whether they might have acquired infection from badgers.

8. Overspill of infection to domestic animals

The badger's habitat is shared with a variety of wild and domestic animals. It might seem ironic but grazing stock, especially cattle, improves that habitat for badgers. One of the main determinants of badger numbers is the abundance of earthworms, the biomass of which is considerably higher on pastureland than on arable. Also a shorter sward resulting from grazing is preferred by badgers for foraging (Kruuk, 1978b). The other major determinant is a suitable terrain for excavation of the sett (Roper, 1993). Cattle and sheep are the most abundant domestic animals using pastureland in the south west of England and cattle are considerably more susceptible to tuberculosis than sheep (Francis, 1958). Pastures may be contaminated by diseased badgers with sputum, faeces, urine and discharges from bite wound fistulas.

Benham and Broom (1991) observed that cattle generally avoided areas of grass soiled with badger faeces and urine with 99.3% of cows taking no bites of faeces contaminated pasture for up to 28 days, whilst 88.7% avoided urine contaminated pasture for up to 14 days. However, 2/240 cows used in the trial were willing to graze close to the faeces and 7/240 grazed close to the urine contaminated areas. White et al. (1993) studied the urinatory patterns of badgers, noting that urinations were frequently near territorial margins along linear habitat features or boundaries. Brown et al. (1993) fitted a spool releasing a light line to badgers which were injected with fluorescein and then tracked their movements using an ultra violet lamp to detect urine and faeces depositions. They noted fairly large areas of pasture soiling, particularly as a result of the badger's habit of dribbling urine whist still walking. Pasture contaminated with urine in this manner appears to be avoided less and has

been observed to be grazed within 24 hours of deposition by cattle (White et al., 1993) Benham and Broom (1991) found faeces or urine contaminated herbage was eaten most when the pasture became bare and grazing became relatively unselective. Thus although most cows normally avoid badger products a very small number do not and risk ingestion of infected herbage, particularly when overgrazing occurs. Hutchings and Harris (1997) corroborated these findings and observed that lower ranked members of the herd were more likely to graze the contaminated herbage.

Direct contagion between badgers and cattle was considered by Benham and Broom (1989) to be extremely unlikely, as both usually avoid each other keeping 10-15 metres apart. Even when approached by inquisitive cattle badgers would always move away maintaining a distance of at least 2-3 metres from the approaching animal. However, close contacts have occasionally been observed at pasture (Kruuk, 1989; Brown et al.; Sleeman and Mulchay, 1993) as well as entry into cattle buildings (Muirhead et al., 1974; Sleeman and Mulchay,1993).
Cattle may become infected from badger products by inhaling during investigative behaviour. Alternatively they may ingest contaminated grasses. When in the rumen, fermentation activity is likely to generate aerosols of a variety of bacteria in rumen gasses. A large proportion, of eructated gas from the rumen, passes through the lungs prior to being expelled (Waldo and Hoernicke, 1966). Thus it seems likely that aerosols of tubercle bacilli may be transported to the lungs in this manner. Using non-pathogenic marker organisms placed in the rumen of a fistulated heifer, Mullenax et al. (1964) showed that in fact appreciable numbers of bacteria were conveyed to the lungs during eructation. This finding is pertinent to the possible establishment of a tuberculous infection, since the number of tubercle bacilli required to establish infection via the pulmonary route is considerably less than is required to establish infection via the alimentary route (Sigurdsson, 1945). In an area where diseased badgers were confirmed as the primary origin of infection for the cattle, Wilesmith et al. (1982) found a pattern of lesions consistent with mainly respiratory origin infection. The frequency of lesions in the tissues of 126 visible lesion cattle reactors slaughtered was lungs 5, retropharyngeal/submaxillary lymph nodes 50, bronchial/mediastinal lymph nodes 87, mesenteric lymph nodes 9, and precrural/prescapular lymph nodes 4.

