



## TUBERCULOSIS AND OTHER MYCOBACTERIAL DISEASES IN CATS

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### INTRODUCTION

Several species of mycobacteria can cause disease in veterinary species, being either primary pathogens, or becoming pathogenic under certain circumstances.

Tuberculosis can be caused by a number of different, but closely related, bacteria. Members of the tuberculosis complex include *Mycobacterium* (*M.*) *tuberculosis*, *M. bovis* and *M. microti*. *M. tuberculosis* causes over 90% of tuberculosis in man, but rarely infects other mammals, except for dogs. *M. bovis* is the main cause of tuberculosis in cattle. It can also infect various other mammals, including humans, dogs, cats and pigs. *M. microti* causes tuberculosis in voles and cats (in the latter it was previously termed *M. microti*-like and has culture characteristics between *M. tuberculosis* and *M. bovis*). *M. avium* causes tuberculosis in birds, and can also infect man, dogs and cats. *M. avium*-intracellulare complex (MAC) are mainly saprophytic organism, but it is often considered with the tuberculosis complex, since it can cause clinical disease indistinguishable from that caused by members of this group.

Other potentially pathogenic mycobacteria include *M. lepraemurium*, which causes leprosy in rats, and a similar, or possibly the same, organism which causes feline leprosy. Opportunistic mycobacteria are usually saprophytic, but a number of species have been reported to cause disease in cats. These include *M. chelonae*-abscessus, *M. fortuitum* / *peregrinum* group, *M. smegmatis*, *M. phlei*, *M. genavense*, *M. simiae*, *M. thermoresistibile*, *M. xenopi* and *M. terrae* complex.

Mycobacterial syndromes seen in cats therefore include tuberculosis, feline leprosy and opportunistic mycobacteriosis. All three syndromes have been reported in the UK, where the majority of cases appear to be cutaneous in nature. All three syndromes can present with nodules, draining tracts and/or ulceration. In some cases, the disease may become generalised secondary to skin inoculation, but only occasional cases present with primary systemic disease. Where systemic disease is seen, infection with a member of the tuberculosis group is most likely. In many cases of feline mycobacteriosis, infection can be related to percutaneous injury, contamination via soil or the presence of devitalised tissue. These factors tend to be reflected in the distribution of the lesions.

## TUBERCULOSIS

### CLINICAL BACKGROUND:

#### Epidemiology and aetiopathogenesis:

In cats, tuberculosis has classically been described as being caused by *M. bovis*. Historically, infection resulted from the ingestion of milk from tuberculous cattle. With the virtual eradication of tuberculosis from the national herd there has been a marked decline in the prevalence of the disease seen in cats.

Currently, tuberculosis in cats is seen infrequently. When it is diagnosed it is usually caused by infection with either the cattle form of the infection (*M. bovis* [41 cases]) or the vole form (*M. microti* [22 cases - recent figures from Keith Jahans, Veterinary Laboratories Agency, 2004, data collected since 1980]). Infection of cats with *M. tuberculosis* is incredibly rare, probably because cats are naturally very resistant to it. (Interestingly, this is quite different from the picture seen in humans; where over 90% of cases result from infection with *M. tuberculosis*, approximately 1% is caused by *M. bovis* and disease due to *M. microti* is incredibly rare). Cats can occasionally develop disease due to infection with MAC.

The current epidemiology of tuberculosis in cats is still unclear. While we do see occasional clinical cases, few are believed to relate to direct infection from cattle. This is because when tuberculosis is gained by drinking contaminated cows milk the infection settles within the cat's intestines, and disease results in diarrhoea and weight loss. It is probably because almost all cows' milk is now pasteurised that this type of tuberculosis is currently very rare. The tuberculosis that we now see most frequently in cats affects their skin; where it causes non-healing sores and lumps that fail to heal. This is often associated with swollen lymph nodes, especially those under the chin, and some cases show only the swollen lymph nodes. In chronic cases, where the infection has spread to the cat's lungs, they may develop a soft cough or have difficulty breathing.

