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Cause of ill health and natural death in badgers in Gloucestershire

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During the period 1973 to 1976 inclusive, 1206 badger carcases were examined for evidence of tuberculosis and other diseases. Tuberculosis was the major cause of natural death, killing 39 per cent of the "natural death" cases, followed by bite wounding and starvation. Road traffic accidents were the greatest single cause of death.

THE finding of bovine type tuberculosis in badgers in parts of the south west of England (Muirhead and others 1974, Muirhead and Gallagher 1976) has stimulated increased interest in this species in recent years. Much of the published work to date has been concerned with the natural history of the badger, and no comprehensive reviews of the causes of ill health have been found in the literature by the authors. This article reports on various conditions in wild badgers from the Gloucestershire area found during routine post mortem examination of a large sample of animals obtained for screening for evidence of tuberculosis.

The European badger (*Meles meles*) appears to be an extremely robust animal. With the possible exception of tuberculosis in England, there are no reports of widespread disease conditions decimating large populations. In Switzerland, from 1967 to 1968 inclusive, rabies cases in the badger accounted for 2·3 per cent of the total number of 570 positive diagnoses of this condition in all animals. Foxes comprised 84·8 per cent of cases diagnosed, and cohabitation with the fox was considered to be the source of the badger infection (Steck 1968). However, compared with foxes, badgers appear to have a degree of innate resistance to rabies (Bedford 1976).

Both salmonella and leptospira infections have been found in badgers in the south west of England (Wray and others 1977, Salt and Little 1977) but neither infection appeared to be associated with overt disease. Approximately 10 per cent of badgers sampled were carrying salmonellae and *Salmonella agama* was the principal serotype found. The leptospires isolated belonged to the serogroups Australis, Javanica and Hebdomadis and serological evidence of infection by one or more of these serogroups has also been reported from several other countries. In Switzerland, 4 per cent of badgers tested showed antibodies to the Australis serogroup (Sebek and Rosicky 1975). Bolotzky and others (1974) found antibodies to the Hebdomadis serogroup in Russian badgers and Fennestad and Borg-Petersen (1972) found antibodies to both Javanica and Hebdomadis serogroups in Danish badgers.

Symptomless piroplasmosis due to an unidentified babesia-like organism has been reported in badgers from south west England (Peirce and Neal 1974, Peirce and Gallagher 1974). Parasitaemias were low and the likely vectors appeared to be *Ixodes canisuga* and *I hexagonus,* both of which may be found quite frequently on badgers in this area during mid to late spring and early autumn.

A severe outbreak of canine distemper affecting several species in a zoological park in America also affected captive badgers (Armstrong and Anthony 1942), yet there are no reports of this condition in wild populations. Schlegel (1933) reported the occurrence of fairly widespread lungworm infestation in the Black Forest area of Germany, and many cases were fatal. Otherwise, reports of deaths relate to either single cases or to small numbers of animals only.

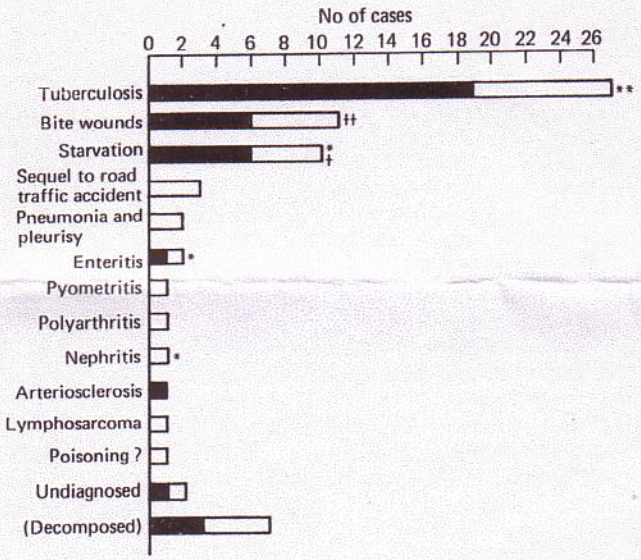

FIG 1: Causes of natural death in badgers—70 cases
*Case in which arteriosclerosis was a contributory cause
†Case with only minor lesions of tuberculosis or no gross lesions from which *M bovis* was isolated
■ Males
□ Females

