Bovine Tuberculosis

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INTRODUCTION

Tuberculosis in animals occurs worldwide and is primarily known from cases in cattle and other bovids for which the disease is generally referred to as bovine tuberculosis. The major causative agent of bovine tuberculosis is *Mycobacterium bovis*, a member of the *M. tuberculosis* complex and closely related to the cause of human tuberculosis. Bovine tuberculosis is a chronic, debilitating disease, characterized by the formation of typical granulomatous lesions, yet slowly progressive both in the individual as well as on population level. It is of high economic relevance within the context of livestock farming as it directly affects animal productivity and also influences international trade of animal products. Where infected cattle have direct contact or share the environment with wildlife, *M. bovis* can be transmitted to a wide range of free-ranging wildlife species. Some of these species can become maintenance hosts of the disease with severe, complex implications for the ecosystem and negative effects on the control of bovine tuberculosis at the wildlife/livestock interface. Moreover, animal tuberculosis bears a zoonotic potential and is therefore of public health concern, especially in countries lacking effective control measures for bovine tuberculosis in cattle.

Video link: [http://www.youtube.com/watch?v=OPBPZkXXlj8&feature=youtu.be](http://www.youtube.com/watch?v=OPBPZkXXlj8&feature=youtu.be)

*Mycobacterium bovis*, causative agent of bovine tuberculosis, is an acid-fast organism belonging to the group of *M. tuberculosis* complex bacteria, along with *M. tuberculosis*, *M. africanum*, *M. canetti*, *M. bovis BCG*, *M. microti*, *M. caprae*, *M. pinnipedii* and the *oryx bacillus*. The *M. tuberculosis* complex is generally considered a family of “ecotypes” of very closely related Mycobacteria, with each ecotype being adapted to cause tuberculosis disease in a specific host species or group, even though inter-species transmission can occur. In contrast to the earlier hypothesis that tuberculosis has evolved from an originally animal disease to a human disease, new findings indicate that in fact tuberculosis first emerged in humans and
was subsequently transmitted to animals. Recent studies suggest that the common ancestor of the *M. tuberculosis* complex emerged from its progenitor perhaps 40,000 years ago in East Africa. Some 10,000 to 20,000 years later, two independent clades evolved, one resulting in *M. tuberculosis* lineages in humans, while the other spread from humans to animals, resulting in the diversification of its host spectrum and formation of other *M. tuberculosis* complex member species, including *M. bovis*. This adaptation to animal hosts probably coincided with the domestication of livestock approximately 13,000 years ago.

**EPIDEMIOLOGY**

Bovine tuberculosis is widespread in cattle throughout the world. According to information on the worldwide animal health information database of the OIE (WAHID Interface), 128 out of 155 countries reported the presence of *M. bovis* infection and/or clinical disease in their cattle population during the period between 2005 and 2008. In wildlife, cases of *M. bovis* infection have been reported in more than 40 free-ranging wild animal species to date.

While bovine tuberculosis has been successfully controlled in most of the developed world through the implementation of test-and slaughter schemes, abattoir monitoring and pasteurisation of milk, the presence and extent of bovine tuberculosis in the developing world has been poorly investigated in the past. A number of recent studies have revealed new data confirming the presence of *M. bovis* in cattle and moreover providing insights into the specific risk factors associated with tuberculosis in cattle in different countries and regions. In Africa, high prevalence rates of bovine tuberculosis (up to 50% at herd level) were reported in areas of Zambia where cattle and Kafue lechwe shared grazing and water as well as in areas where the traditional management of livestock in transhumant herds (herds which are moved to floodplains for grazing during the dry season) prevailed. Under these often nomadic conditions, the risk of exposure to *M. bovis* was increased significantly by creating multiple herd contacts and increasing the total herd size. The latter has also been suggested as a driver of the disease prevalence in Ethiopia and Ecuador. On the other hand, in countries with a rapidly increasing livestock production and intensification of production systems such as Iran, the propagation and insufficient detection of circulating *M. bovis* strains may be the most important contributor to increasing economic losses from bovine tuberculosis, rather than the importation of infected cattle, as previously suggested.