It is important to try to determine how cats are becoming infected. If we look into possible risk factors we find that most of the cats are keen hunters, regularly catching small rodents. Interestingly, studies have shown that in the UK wild mice and voles quite often carry *M. microti*, while moles and rats can carry *M. bovis*. It therefore most likely that cats become infected by hunting small wild rodents. This also accounts for the distribution of the skin lesions seen on these cats, which occur most frequently on the face and legs, i.e. the areas most likely to be bitten when playing with prey. In some areas of Britain *M. bovis* has become endemic in badgers. While cats and badgers rarely interact directly, there may be a potential risk for cats to become infected via local environmental contamination. *M. bovis* can also be endemically present in many other species of free-ranging wildlife, e.g. deer; so the risk of feline infection will vary in each country dependent on the likely interaction between these species and domestic cats.

All members of the tuberculosis complex pose potential zoonotic risks. However, to date, there have been no reported cases of cats passing tuberculosis onto humans. By far the greatest tuberculosis risk to humans is spending time with infected humans or, less frequently, handling infected cattle.

Interestingly, *M. tuberculosis* and *M. bovis* can both cause reverse zoonoses and there have been a small number of cases where humans have infected their cats (usually with *M. bovis*). This may be significant because with the current increase in human tuberculosis associated with HIV infection and poor housing, we may see a concurrent increase in feline tuberculosis caused by these organisms.

#### Predisposition:

Most cases of feline tuberculosis are probably subclinical in nature. Infection usually occurs after protracted exposure, e.g. repeated exposure to infected small mammals, living on a farm housing tuberculous cattle, or living for prolonged periods with infected humans or poultry. Tuberculosis is therefore seen mainly in adult cats, and interestingly, is seen most commonly in males. No evidence of immunosuppression has been found and those cats tested for FIV and FeLV have usually been negative.

#### Clinical signs:

Depending on the route of infection, affected cats may present with systemic signs related to the alimentary, and/or respiratory tracts, or with localised disease affecting the skin. Currently, the most usual presentation for tuberculosis in cats is the cutaneous form, with respiratory and alimentary forms being seen less frequently.

In the cutaneous form the lesions probably arise from infected bite wounds, local spread or haematogenous dissemination to the skin. The lesions often involve the face, extremities, tail base or perineum, i.e. "fight and bite sites". Less frequently they involve the ventral thorax and tail base. They generally take the form of firm, raised, dermal nodules, ulceration, or non-healing wounds with draining sinus tracts. Extension of granulomatous tissue may in some cases involve the subcutaneous structures, muscle and/or bone. Skin lesions are commonly associated with either local or generalised lymphadenopathy. On occasion, submandibular or prescapular lymphadenopathy may be the only clinical finding.

When the infection spreads to the lungs, or where it is acquired through inhalation, tubercles arise in the lungs and/or hilar lymph nodes and affected cats present with weight loss, anorexia, dyspnoea and coughing.

In the alimentary form, tubercles arise in the intestines and/or mesenteric lymph nodes. Affected cats commonly develop intestinal malabsorption and present with weight loss, anaemia, vomiting and diarrhoea. Occasionally tubercles arise in the tonsils, resulting in signs of oropharyngeal disease.

A range of clinical signs may be seen with disseminated disease. These include splenomegaly, hepatomegaly, pleural or pericardial effusions, generalised lymphadenopathy, weight loss and fever. Lameness may result from bone involvement.

Granulomatous uveitis and signs referable to central nervous system involvement have been seen in some cases.

#### DIAGNOSTIC TECHNIQUES:

**Non-specific tests:** A thorough evaluation of the patient is necessary to assess the extent of local infection and the degree of systemic involvement. Changes in serum biochemistry and haematology, if present, are non-specific and vary with the severity of the disease. However, hypercalcaemia has been seen in a number of cases and appears to correlate with a poorer prognosis. Radiography can be useful in the appraisal of lung involvement. However, changes are very variable and include tracheo-bronchial lymphadenopathy, interstitial or miliary lung infiltration, localised lung consolidation, or pleural effusion. Abdominal radiography may reveal hepato- or splenomegaly, abdominal masses, mineralised mesenteric lymph nodes, or ascites. Bone lesions tend to consist of areas of bony lysis and sclerosis, osteoarthritis, discospondylitis or periostitis.

**Specific tests:** Specific tests for the diagnosis of tuberculosis have been investigated in cats, but have generally proved unhelpful. Unlike other species, cats do not react strongly to intradermally administered tuberculin and the results from intradermal skin testing are unreliable. Tests for specific serum antibody responses have also proved unhelpful.