Materials and methods

Badgers killed in road traffic accidents or found dead from apparently natural causes were submitted to Gloucester veterinary investigation centre for autopsy. Badgers were also killed for diagnostic purposes on farms where outbreaks of tuberculosis in cattle had occurred. Detailed examinations were carried out post mortem and, in the absence of any gross lesions, a selection of lymph nodes was taken from all animals for mycobacterial isolations (Gallagher and others 1976). Both cultural and guinea pig biological methods were used for isolation, as previously described (Gallagher and Horwill 1977). Routine bacteriology and histopathology were carried out as necessary where other conditions were observed. Age, based largely on Neal's criteria (1977), sex, and body-weight were recorded in each case.

Results

During the years 1973 to 1976 inclusive, 1206 badger carcases were autopsied. Of these, 460 were road traffic accident cases (RTA) and 70 were natural deaths (ND). The remaining 676 were animals killed for diagnostic purposes (K). The causes of natural deaths are summarised in Fig 1.

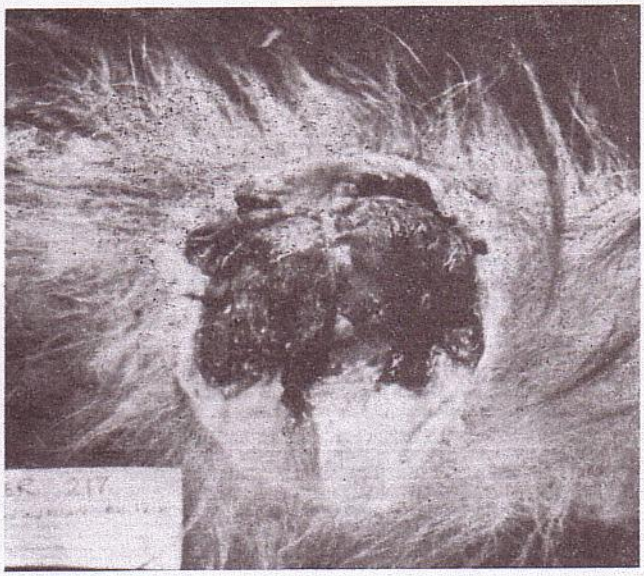

FIG 2: Tuberculous bite wound in the thigh of a young female badger. The wound has been incised downwards and opened to show normal muscle contrasting with the blackened necrotic muscle of the bite lesion. The scattered white dots are caseous/calcified particles

Tuberculosis due to *Mycobacterium bovis* was found in 233 badgers (19·3 per cent). Males showed a greater prevalence of infection with 22 per cent infected compared with 17·3 per cent of females. Prevalences of total infection rates varied between sampling groups, 14 per cent of the RTA sample, 21·6 per cent of the K sample and 42·8 per cent of the ND cases were infected with *M bovis*. In both the RTA and K sample groups the nature and severity of lesions were similar, 71·8 per cent showing gross lesions at autopsy and 28·2 per cent showing only non-specific enlargement of one or more lymph nodes, or no abnormalities. These latter cases were considered to be latent infections as *M bovis* was isolated from lymph node collections taken at autopsy.

Of the total of infected animals in the RTA and K sample groups 47·6 per cent showed tuberculous lung lesions. Approximately a third of these were in the advanced stages of disease showing miliary tuberculous pneumonia, usually accompanied by renal lesions. All of these cases would probably have died within a short period of time as a result of this condition. A similar proportion of animals had multiple tuberculous lung lesions of lesser severity often accompanied by renal lesions. The remainder showed only minor lung lesions.