Transmission of the disease occurs most commonly during direct, close contact between uninfected and infected animals, especially those in advanced stages of the disease as shedding increases with the development of gross lesions. In cattle, other bovids and some social wildlife species bovine tuberculosis affects primarily the lungs and hence transmission occurs mainly via contaminated aerosols. Under extensive farming conditions the disease spreads as a result of cattle congregating in communal kraals (pens) at night or at watering points under dusty conditions. Ingestion of infected milk is a significant route of infection from infected dams to calves and a risk factor in humans consuming unpasteurised milk from
infected cows. In carnivores and scavenging wildlife species infection per os is an important route of infection, whereby secondary involvement of the lungs as a result of haematogenous spread of the organisms allows excretion of high doses of infectious organisms via the respiratory route. Excretion of *M. bovis* from discharging skin wounds, draining lymph nodes and in urine is of importance in the epidemiology of *M. bovis* infection in meerkat (suricate), greater kudu and the European badger, respectively. Congenital infection is possible and can affected 1 - 5 per cent of calves in cattle herds with a high prevalence of tuberculosis.

The environment generally plays a minor role in the epidemiology of bovine tuberculosis due to the fact that *Mycobacterium bovis* is an intracellular pathogen and cannot survive outside the host for prolonged periods of time. Under environmental conditions of high temperature, low moisture content and UV radiation, the survival time is measured in days with a maximum of 2 weeks. Despite this, circumstantial evidence has indicated the spillover of *M. bovis* from cattle to greater kudu and from kudu to buffalo, which is less likely to occur via direct contact but is probably facilitated by contaminated environmental sources such as soil, vegetation or water.

African buffalo, which had originally contracted the disease by close contact with infected cattle, have introduced bovine tuberculosis into several wildlife populations. They can serve as direct source of infection to predators (alimentary route) and other herbivores sharing the same habitat (e.g. by aerosol during close contact). Scavengers and omnivores in southern Africa and elsewhere can contract *M. bovis* from contaminated carcasses or other environmental sources in a high prevalence area. *Mycobacterium bovis* infection has also been diagnosed in a wide variety of free-living wildlife species in some game parks in South Africa including African buffalo (*Syncerus caffer*), greater kudu (*Tragelaphus strepsiceros*), lion (*Panthera leo*), cheetah (*Acinonyx jubulatus*), baboon (*Papio ursinus*), grey duiker (*Sylvicapa Grimmia*), spotted hyena (*Crocuta crocuta*), leopard (*Panthera pardus*), honey badger (*Mellivora capensis*), warthog (*Phacochoerus aethiopicus*), bushpig (*Potamochoerus porcus*), impala (*Aepyceros melampus*), bushbuck (*Tragelaphus sylvaticus*), eland (*Taurotragus oryx*), spotted genet (*Genetta tigrina*). It has been isolated from wildebeest (*Conochaetes taurinus*), topi (*Damaliscus lunatus*) and lesser kudu (*Tragelaphus imberbis*) in Tanzania and from lechwe (*Kobus leche*) in Zambia and wild boar in Spain. With the exception of buffalo, kudu, lechwe and wild boar these species are considered to be spill-over species of *M. bovis*.