Sheep are fairly abundant in many areas where infection of badgers has been disclosed but no cases of tuberculosis have been found in slaughterhouses in this country. Cases have been recorded in New Zealand where on one farm a prevalence of infection of 18% was found (Davidson et al., 1981). Goats also appear to be fairly resistant (Francis, 1958) and there have been no reports of infection in this species in Britain since the 1930s (O'Reilly and Daborn, 1995). However, there have been reports of serious disease in milking goats in north east Spain in which unfettered progress of infection has resulted in severe disease and deaths (J.F.Garcia Marin cit O'Reilly and Daborn, 1995). In areas of endemic tuberculosis in New Zealand infection of feral goats has been found (Sanson, 1988).

Horses are reputed to be very resilient to infection with bovine tubercle bacilli (Luke, 1958) although a sole case of generalised disease due to *M.avium* infection was encountered in south Gloucestershire. The animal had been grazing lands adjacent to a wildfowl park in which there was a considerable problem of avian tuberculosis in wild ducks (Gallagher, 1980).

Pigs by contrast are susceptible to tuberculosis of both the bovine, human and avian strains (Francis, 1958; O'Reilly and Daborn, 1995). However, despite the increase in outdoor pig rearing in Britain, to date only cases of *M.avium - intracellulare* infection have been found at slaughter.

Bovine tuberculosis in cats used to be fairly common in Britain and Jennings (1949) reported infection in 13% of cats in a study at Liverpool. Raw milk was the usual source of their infections. Where severe outbreaks of tuberculosis were seen in herds of cattle in Pennsylvania in the United States during 1966-1968, four of nine dogs and 24 of 52 cats living on the farms were found to have tuberculosis (Snider et al., 1971). These authors considered that after slaughter of the cattle on these farms had been completed, these animals were maintaining infection and posing a risk of re-establishing it in the herds.

Cases of tuberculosis in cats in Britain have recently been reviewed by Gunn-Moore et al. (1996). Of 19 cases they investigated all were infected with an organism showing features intermediate between *M.tuberculosis* and *M.bovis* which is possibly *M.microti*. Orr et al. (1980) encountered 2 cases of cats infected with " a low pathogenicity" strain of *M.bovis* with rather speculative contact with badgers. But more recently an outbreak in cattle in north Cornwall has been associated with infected cats (Durr et al., in preparation). Four of 6 semi wild farm cats examined post mortem showed lesions of tuberculous pneumonia from which *M.bovis* was recovered. Shortly after the index case of this extensive outbreak, one of the reactor cows was found to have udder tuberculosis. Raw milk had been liberally fed to the cats and appeared to have established infection amongst them for over a year. The cats lived in the calf shed clearing up left over milk from the calf buckets and subsequently a high proportion of the calves developed tuberculosis. Checks of a wide range of wildlife species using this farm, including badgers, were carried out with negative results and the original source of infection remained undetermined. Subsequently, Monies et al. (2000) confirmed tuberculosis in 4 of 12 cats on a premises in Cornwall where 3 months previously an emaciated tuberculous badger had been found. The badger was thought to have passed infection to the cats by contaminating the cats feed bowls outside the house when eating left over food scraps.

9. Control of infected badgers

That cattle can contract infection from badgers under experimental conditions was demonstrated by Little et al. (1982a). A total of 8 calves, exposed to diseased badgers which were shown to be excreting *M.bovis* in their faeces, all became infected mostly within 6 months of exposure. But the conditions of this experiment were necessarily artificial, since for disease security reasons, badgers and calves were housed together on concrete within a brick building. Transmission was thought to have resulted from contamination of the drinking trough or hay that was fed. Six months would appear to be an unduly long period but cattle to cattle transmission can sometimes require protracted exposure. O'Reilly and Costello (1988) found no infection occurred when a total of 22 infected cattle were in close contact with 32 uninfected cattle. Twenty of these were housed with 10 of the infected animals for 6 months. The remainder were exposed for 5 months. Yet of the 22 index cattle used, 20 showed gross lesions of tuberculosis and in 12 small lung lesions were present. In a further trial, Costello et al. (1998) housed each of 10 uninfected steers separately with 2 tuberculin reactor