In the ND sample tuberculosis was the most frequent single cause of death, killing 27 (39 per cent) of the 70 cases examined. *M bovis* was isolated from a further three cases but either no lesions or only minor lesions were found and death had resulted from other conditions. Of the 27 tuberculosis deaths 19 were males. Nine of the 27 were aged and four were two years old or less. In 15 of the 27 animals (57 per cent) which had died of tuberculosis, nine of them males, the lesions present indicated that infection had been introduced by a bite wound inflicted by another badger. Characteristic canine tooth penetration marks were found, usually in the head and neck region, with the subsequent development of tuberculous abscesses. Such abscesses measured up to 6 cm by 3 cm, often showed a nodular lining, and were filled with yellowish-brown pus. Sometimes the wounds were ulcerous (Fig 2). Infections originating from bite wounds were seen in all the sample groups and were present in 14·1 per cent of all the infected badgers. The resultant form of tuberculosis generally appeared very severe and of an acute nature (Fig 3). Haematogenous generalisation with the development of miliary lung and kidney lesions was seen in eight of the 15 fatal cases and in one case tuberculous encephalitis and endocarditis were also seen.

A form of acute tuberculous pneumonia was seen only in bite introduced infection and was characterised by bilateral widespread amorphous tumescent lesions bordered by intense congestion. Histologically, extensive necrosis was found throughout the lesions, accompanied by an acute inflammatory reaction. The orderly development of granulomata seen in infections of respiratory origin was lacking.

The remaining cases of tuberculosis in the ND group were ascribed to respiratory infection, and the lung lesions appeared to be of a more chronic nature.

Non-tuberculous bite wounds were the second most frequent cause of natural death (11 cases). Mixed infections of beta-haemolytic streptococci, *Staphylococcus pyogenes*

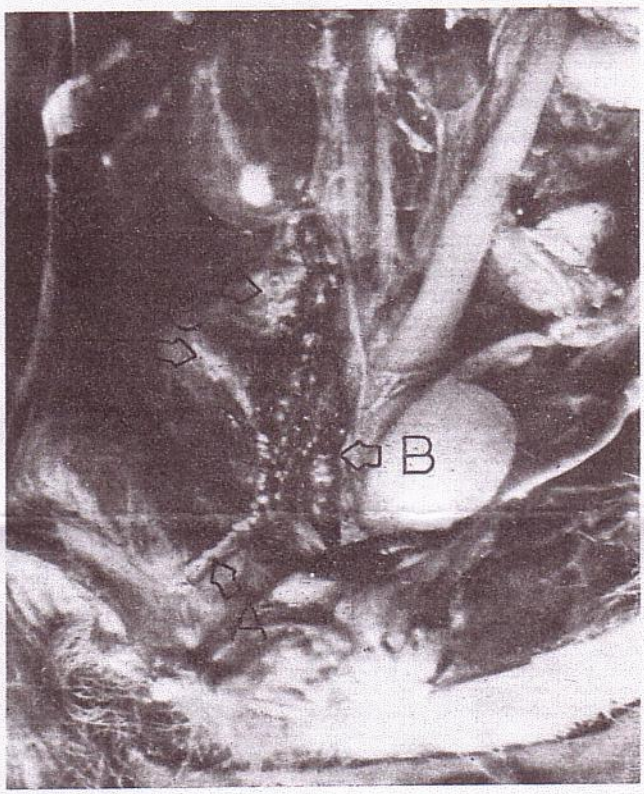

FIG 3: Dissection of a badger with generalised tuberculosis showing:
(A) Caseous calcified material occluding the lymph duct from a bite wound in the thigh (shown in Fig 2)
(B) Caseous calcified lesions are present in the incised internal iliac node and also (C) in the area of a second bite wound in the lumbar region

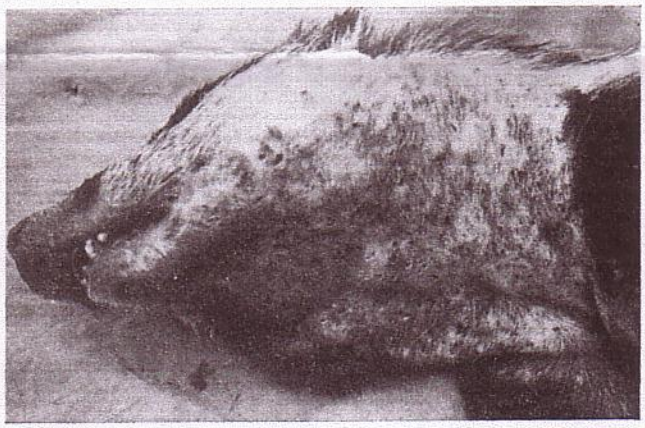

FIG 4: Male badger, shaved to show multiple bite wounding. Note swelling of the neck due to massive fatal haemorrhage resulting from severance of the jugular vein and smaller vessels