**PATHOGENESIS**

Although many aspects of the pathogenesis of bovine tuberculosis are still not fully explained, the outcome of *M. bovis* infection in an animal is largely dependent on the effects of cell-mediated immunity, associated with protective immune responses, and hypersensitivity reactions. This results in a spectrum of possible outcomes ranging from elimination, self-limiting infection, localized lesions or a life-threatening systemic disease.
The course of events, which follow initial infection in a previously uninfected animal depends to some extent on the route of infection which, in cattle and most wildlife species, is either via the respiratory or gastrointestinal tract. Ingested or inhaled organisms may enter an animal's tissues through the mucous membrane of the pharynx in which they multiply and induce the development of a primary, microscopic lesion. It is possible that in cases of a low infectious dose and an immune-competent host the bacilli in this first focus of infection are killed and the infection sterilized. Otherwise the organisms are able to multiply rapidly and spread in macrophages via lymphatic vessels to region lymph nodes in which they again multiply and cause lesion development. These two lesions (in the mucous membrane and the lymph node) are known as a complete primary complex. In many instances, the mucous membrane lesion heals and only that in the lymph node remains. This is known as an incomplete primary complex. A similar course of events generally occurs if ingested mycobacteria enter an animal lower down the gastrointestinal tract. A primary complex formed in the lungs as a result of inhaled organisms in aerosols consists of the lesions in the lung parenchyma (which usually does not heal) and that in the mediastinal and/or bronchial lymph nodes.

From a primary complex (complete or incomplete) in an unsensitized host the infection may spread locally (e.g. in the lungs) or via lymph and/or blood to involve other organs and tissues, a process known as post-primary dissemination. A bacteraemia may develop as soon as 20 days after initial infection has taken place. With the exception of those secondary (metastatic) lesions in certain "vulnerable sites", such as the lungs, bone, kidneys and meninges, many undergo resolution after cell mediated immunity (CMI) has been elicited. The latter may reduce, but at this stage no longer eliminate, the bacterial population in the "vulnerable sites".

Development of granulomatous lesions

Intracellular mycobacteria are able to survive by the production of sulfatides that prevent phagosome fusion, and heat shock proteins that protect against the respiratory burst reaction elicited by activated phagocytes. After 10-14 days of infection cell mediated immunity responses develop and host macrophages acquire an increased capacity to kill the intracellular bacilli. In the centre of these cell-mediated immune responses are lymphocytes, which release a range of cytokines, among which interferon gamma plays a leading role, that attract, immobilize and activate additional blood-borne mononuclear cells at the site where mycobacteria or their products are present. All these factors contribute to cell death and tissue destruction (caseous necrosis). Mycobacterial glycolipid (cord factor) inhibits chemotaxis of leukocytes and is leukocidal, which further contributes to cell destruction. In some instances, liquefaction and cavity formation occur due to enzymatic action on proteins and lipids. Low numbers of bacteria mainly cause proliferative lesions, whereas high numbers result in an exudative response. Caseous lesions tend to inhibit bacterial multiplication, whereas bacteria are able to multiply in liquefied lesions resulting in extension of the areas of caseous necrosis. In addition, these cavities may rupture enhancing the spread of the bacilli, which is of special importance in the lungs as it facilitates aerosol transmission. The transition of cell mediated immune response is marked by decrease in cytokine
release, paralleled by an increase in antibody production which is generally followed by increased lesion formation (high antibody levels are indicative of extensive pathological changes).

Granuloma or tubercle formation is an attempt of the host to localize the infection and to allow inflammatory and immune mechanisms to destroy the bacilli. Some of the lesions may appear to regress and become encapsulated by well-organized connective tissue. Such lesions may contain viable bacilli, which can be reactivated when the host's resistance decreases.

The microscopic appearance of a typical tubercle is characterized by a central area of caseous necrosis encircled by a zone of epithelioid cells, multinucleate giant cells, lymphocytes, plasma cells and small amounts of granulocytes, around which there is a connective tissue capsule. Mineralization may be present in the necrotic centres.

**DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS**

**Clinical signs and pathology**

Bovine tuberculosis is generally a chronic debilitating disease but can occasionally be more acute and rapidly progressive. Early infections are often asymptomatic. In cattle with extensive miliary tubercular lesions a progressive loss of condition in the absence of other clinical signs may be the only observable sign. The condition of the hair-coat is variable but is usually dull and rough. Anorexia, stunted growth, enlarged lymph nodes and lethargy are observed but the temperature may be non-specific, fluctuating in most cases. Superficial lymph nodes are enlarged and palpable.