cattle, for 12 months; no transmission occurred in 6 cases. In the 4 groups where infection did occur, lung lesions were found in the reactor animals in 3 cases and in the fourth a mediastinal lymph node lesion was found. Infection was confirmed at slaughter in 9 of the 10 groups of reactors. These findings indicate that infection of cattle by other cattle may not readily occur. The infectiousness of cattle for others is highly variable and difficult to ascertain but it is apparent that dose and ability to produce fine aerosols of bacilli are important determinants (O'Reilly and Daborn, 1995).
The hypothesis that wild badgers are an effective reservoir host of infection for cattle has been tested at two sites in the south west of England. The larger site, the Thornbury study area of 104 km^2 was delineated by rivers on two sides and motorways on the other two. At the start of the trial in 1975 there were 158 herds, mainly dairy, with a total of just over 12,000 cattle. The area had a history of outbreaks of TB of obscure origin for many years and the situation had greatly worsened just prior to the trial with 16 herd outbreaks in 1973 and 13 in 1974. Infected badgers had been found in the vicinity of a number of the affected farms.

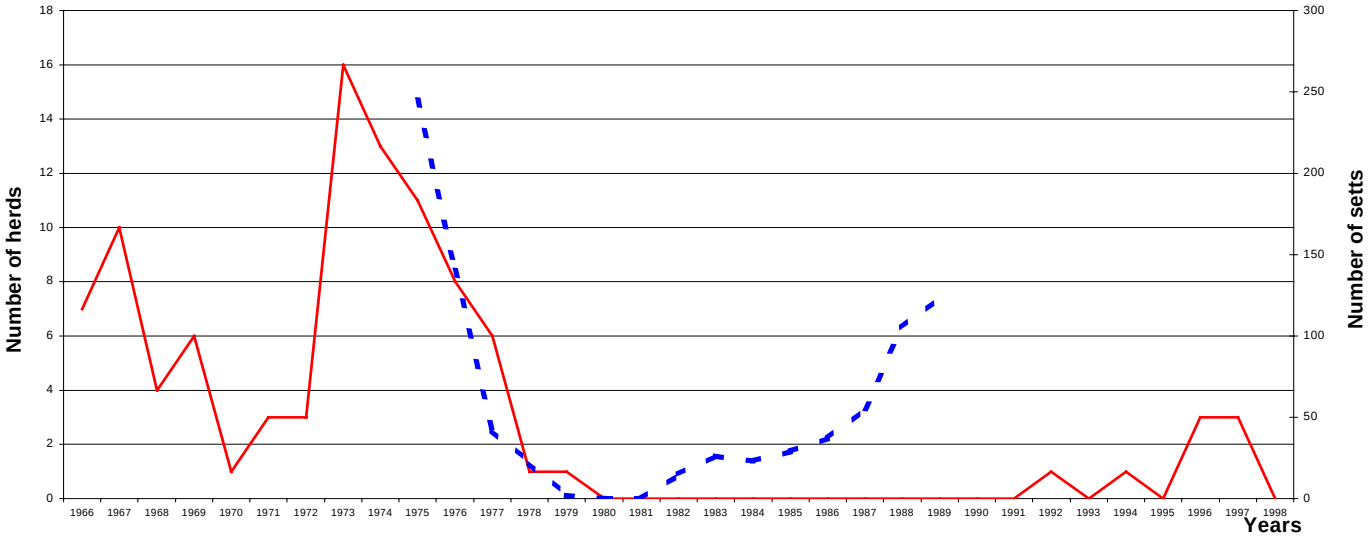
The trial was required to be long term as the reactor rate in the area was starting to decline at the beginning of the exercise. A period of at least 10 years was considered necessary as an apparent cyclical pattern of reactors was noted of 5-6 years between peaks. Any beneficial effect would need to extend through at least one cycle.

The whole area was extensively surveyed for badger setts during the summer of 1975 and control was started in December of that year. Over the next 6 years, all setts were gassed (fogged with anhydrous sodium cyanate – policy ceased in 1981) and many were re-gassed, until there were no signs of badger occupation. Most were cleared during 1976. The last infected badger was found in 1981 when control action was stopped and recolonisation allowed.

Trapping and snaring had also been carried out in the trial area to obtain badger carcases for examination. A total of 223 carcases were obtained from 1972 until 1988 of which 31 were positive for M.bovis. However, up to and including 1976, a total of 130 carcases had been obtained of which 24 (18.6%) were positive. After 1976, when the main clearance was carried out, only 5 positives were found from 93 carcases (5.4%) (Report 1989).
Other wildlife species were also sampled and a total of approximately one thousand individuals from 12 species were examined but none were positive for *M.bovis* (Gallagher, 1980).