FIG 5: Rump area of badger shown in Fig 4 shaved to show extent of bite wounding

and coliforms had been introduced into the wounds and extensive abscesses had developed, with death resulting from septicaemia/toxaemia. In three cases the bites had penetrated the chest wall and a fatal pyothorax had developed. The thoracic cavity often contained from 100 to 300 ml of liquid blood-tinged pus and fibrin clots. In one case, in which a large number of bites had been inflicted on a two-year-old male, a jugular vein and other smaller vessels had been severed (Figs 4 and 5). Death was due to extensive trauma and haemorrhagic shock.

Records of all recent bite wounds found at autopsy were kept only during the three-year period 1975 to 1977 inclusive.

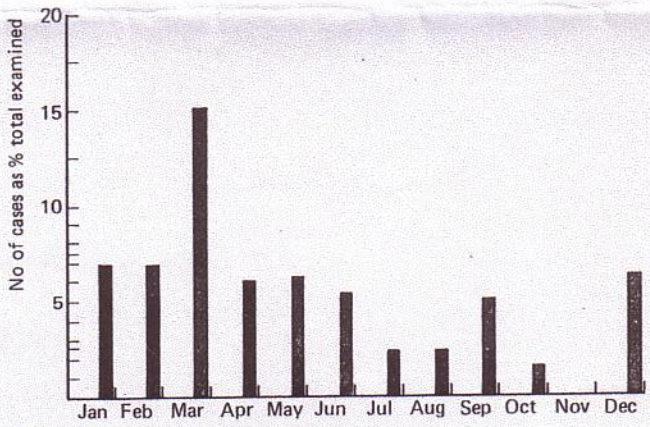

FIG 6: Seasonal distribution of cases of recent bite wounding

FIG 7: Partially healed bite wound with skin loss at the tail base of a male badger

Bite wounds, tuberculous or otherwise, were found in 6 per cent of badgers examined. As shown in Fig 6, a peak incidence occurred in March. Some wounds were of several days standing, showing signs of swelling, congestion and exuding serous fluid or pus. Others were of some weeks duration showing abscessation with granulation. Multiple bite wounds were usually present and numerous paired holes consistent with canine teeth penetration were seen. The face, upper neck, shoulder and rump areas were the usual sites and frequently bites were seen at both front and back ends. Skin tearing was common, especially in wounds inflicted around the base of the tail (Fig 7). Healed bite wound scars were seen in a very large proportion of males and in many of the females.

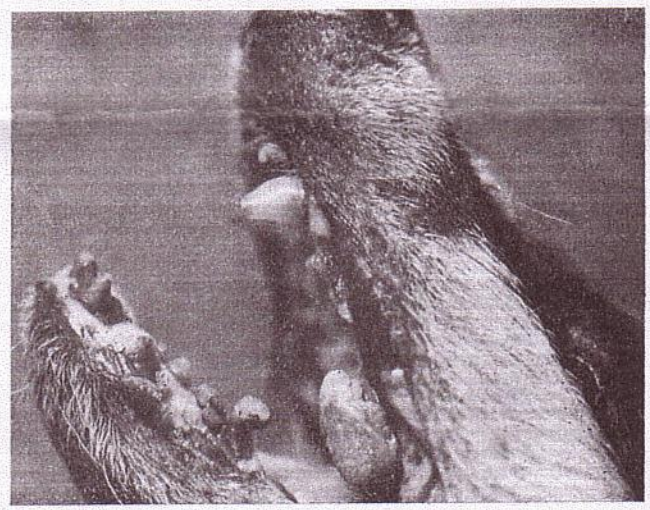

FIG 8: Marked tooth wear and tartar formation in an aged badger

Starvation was the cause of death in 10 cases. Five of these were recently weaned cubs which were found during midsummer and apparently had been unable to forage successfully. The other five cases involved aged animals which were obviously unable to feed properly due to extensive tooth wear and tartar formation (Fig 8).