Tubercular lymphadenitis at the angle of the jaw of a kudu bull

Tubercular lymphadenitis of the head of a kudu
In the majority of cases (90-95%) the route of transmission is by inhalation resulting in the respiratory tract being affected and often in pulmonary tuberculosis. A chronic cough due to bronchopneumonia indicates pulmonary involvement, but is never loud but moist and worst in the morning. Dyspnoea is more common in advanced or terminal stages. A low grade fever is usually observed, as well as weakness and inappetence.

When the GIT is involved, intermittent diarrhoea and constipation may be observed. Reproductive disorders include uterine tuberculosis in advanced cases and may result in infertility or recurrent abortion following conception. Clinical signs may further include mucopurulent discharges, vulvar discharges and a placentitis similar to *Brucella abortus* infection. Irregular oestrous and abscesses in testes may be observed.

Lesions of bovine tuberculosis may also be found in the vertebrae, ribs and flat bones of the pelvis, especially in young animals. In advanced cases, lesions in the central nervous system may lead to loss of vision, incoordination in gait, partial paralysis, and circling.

Under stressful conditions, such as in malnutrition and post-calving, the clinical signs of BTB are generally more pronounced.

In cattle, lesions usually appear in the pharynx, thoracic cavity and the lungs. In the affected respiratory tract the granulomatous lesions, so-called tubercles, are found in the retropharyngeal, mediastinal and bronchial lymph nodes. Lesions are also found in the lungs to complete the primary complex. Lung lesions may appear microscopically or macroscopically as encapsulated yellowish foci of caseous necrosis. The lung lesions may spread to the visceral and parietal pleura, where they occur in masses and sometimes fuse together.
Severe tuberculous pneumonia

A section through tubercular lesions in the mesenteric lymph nodes

Tuberculous pleuritis. Areas of the lung have adhered to the parietal pleural surface

Tubercular lesions in the ovary and fallopian tube of a cow
Gastro-intestinal tract (GIT) lesions are the second most common form of bovine tuberculosis. The lesions appear as nodules and/or ulcers in the mucosa of the upper alimentary tract, abomasum, small intestine and large intestine. Ulcers develop first in Peyer's patches in the small intestines.

Generalized or miliary tuberculosis manifests as small but numerous lesions in a number of organs of the body, following dissemination of the bacilli by haematogenous spread and subsequent localization in various organs of the body, followed by development of tubercles in the affected organs.

Various forms of tuberculosis can be manifested in the udder of bovines. This ranges from a scenario where most of the glandular lobules are replaced by granulomatous tissue to a case where there are clusters of small lobules that have calcified. The resultant mastitis is therefore variable, ranging from caseous tuberculous mastitis to tuberculous galactophoritis.
Laboratory confirmation

Indirect detection methods comprise those techniques which exploit the immune response mounted by infected animals. A distinction is made between the early, cell mediated immune response and the antibody based, or humoral, immune response which is elicited during the advanced stages of bovine tuberculosis.

The first and best-known immunological test for tuberculosis diagnosis is the tuberculin skin test. The same principle is used for testing in both, animals and humans and although imperfect, the tuberculin test has not yet been replaced by any other more accurate or satisfactory method. Some of the main deficiencies of the test are its inability to differentiate between distinct species of the *M. tuberculosis* complex, failure to distinguish between latent stages of infection and disease and failure to distinguish vaccinated and infected individuals. In addition, anergy (the disease stage when the immune response has switched from cellular to humoral immunity and consequently infected animals are no longer reactive to the skin test or interferon gamma assay), exposure to environmental mycobacteria and operator errors can lead to false results. Two recent studies also suggested that the cut-off generally used for positive test interpretation (>4mm) may not be applicable for at least some countries in sub-Saharan Africa and that a cut-off >2mm could be more appropriate in some settings. The more recently developed Bovigam® test for cattle detects the production of interferon gamma in (in vitro) stimulated blood samples. Applied in both standard commercial and customised test formats this assay has contributed significantly to the improved detection of early *M. bovis* infection in cattle as well as an increasing number of wildlife species (e.g. non-human primates, cervids). Recent improvements of the test include the use of two *M. tuberculosis* complex specific antigens, ESAT-6 and CFP-10, which has resulted in increased test
specificity. The tuberculin skin test and the interferon gamma test are both based on the detection of the early cell-mediated immune response in tuberculosis infection. However, at late disease stages, the cell-mediated immune response can wane as opposed to a generally increasing humoral immune response and the tuberculin skin test or Bovigam® tests can therefore give false negative results in these so-called anergic animals. This is of importance for the diagnosis of bovine tuberculosis in settings where no or little disease control measures are applied and where the percentage of late stage diseased animals is believed to be high. Therefore, in developing countries, serological tests, which are based on the detection of the humoral immune response, may be of particular use. Unfortunately, to date, no satisfactory serological test is available. Some of the problems related to the development of serological tests for tuberculosis diagnosis include the observed highly variable antibody responses between individuals to mycobacterial antigens and antigenic variation between mycobacterial strains. However, a recently developed lateral flow test that is based on the detection of more than one antigen has shown promising results for tuberculosis diagnosis in certain animal species (e.g. in elephant), although it may not be suitable for others, such as buffaloes. Another recently developed serological test for animals is based on antibody detection using fluorescence polarisation but has shown variable effectiveness in different settings.