To confirm mycobacterial involvement, aspirates and/or biopsy samples of affected tissue should be stained with Ziehl Neelsen (ZN) or other specific special stains. The number of acid fast bacilli seen within affected macrophages may be variable, depending on the species of mycobacteria involved, the location of the granuloma and, probably most importantly, the nature of the cat's immune response. While finding acid fast bacilli confirms the presence of mycobacteria, it is essential to culture the organism to determine the exact species involved. Once the species has been identified it is possible to evaluate zoonotic risk, potential sources of infection, and feasible treatment options. Unfortunately, many samples that are seen to have ZN positive organisms fail to culture positive, and even those that do typically take approximately two months.

#### Correct handling of biopsy material:

In practice, this usually involves taking a biopsy from a case where mycobacterial disease is only one of a large number of possible differential diagnoses. If in-house facilities are available for ZN staining, this can be performed on aspirates or biopsy impression smears. However, in most cases biopsy material must be sent to a veterinary diagnostic laboratory. It is practical to collect the biopsy, cut it into three pieces, fix one in formalin for histopathological examination and ZN staining and, pending results, place one in a sterile container and freeze it. Where other bacterial infections are suspected, the third sample should be sent unfixed for routine bacterial culture at which time ZN staining can also be requested. That way, if the sample is found to have ZN positive organisms, the frozen portion can be defrosted and sent to either the Veterinary Laboratories Agency or a Mycobacterial Reference Laboratory for specialist culture (see end of text for addresses).

Until the organism has been properly characterised, it should be considered a potential human pathogen.

Whenever handling potentially tuberculous material it is necessary to take certain precautions. In the UK, the law dealing with material KNOWN to be tuberculous is very exact and requires the use of specialist laboratories. However, the law relating to material taken from animals where tuberculosis is only one of a number of possible differentials is quite different. In the latter case routine aseptic practices are generally adequate, although gloves should be worn when handling either the biopsy site or the biopsy material. ZN staining of aspirates or biopsy impression smears may be performed where suitably equipped in-house laboratories are available (correct work surfaces, separate refrigerator, etc.). However, before undertaking in-house diagnostics it is prudent to contact the local Health and Safety Executive for guidance.

While tuberculosis in cats is not currently a noticeable disease DEFRA expects to make the diagnosis of *M. bovis* infection notifiable in all mammals (except humans) by the end of 2004. It is therefore essential to inform the local DEFRA office of any confirmed cases, and when a confirmed case is euthanased it is advisable to have the body cremated.

Histopathology:

Histopathology of affected tissue generally reveals granulomatous inflammation, with foamy macrophages containing variable numbers of acid fast bacilli.

MANAGEMENT:

Interim Management: Deciding to treat a case of suspected feline tuberculosis is always contentious. Before undertaking treatment it is important to address a number of points:

- Consider the potential zoonotic risk. All members of the affected cat's household must be involved in any decision making. Particular consideration should be given to those individuals most susceptible to the infection, e.g. household members with HIV infection, or those undergoing chemotherapy or organ transplantation. We strongly advise against treatment where such individuals may be exposed to an infected cat. We also advise against treatment if the affected cat has generalised disease, respiratory tract involvement, or extensive draining cutaneous lesions, since any of these findings may increase the risk of transmission. In addition, uncomplicated cutaneous disease appears to carry the most favourable prognosis.
- Where the cat is a suitable candidate, it should be emphasised that treatment is long-term and difficult to maintain given patient non-compliance, the inherent toxicity of some of the drugs and the financial costs involved. In some cases the drugs may at best suppress the disease and indefinite treatment may be required.
- Tailoring treatment is difficult as sensitivity testing does not always correlate with in vivo results.
- Surgical excision of small cutaneous lesions may be considered, but is successful in only a few cases. Debulking larger lesions risks wound dehiscence and local recurrence of infection.

Pending a definitive diagnosis, interim therapy with enrofloxacin (or another fluoroquinolone) is recommended; but only in cases with localised lesions. If additional signs of regional spread or systemic involvement are present (e.g. radiographic changes or

hypercalcaemia), it is strongly recommended that double or triple therapy be initiated immediately (Table 1). This not only gives the best chance of clinical resolution, but also decreases the potential for the mycobacteria to develop resistance to the fluoroquinolone. This is an important consideration since generating drug resistance will be detrimental not only to the individual cat, but may also endanger human patients.