Arteriosclerosis was considered a probable cause of death in one case and a possible contributory cause in a further five cases of natural death. The aortas of 145 badgers from all sampling groups were examined carefully following the initial finding of this condition and 26 per cent showed arteriosclerotic lesions. These varied from one or more 3 to 4 mm diameter raised plaques of firm tissue on the intima of the ascending aortic arch, to complete annular calcification of both the aortic arch and a large part of the thoracic aorta. In the latter area plaques were usually present at the exits to the intercostal arteries. Small plaques were sometimes seen in the abdominal aorta near the orifices to the various arterial off-shoots. Lesions were more frequently observed in older badgers and severe lesions were seen only in aged animals but were not consistently accompanied by signs of general ill health. Indeed some affected badgers appeared in very good bodily condition.

Pneumonia and pleurisy, nephritis, enteritis, polyarthritis and lymphosarcoma were the remaining causes of natural death. The aetiology of the pneumonia and enteritis cases was not established. In the former there was extensive consolidation of the anterior lobes and the dependent portions of the diaphragmatic lobes of both lungs. In the latter case there was a severe catarrhal enteritis affecting the entire length of the small intestine and parts of the large intestine were also inflamed. No salmonellae were isolated. The suspected lymphosarcoma case showed emaciation and enormous enlargement of all the carcase nodes, together with variable enlargement of the visceral nodes. The enlarged follicles of the spleen were very pronounced. Gross involvement of the other viscera was not apparent and, unfortunately, autolysis precluded histological confirmation of this condition. The polyarthritis case was a four- to five-year-old female showing lesions of chronic erosive arthritis with pronounced thickening of the joint capsules, producing gross joint enlargement. The knee and stifle joints on both sides were severely affected and all the remaining limb joints showed lesions of variable severity. No bacteria were isolated from the joints.

The pyometritis case, an aged female, showed a foetid liquid brown vaginal discharge which had developed subsequent to recent parturition. Lung penetrations, haematomata formation and fractures, probably sequelae to earlier road traffic accidents resulted in death in three cases. One case of possible poisoning was encountered. This animal was in good body condition and showed lesions of haemorrhagic gastritis only

Of the remaining nine cases, for which a diagnosis was not reached, seven were too decomposed for a reliable diagnosis to be made. The carcase of one of the remaining two cases had been scavenged, and the other animal was in poor body condition but showed no obvious gross abnormalities at autopsy.

Miscellaneous conditions encountered in the RTA and K sample groups were few. Severe dental attrition with tartar formation accounted for loss of body condition in some aged animals, but many with marked dental wear had apparently successfully adjusted their diet and were in very good body condition. Single cases of vegetative valvular endocarditis and cutaneous fibropapilloma of the face and forelegs were encountered. In the latter, examination by electron microscopy for virus particles and attempted virus isolation gave negative results.

Haemangiomata, sometimes quite extensive, and polyploid hyperplastic nodules, similar to those seen in old dogs (Jubb and Kennedy 1970), were seen on rare occasions in the livers of older badgers. Cystic kidneys were occasionally found but the condition was benign and not usually very extensive. In only one instance was a renal calculus found and this exactly mirrored the architecture of the distended renal pelvis.

FIG 9: Overgrowth of fore claws from reduction in digging activity in a debilitated badger

In animals from all the sample groups, healed lesions of rupture of the liver and/or spleen were frequently seen, indicating an earlier severe trauma such as an RTA.

Debilitated badgers, usually cases of tuberculosis, dental attrition or arteriosclerosis, commonly showed heavy burdens of lice and fleas (*Trichodectes meles* and *Paracerus meles*). Such animals often "seethed" with these ectoparasites and typically showed a very low body-weight (4·5 to 6 kg), very long fore claws, probably due to reduction in wear through cessation of digging activity (Fig 9) and severe loss of suborbital fat giving a "deep sunken-eyed" effect (Fig 10).