Direct methods for tuberculosis diagnosis are based on the isolation or detection of the bacterium in sputum samples or biopsies (mostly in humans) or at post-mortem examination, from tuberculous organ lesions (generally in animals). The presence of Mycobacteria in a given sample can be assessed by Ziehl-Neelsen staining followed by light microscopy or auramine O staining and fluorescence microscopy. Recent work with *M. tuberculosis* suggests that the auramine O staining technique may be more sensitive and specific than Ziehl-Neelsen staining. However, microscopic detection of Mycobacteria shows a generally low sensitivity (from 50-70%) for human sputum samples. A much higher sensitivity can be achieved by prior culture of the bacteria. Culture is still regarded as the gold standard for tuberculosis diagnosis despite certain deficiencies. For example, the yield of culturable (quantities) of bacteria from blood, urine, lavage and cerebrospinal fluid is very low. Bacterial culture is also time consuming and does by itself not allow the differentiation between distinct mycobacterial species However, in many cases, culture is a prerequisite for further testing and characterization of Mycobacteria.
Ziehl-Neelsen stained film with large numbers of acid-fast mycobacterial organisms appearing as small pink-staining rods

Video link: http://www.youtube.com/watch?v=NnZsh7i6iB4&feature=youtu.be

**Tuberculin skin test**

PCR (polymerase chain reaction)-based techniques are indispensable for the direct detection and accurate differentiation of mycobacterial species and molecular epidemiological investigations of tuberculosis transmission (for detailed technical information on this subject the reader is referred to the more specific literature). Although biochemical techniques also allow the differentiation between distinct mycobacterial species, these methods are very laborious, time consuming and
appear to be erroneous. PCR can be used for any sample material in theory, but has some problems of its own e.g., certain samples may contain PCR inhibitors, which could lead to false negative results. Conversely, the generation of a vast number of DNA amplicons can quickly give rise to false positive results. Moreover, the technique requires a relatively sophisticated laboratory and well trained technicians.

**Differential diagnosis**

Conditions in cattle that may cause clinical signs similar to bovine tuberculosis are:

- Traumatic reticulitis.
- Chronic pasteurellosis.
- Foreign body or aspiration pneumonia.
- Pharyngeal obstruction.
- Actinobacillosis.
- *Arcanobacterium pyogenes* infection.
- *Corynebacterium pseudotuberculosis* infection.
- Mastitis caused by other organisms.

At necropsy or inspection of carcasses at abattoirs, the granulomatous lesions of tuberculosis may be confused with:

- those caused by infectious agents such as fungi, staphylococci, *Arcanobacterium* and *Actinobacillus* spp.,
- foreign body pneumonia, and
- "pearl disease" with pleural or peritoneal mesothelioma.