From 1980 until 1991 no cases of tuberculosis were found in the cattle in the trial area (*figure 5*). By 1988 when the last sett survey was carried out, most of the area had been recolonised with badgers from the surrounding areas. Subsequently small numbers of herds with confirmed cases of tuberculosis started recurring at a low level mostly at the periphery of the area (J.Smith, personal communication). Analysis of the trial data in comparison with data from herds outside the trial area, on some of which control action had been taken, showed a clear statistically significant advantage of removal of badgers in reducing outbreaks of tuberculosis in cattle (Clifton Hadley et al., 1995b).

Figure 5 **The Thornbury study area – badger removal trial. Cattle herds with confirmed TB and badger sett numbers.**



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

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

The second clearance exercise was carried out in an area of south Dorset of 12 km^2 where there was a major outbreak of tuberculosis involving 7 herds (Wilesmith et al., 1982; Little et al., 1982b; Little et al., 1982c). Similar dramatic results occurred during this trial with no cases of infection for 7 years after badger clearance (Krebs, 1997). After that the farm management system changed and the stock farming was replaced by arable.

During this trial Wilesmith et al. (1982) noted that as a result of the high frequency of tuberculin testing of these herds a clear seasonal pattern of reactors to the test was apparent during May/June. According to Francis, (1947) in cattle the median time from infection to development of hypersensitivity, is about one month. Thus in four years of this trial in which testing was of sufficient frequency to indicate a seasonal effect, cattle were at greatest risk of infection in April/May. This finding is in marked contrast with the pattern of infection in herds when the prevalence of infection in cattle was high. The major risk was then of cattle to cattle spread which was at its greatest during the winter housing period (Wilesmith et al., 1982). However, the pattern of peak exposure in spring seen during this trial does fit the observed frequency of maximal occurrence of cases of disease and deaths of badgers due to tuberculosis found in Gloucestershire (Gallagher, 1998).

In Ireland an extensive trial of badger clearance has been carried out in Co.Offaly. The intervention area was 738 km^2 which included a buffer zone 1.5 km^2 wide. Surrounding this area there was a control zone of 1,456 km^2. Removal of badgers was carried out using snaring which is considered to have about an 80% capture success rate. A limited number of badger removals had to be carried out in the control zone in response to serious outbreaks. Despite these constraints the reactor numbers over the

seven year period of the project fell in the intervention area from 3.91 APT (reactor animals per thousand animal tests) to 0.46 a fall of 88%. In the control area the fall was 3.39 APT to 2.10 (38%). Over the same period the national APT increased by 19% from 2.70 to 3.20 (Eves, 1999).

10. Conclusions

The relationship of badgers with bovine tuberculosis is probably fairly ancient extending over much of the twentieth century and probably considerably longer as elucidated well by Little et al. (1982b). Infection is likely to have seeded in badger populations at a time when it was rife amongst cattle. In areas where the badger has remained sufficiently populous since those times, it has probably persisted. It could reasonably be speculated that the adaption to infection shown by the badger may be evolutionary. Whilst genetic selection against disease may have been occurring in the badger population over a lengthy period, no such selection has occurred in cattle. Any exposure of cattle to tubercle bacilli detected by tuberculin testing has resulted in the slaughter of that animal thus eliminating any selection pressures since the start of testing in 1935.

The evidence so far indicates that the badger is the primary reservoir of tuberculosis infection in the south west of England and probably in many other areas of the country. Most of Ireland appears similarly affected.

The finding that a relatively large number of infected but apparently healthy badgers with no gross lesions, are likely to have arrested lesions resulting from an early immune reaction, indicates how well this species has developed as a maintenance host for *M.bovis* infection. Certainly it appears that some badgers self cure of infection whilst the majority may show a long dormancy or latency and it is not unreasonable to postulate, that some infected badgers may never develop disease. Others succumb to florid disease and whilst those with lesser lesions may merely act to maintain infection amongst the badger population, it is probably the smaller number with advanced disease, that result in overspill from this reservoir of infection, to infect cattle and to a lesser extent other species.

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