FIG 10: Loss of suborbital fat in an emaciated badger producing a "sunken eyed" effect. The eye sockets appear almost empty due to retraction of the eye almost 1 cm from the medial canthus. The badger died from tuberculosis with a body-weight of 5 kg

Firm white spherical lesions measuring approximately 1 mm diameter were frequently found scattered throughout the lungs of animals in all sample groups. These lesions and associated minor areas of pneumonitis were not considered to be of clinical significance. On histological examination the lesions were found to be granulomata, each containing a large central spherical periodic acid Schiff (PAS) positive inclusion typical of adiaspiromycosis. The fungi responsible for this condition are common in soil and lesions have been encountered in the lungs of other digging animals such as moles (McDiarmid and Austwick 1954), small rodents, hares and occassionally man (Ainsworth and Austwick 1973).

Discussion

Clinical observations of sick badgers are rare because this nocturnal animal is rather shy and retiring. The fatal case of arteriosclerosis was one of the few seen alive. This was an aged female seen walking aimlessly in a field during the early afternoon. A mild lameness of one hind leg was noted and the approach to the animal initiated excitement and incoordination. The lameness became severe and the affected leg was dragged as the animal circled in distress before collapsing and dying. Gross distension of the aortic arch and extensive arteriosclerotic lesions of the thoracic and anterior abdominal aorta were found at autopsy. Constricting lesions were seen at the base of the renal arteries and also of the femoral artery to the affected leg.

Another sick badger observed alive was one which had contracted tuberculosis through a bite wound. It was seen staggering out of a hedgerow in late morning and when approached showed no apprehension. Silently and dejectedly, stumbling into tufts of grass, it followed closely behind the farmer as he crossed the open field. Later that day it was found dead in a hedgerow and autopsy revealed severe miliary pneumonia and nephritis, as well as tuberculous endocarditis and encephalitis.

Few other tuberculous badgers were seen alive but in those that were, activity during the late morning or afternoon, dullness, and general weakness were the usual signs observed. Both these and carcases of badgers which had died of tuberculosis were often found in unusual sites, such as a pig sty, horse loose-box, under bins in a farm building and in gardens. It appeared that ailing badgers might leave their home set and be attracted to an easier source of food to be found in farm buildings and gardens (Muirhead and others 1974).

A difference in the incidence of tuberculosis was noticed between the road traffic accident sample (14 per cent infected) and the K sample (21·6 per cent infected). The higher incidence in the latter was considered attributable to sampling bias since badgers were killed only for diagnostic purposes in locations where outbreaks of cattle tuberculosis had been found.

Our findings suggest that tuberculosis is probably the major cause of natural death in badgers in the Gloucestershire area. Almost 40 per cent of deaths were due to this condition. Non-tuberculous bite wound infections were the next most important cause and both these conditions are likely to be related to population density.

Clements (1977) mapped the density of badger sets over most of Britain and considered Gloucestershire to have a set density of over 100 per 10 km². However, Cheesman (personal communication) found a density of over 200 sets per 10 km² in areas of the Cotswold Hills, and at one site he found the equivalent of 400 sets per 10 km².

These hills, which are especially heavily populated, provide an abundance of both food and habitats. Earthworms are the food most sought after by badgers (Kruuk and Parish 1977) and these are plentiful in the cattle pastures along the hills. Deciduous woodland is a preferred habitat for badgers (Neal 1977, Middleton and Paget 1974), and both this and an ideal digging soil of sand beneath a solid roof of hard oolite limestone are provided. Badger sets are far more numerous here than in surrounding areas and the incidence of tuberculosis differs accordingly. Of those badgers examined from the Cotswold Hills, 27 per cent were found to be infected with tuberculosis, whereas in the countryside below the hills where badgers are far less abundant, only 10 per cent were infected.

From the high density of sets throughout much of Gloucestershire and Avon there is little doubt that the territories of the different social groupings must adjoin over large parts of this area and especially along the Cotswold Hills.

Badgers are strongly territorial and will defend their ground ferociously (Kruuk 1978). Border skirmishes and fierce fighting appear to occur frequently and may not only be the cause of severe injury and death, but also an important means of transmission of tuberculosis from one group to another.