**CONTROL / PREVENTION**

In large parts of the developed world, policies regulating the control of bovine tuberculosis are aimed at complete eradication of the disease from its livestock populations as part of an integrated approach to food safety. These policies follow an expensive test-and-slaughter strategy for the control of bovine tuberculosis and significant successes have been achieved in many countries. On the other hand, the benefit and sustainability of such costly programmes have been increasingly questioned in the light of the
rising economic burden and social impacts on and reduced acceptance by farmers. However, in general, with the exception of a few countries with a wildlife reservoir of *M. bovis* (see further below), the prevalence of bovine tuberculosis has reached very low levels, in most developed countries.

The situation is profoundly different in developing countries, which are unable to apply expensive test-and-slaughter schemes for the control of animal tuberculosis. Although in parts of the Latin American and Caribbean countries there has been significant progress in bovine tuberculosis control and infection rates under 1% have been reported for 30% of the region’s cattle, 70% of cattle are kept in areas where rates of infection are higher and where herd prevalences of up to 56% have been reported. On the African continent very few countries apply control measures (http://www.oie.int/wahis).

Risk factors contributing to difficulties in controlling bovine tuberculosis in cattle across continents can have their origin at farm-level, e.g. cattle breed, age, behaviour, and nutrition of animals. However, host independent factors are considered more important in most cases and include, amongst others, production types and management practices as under circumstances of high population density and production stress, progression of disease is enhanced. Cattle movement, existence of a wildlife reservoir and possibly strain related differences are of additional significance. Tuberculosis in wildlife, in particular, can pose serious difficulties for bovine tuberculosis control and eradication. Particularly noteworthy is the case of the British Isles, where the Eurasian badger represents an important and well documented disease reservoir. In several industrialized countries that have adopted animal tuberculosis control programs and in which wildlife has been involved, control programmes were designed to exclusively benefit the livestock sector with less importance given to wildlife conservation or protection. This is mostly due to the fact that many of the wildlife maintenance host species, with the exception of badgers in the United Kingdom and Ireland, score a low priority on their national wildlife conservation listings and enjoy, at best, the status of valued, sought-after hunting trophies. In some cases these reservoir species are classified as alien or feral with well-documented examples being the brushtailed possums in New Zealand and feral water buffaloes in Australia leading to the implementation of radical disease eradication campaigns by culling programmes.

Bovine tuberculosis can be considered a re-emerging disease due to several factors, e.g. the wide host spectrum of *M. bovis*, the presence of wildlife reservoirs, the insidious nature of the disease allowing widespread distribution of *M. bovis* before clinical or post mortem findings become apparent. Preventive strategies and those aimed at early recognition of the infection are therefore considered more effective measures than trace-back and culling operations. Vaccination is a low cost and effective strategy for the prevention and reduction of infectious diseases in general worldwide and is likely to play an important role in the control of bovine tuberculosis in developing countries and those with a wildlife reservoir. Although to date no commercial vaccine is available for animals, considerable progress has been made in studying the protective efficacy of BCG (Bacillus Calmette Guerin) in reservoir hosts such as cattle, deer, badgers and brushtail possums. In African buffaloes, however, BCG did not induce significant levels of protection to challenge with *M. bovis*. For new vaccine development against both, bovine and human tuberculosis,
prime-boost strategies involving combinations of BCG with a protein or DNA vaccine, to improve on BCG vaccination alone, have produced encouraging results recently.

MARKETING AND TRADE / SOCIO-ECONOMICS

In developed countries, the driving forces for the control and eradication of bovine tuberculosis from the national domestic herd are indisputably of economic and socio-political nature, based mainly on the negative economic impact of the disease. Financial losses are encountered foremost through the costs for the control of the disease (testing and compensation expenses, losses from animal movement and sale restrictions) as well as decreased milk and meat production. In contrast, bovine tuberculosis is endemic in numerous developing countries and can have devastating impacts on the livelihood of millions of the world’s most vulnerable communities as the disease compromises their sustainable food supply, income, social status and potentially also their health in the mainly rural livestock producing areas (http://www.who.int/zoonoses/Report_Sept06.pdf). Although recent studies have provided insights into the significance of zoonotic tuberculosis in developing countries in Africa, the extent to which zoonotic transmission contributes to the burden of human tuberculosis in these areas is still largely unknown.