Before deciding on continued treatment it is highly desirable to know exactly which form of mycobacteria is responsible. This is because it is strongly inadvisable to continue treating a cat with *M. tuberculosis* or disseminated *M. bovis* (and in the UK the law may shortly demand the euthanasia of any cat confirmed to have *M. bovis*). Unfortunately, in many cases it is not possible to culture organisms from tissue samples that have been seen to have positive ZN staining. In addition, molecular PCR techniques have still to be perfected. Because of this it is essential to counsel owners very carefully, making them aware of all of the potential risks and complications.

Treatment of choice:

Ideally, anti-tuberculosis treatment should consist of an initial and a continuation phase. The initial phase usually requires at least three drugs and lasts for two months, while the continuation phase requires two drugs and lasts for perhaps a further four months, depending on the type and extent of the disease. In those cats where triple therapy is not feasible, treatment should still involve at least two drugs and should be given for a minimum of six to nine months.

Traditionally, the rifampicin-isoniazid-ethambutol combination has been considered the most effective regime for the treatment of tuberculosis in animals. However, some newer and less toxic drugs are worth appraisal. The fluoroquinolones, e.g. enrofloxacin and marbofloxacin, have potential in the treatment of feline tuberculosis, as well as opportunistic mycobacteriosis. However, they do not appear to be effective against MAC infection. Clarithromycin is a modern macrolide which is used in the treatment of human tuberculosis; while there is little published information on its use in cats, it appears to be effective when given in combination with rifampicin and/or a fluoroquinolone. To date, the only side effect seen with clarithromycin treatment has been pinnal or more generalised erythema which resolves on discontinuation of the drug. A useful once daily alternative to clarithromycin is azithromycin. From clinical experience gained over the past 10 years we recommend treatment consisting of an initial phase of rifampicin-fluoroquinolone-clarithromycin/azithromycin, followed by a continuation phase of rifampicin and either fluoroquinolone or clarithromycin/azithromycin (Table 1).

In cases where resistance develops, the rifampicin-isoniazid-ethambutol combination may be considered. If necessary, ethambutol can be substituted with dihydrostreptomycin or pyrazinamide. However, where *M. bovis* has been confirmed, pyrazinamide is not recommended due to the organism's natural resistance. Rifampicin and isoniazid are more effective and less toxic than ethambutol and dihydrostreptomycin and consequently are more appropriate choices if only two drugs are required.

#### Prognosis:

The prognosis depends on the type of mycobacteria involved, and the extent and severity of the infection. While many cases, especially those caused by *M. microti* infection, have responded very favourably to treatment, and have achieved apparent cure or long-term remission, the prognosis should always be stated as guarded.

## FELINE LEPROSY

#### CLINICAL BACKGROUND:

##### Epidemiology and aetiopathogenesis:

Infection with *M. lepraemurium* is largely assumed as the organism cannot be cultured using standard techniques. However, recent reports from Australia show that feline leprosy can take one of two different forms and that while disease in younger cats does appear to be caused by *M. lepraemurium*; the disease seen in older cats appears to be caused by a novel, but as yet undefined, mycobacterial species. Infection is believed to be gained by the introduction of the organisms through bite wounds from rodents. However, this is not proven and it is also possible that infection is gained via soil contamination of cutaneous wounds. As yet, there is no known zoonotic potential for this disease.

##### Predisposition:

There is no breed or gender predisposition but adult cats are more often affected. The prevalence of feline leprosy is higher in areas with a temperate maritime climate, e.g. Australia, New Zealand, Europe (UK, the Channel Islands, the Netherlands), western Canada and western parts of the USA (California, Oregon).

##### Clinical signs:

Feline leprosy is primarily a cutaneous syndrome. Single or multiple nodules, which may be haired, alopecic or ulcerated, may be seen on the head, limbs and occasionally the trunk. They are non-painful and freely mobile. Regional lymphadenopathy may be present but systemic disease is rare. In Australia this disease appears to have two different forms: one

type affecting young cats, which initially develop localised nodular, often ulcerated, lesions on the limbs, which progress rapidly, while the other type affects older cats, which develop more generalized skin involvement with no ulceration and a slower clinical progression.

#### DIAGNOSTIC TECHNIQUES:

Cytology and histopathology (with the use of special stains) are the major methods of diagnosis. In young cats there are typically few acid fast organisms present. However, in older cats the lesions often contain large numbers of acid fast organisms, which can be clearly seen within macrophages. Culture is usually unrewarding, but should be performed in all suspect cases as the clinical signs and histopathology of feline leprosy can mimic those of feline tuberculosis. Molecular PCR techniques are currently being investigated and do show promise. The diagnostic approach discussed previously for tuberculosis should be followed.