Pulmonary tuberculosis is likely to cause contamination of the mouth from coughed up lung discharges, and bites from such animals may result in the injection of tubercle bacilli on the teeth into the subcutis, muscle or even into blood vessels. Since tuberculosis lesions in this species usually contain large numbers of bacilli (Gallagher and others 1976) bite wounds may present a heavy challenge resulting in a fulminating disease. In experimental animals infection by these aberrant routes has been shown to result in rapid generalisation and death after a short incubation period. Rabbits given 1 mg of culture of *M bovis* by the subcutaneous route died 40 to 80 days post infection and calves given 50 mg by this route died within a similar period. Infection of rabbits by the intravenous route with 1 mg of *M bovis* culture produced death in 15 days. In contrast direct respiratory infection results in a longer incubation (Francis 1958).

The higher incidence of tuberculosis in males (22 per cent) compared with females (17·3 per cent) is probably a reflection of the greater territorial aggression displayed by the male, together with the increased chance of contact with infected animals to which the male is exposed because of his wider ranging activity.

In the Gloucestershire area a peak incidence of bite wounding occurs in March. While working this area Cheesman (personal communication) observed the maximal territorial and mating activity during February-March and it seems likely that the stimulus for most of the fighting was violation of territorial boundaries. Most of the bite wound induced tuberculosis cases were found at about this time.

Neal (1977) considered that the death of young cubs probably due to starvation/mismothering may result in the loss of many cubs before they are of an age to emerge from the set. Further deaths are likely following weaning, because of an inability of some cubs to forage successfully. He also found evidence that another crop of deaths occurs when one- to two-year-old badgers roam to new territories. This migration is likely to result in increased risk of road traffic casualties and an increase in injuries and infections due to involvement in territorial fights. Neal (1977) and Corbet and Southern (1977) considered that these factors are likely to reduce the number of badgers reaching three years of age by at least half, but thereafter the death rate appears to be very low.

These figures differ somewhat from those obtained during our work in that few cubs were found dead. However, both Neal's figures and those quoted by Southern and Corbet were based on the assumption that mature females normally produce a litter of cubs each year, although occasionally missing a year. We estimated from autopsy that less than half the mature females were pregnant. In a small check sample of 23 carcases, this was confirmed. If this is representative, there are far fewer cubs born in this area than might be expected. This apparently reduced fertility suggests the operation of an intrinsic population control mechanism in the high density population of this area, as has been found in other species such as the rabbit (Myers and others

1971).

With the exception of starvation, bite wounding and tuberculosis, the variety of miscellaneous conditions encountered appears to have little effect on the population dynamics of badgers in Gloucestershire.

The total impact of tuberculosis on the badger population in the Gloucestershire area is extremely difficult to assess. Not all badgers dying naturally die above ground and, indeed, Neal (1977) considered that the majority die within the set. The non-recoverable below-ground mortality, as well as those surface deaths which remained unlocated, make estimates of total mortality based on these figures alone quite unrealistic. However, analysis of the natural death cases does yield information on the relative incidence of the various conditions encountered.

Four to 5 per cent of all the badgers in this survey had miliary tuberculosis lesions from which they had died or were dying. The corresponding figure for the Cotswold Hills only, where the overall incidence of tuberculosis is 27 per cent, will be higher, probably at least 10 per cent. Similar percentages of badgers showed well developed, active, but not miliary lesions of tuberculosis of the lungs or of both lungs and kidneys, and most lesions were progressive although the rapidity of their progression is uncertain. Resolved lesions were extremely rare. In both the RTA and K groups, with the exception of tuberculosis, the overall incidence of disease was low and their relative incidences similar.

The frequent finding of healed lesions of rupture of the liver or spleen consistent with RTA damage, indicates that badgers may commonly be involved in traffic accidents. The very high density of badgers in the Gloucestershire area must increase the likelihood of such encounters and indeed comparison of the numbers of badgers in the RTA and ND sample groups shows that, in terms of mortality, the effect of disease on the dynamics of the adult badger population is far less than that produced by motor vehicle accidents. In the period from 1973 to 1976 inclusive, when 70 badgers were found dead from natural causes, 460 road traffic casualties were found. Although many chronically sick badgers may die below ground, from the low proportion of sick animals seen in the RTA and K groups there seems little likelihood that the toll due to natural causes can in any way approach that due to the motor vehicle.

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