#### MANAGEMENT:

Interim management: A fluoroquinolone (Table 1.) should be used pending diagnosis.

Treatment of choice: Surgical removal of small nodules is recommended. Clofazamine (Table 1.) has been used in a limited number of cases where surgical removal was difficult. Dapsone is considered too toxic for use in cats.

#### Prognosis:

The prognosis is good and spontaneous resolution may occur.

## OPPORTUNISTIC MYCOBACTERIAL DISEASE

#### CLINICAL BACKGROUND:

##### Epidemiology and aetiopathogenesis:

This syndrome is caused by saprophytic, usually non-pathogenic, organisms which are found in soil, water and decaying vegetation. The "fast growing" representatives of this mycobacterial group are most commonly implicated in feline skin disease. However, as our ability to recognise the implications of "bite site" lesions improves, along with our access to the expertise of the specialist laboratories, slow growing variants are being recognised more frequently, as they are in human medicine.

The following organisms have been implicated in causing this syndrome; *M. chelonae-abscessus*, *M. fortuitum* / *peregrinum* group, *M. smegmatis* and *M. phlei*. Other opportunist mycobacteria that have been found causing disease in cats include *M. genavense*, *M. simiae*, *M. thermoresistibile*, *M. xenopi* and *M. terrae* complex. All of these organisms can cause disease through contamination of cutaneous wounds and are particularly pathogenic if inoculated into adipose tissue. Entry through the gastrointestinal or respiratory tracts is rare.

##### Predisposition:

In general, cats appear to be predisposed to infection with this group of mycobacteria. Adult cats with a hunting or fighting lifestyle are more likely to be affected. Disease caused

by these organisms is rarely reported in the UK and appears more common in tropical and subtropical areas of the world. However, difficulties associated with diagnosis may influence its true prevalence. Unlike the situation in humans, immunosuppression has only been found in a small number of the affected cats.

Clinical signs:

Many of the different species of opportunistic mycobacteria produce similar clinical syndromes. The most common of which is typified by panniculitis, where multiple, punctate draining tracts occur with a "salt and pepper shaker" appearance. These are associated with subcutaneous nodules and coalescence produces large areas of ulcerated, non-healing tissue. Affected areas can be extremely painful. The inguinal fat pads, flanks and the tail base are affected most frequently. However, any area may be affected if it is prone to injury (and has sufficient subcutaneous fat). The lesions may be exacerbated by surgery and dehiscence associated with satellite lesions is common. Although systemic spread is rare, fever, anorexia and reluctance to move may be seen. Primary pulmonary infection with *M. fortuitum* and disseminated infection with *M. smegmatis* have been reported and may have arisen from non-cutaneous routes of entry.

DIAGNOSTIC TECHNIQUES:

Histopathology:

Pyogranulomatous panniculitis is seen and should automatically warrant a search for mycobacteria. These organisms are difficult to identify in histopathological sections even when acid fast stained, but the use of modified Fite's or rapid ZN methods will increase the sensitivity of detection.

Culture:

Culture from a biopsy specimen is the diagnostic test of choice. The organisms are usually relatively easy to grow on Lowenstein Jensen media, but molecular PCR techniques are also currently being investigated.

MANAGEMENT:

Interim management: A fluoroquinolone is the drug of choice while waiting for culture.

Treatment of choice:

This is controversial and evaluation of the individual case is required. Ideally, antimicrobial therapy should be determined by culture and sensitivity where possible. This is because different species of opportunist Mycobacteria have differing sensitivity patterns. *M. fortuitum* and *M. smegmatis* are sensitive to fluoquinolones, while *M. chelonae* is sensitive to clarithromycin. It is possible that double or triple therapy with a combination of fluoquinolone, clarithromycin or azithromycin and/or rifampicin should be considered as for the tuberculosis syndromes (Table 1.). Antibiotic therapy should be continued for protracted periods of time, ie. six to twelve weeks. Surgical intervention should be radical and planned with the same precision as removing a locally invasive neoplasm. Antibiotic therapy in combination with surgery has been recommended.

Prognosis:

Prognosis is poor to guarded. The prognosis deteriorates further when there have been previous unsuccessful attempts at surgery.

References and further reading:

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Malik, R., Hunt, G.B., Goldsmid, S.E., Martin, P., Wigney, D.I. & Love, D.N. (1994) Diagnosis and treatment of pyogranulomatous panniculitis due to *Mycobacterium smegmatis* in cats. *J Sm An Pract.* 35, 524-530

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Table 1. Potentially useful drugs for the treatment of feline mycobacterial disease.

Uses	Drug (Trade name)	Dose (mg/kg)	Interval (hours)	Toxicity
1 <sup>st</sup> line tx for TB & opportunistic myco. infections <sup>a</sup>	Enrofloxacin	5 per os	12-24	Retinal degeneration.
	Marbofloxacin	2 per os	12-24	Retinal degeneration.
1 <sup>st</sup> line tx for TB	Rifampicin <sup>b</sup>	10-20 per os	12-24	Hepatotoxicity, induction of liver enzymes, discoloration of body fluids.
1 <sup>st</sup> line tx for TB	Clarithromycin <sup>bc</sup>	5-10 per os	12-24	Pinnal or generalised erythema. G-I signs?
	Azithromycin	7-15 per os	24	
2 <sup>nd</sup> line tx for TB	Isoniazid <sup>b</sup> (Sold in combination with rifampicin +/- pyrazinamide)	10-20 per os	24	Hepatotoxicity, peripheral neuritis.
2 <sup>nd</sup> line tx for TB	Dihydro-streptomycin <sup>b</sup>	15 im	24	Ototoxicity.
2 <sup>nd</sup> line tx for TB <sup>d</sup>	Pyrazinamide <sup>b</sup>	15-40 per os	24	Hepatotoxicity.
Second line tx for TB	Ethambutol <sup>b</sup>	15 per os	24	Optic neuritis.
Tx for leprosy	Clofazamine <sup>b</sup>	8 per os	24	Hepatotoxicity.
2 <sup>nd</sup> line tx for opportunistic myco. infections	Doxycycline	5-10 per os	12	G-I signs

tx - treatment, TB - tuberculosis, im - intramuscularly

<sup>a</sup> Not effective against MAC infection.

<sup>b</sup> These drugs are not licensed for use in cats

<sup>c</sup> Doses are extrapolated from human recommendations since there is little information published relating to the use of clarithromycin in the cat.

<sup>d</sup> Not effective against *M. bovis* infection

Second line treatments for tuberculosis should be reserved for resistant infections only.

Drugs licensed for human use can be obtained by veterinary prescription from larger chemists.

Useful addresses in the UK:

Dr. Danièle Gunn-Moore, Senior Lecturer in Feline Medicine,  
University of Edinburgh Small Animal Hospital,  
Easterbush Veterinary Centre,  
Roslin,  
Midlothian,  
Scotland, EH25 9RG  
Tel: 0131 650 7650  
Email: [Danielle.Gunn-Moore@ed.ac.uk](mailto:Danielle.Gunn-Moore@ed.ac.uk)

Rachel Dean, Feline Fellow,  
Feline Centre, Department of Clinical Veterinary Studies,  
University of Bristol Veterinary School,  
Langford House,  
Langford,  
North Somerset, BS18 7DU (check post code)  
Tel: 0117 920 9577  
Email: [R.Dean@bristol.ac.uk](mailto:R.Dean@bristol.ac.uk)

Please contact either of these people to discuss the case in more detail. We are currently trying to collate all of the cases in cats in the UK so that we can gain a better understanding of the presentation, causes, and treatment responses of the condition.

Mr. Keith Jahans,  
Veterinary Laboratories Agency (DEFRA),  
Weybridge, UK.  
Telephone number: 01932 357280  
Email: [kjahans@vla.defra.gsi.gov.uk](mailto:kjahans@vla.defra.gsi.gov.uk)

All potential cases should be reported to the VLA. The VLA is currently willing to undertake mycobacterial culture free of charge in cases where the history, clinical signs and/or histopathological findings are suggestive of mycobacterial infection. Please contact Mr. Jahans prior to sending samples to ensure appropriate samples are submitted, and enclose case details when submitting samples.

Feline Advisory Bureau,  
Taeselbury,  
High Street,  
Tisbury,  
Wiltshire, SP3 6LD.  
Tel: 0870 742 2278 Fax: 01747 871873  
Email: [information@fabcats.org](mailto:information@fabcats.org) Web site: [www.fabcats.org](http://www.fabcats.org)

Contact the FAB for more information on tuberculosis in cats, and information on what to do if you have a suspected case.