In South Africa, like other countries in the region, communities facing a higher disease risk from *M. bovis* include those living at the livestock-human interface, consuming mostly unpasteurised milk and dairy products derived from cattle herds with an uncontrolled bovine tuberculosis disease status. Close contact of cattle owners and herd boys with their cattle constitute a further undetermined risk factor. According to recent reports, 85% of cattle and 82% of human populations co-exist in areas where bovine tuberculosis control in cattle is nonexistent or minimally implemented. At the same time they also include those population groups who are suffering from the world’s highest HIV/AIDS infection rates and the associated increased susceptibility to co-infection with *M. tuberculosis*, the main cause of tuberculosis in humans. To make matters worse, the risk groups mentioned are not mutually exclusive but may be identical in many cases. In recent years a growing awareness of neglected zoonoses including bovine tuberculosis has led to initiatives in developing countries, supported by the WHO/FAO/OIE to investigate, calculate and mitigate the unknown risk from these animal diseases on livestock productivity, human health and livelihoods (WHO 2009).

In South Africa and other African countries, *M. bovis* has been transmitted from livestock to wildlife reservoirs in free-ranging ecosystems with potentially far reaching direct and indirect implications on wildlife, livestock and human populations. Perhaps most importantly, *M. bovis* has established itself in the African buffalo (*Syncerus caffer*), a wildlife species of outstanding economic and ecological value, also reflected in its ranking among the ‘Big Five’ wildlife species. Bovine tuberculosis in buffalo poses a threat not only to species conservation efforts and ecotourism but to commercial game farming which has, through the historically embedded prestige associated with keeping indigenous game, created a unique and sustainable niche in African agriculture (www.daff.gov.za). The wildlife industry in e.g. South Africa
today enjoys the status of a specialised sector within agriculture and the land surface presently utilised for
game farming is equal to or has, in some parts of the country, exceeded that of livestock farming. The
potential for spillover of *M. bovis* from buffalo to other wildlife species extends the risk of infection to all
types of wildlife and mixed livestock/wildlife operations.

**FAQ**

1. **Can infected cattle recover from bovine tuberculosis?**

   Like in humans, mere infection with *M. bovis* will not necessarily lead to clinical disease but may
   be eliminated by the host immune system or lie dormant and be activated at a later stage.
   However, once the infection has established itself in the host, tuberculosis is a chronic,
   progressive disease and when the affected individual has developed lesions and clinical signs,
   there is no self-limitation or full recovery without treatment.

2. **How long is the incubation period for bovine tuberculosis?**

   The incubation period in the case of a chronic disease like tuberculosis is highly variable and can
   range from several weeks to months.

3. **Is bovine tuberculosis transmissible to humans?**

   Bovine tuberculosis is transmissible to humans and hence constitutes a zoonotic disease.

4. **Is bovine tuberculosis treatable?**

   There are drugs which are effective against *M. bovis*, comparable to *M. tuberculosis*, whereby the
   practicality of long term treatment of animals in bovine tuberculosis needs to be evaluated
   carefully when the prognosis is assessed.

5. **In the absence of control measures, what is the mortality rate from bovine tuberculosis?**

   The mortality rate largely depends on the prevalence and chronicity of the disease present in a
   population. Under high prevalence conditions a mortality rate of 10% has been estimated.

6. **Are some animals resistant to *M. bovis* or will eventually all cattle in an infected herd contract the infection?**

   Although it is not known beyond doubt, experience has shown that infection rate never reaches
   100%, which is partially due to varying exposure risks in a herd, but has been partially attributed
   to resistance in a small number of animals.
7. Are indigenous cattle breeds less susceptible than exotic breeds?

A difference in susceptibility between breeds has been documented (published information) with cattle of the Holstein-Friesian breed showing a higher susceptibility to bovine tuberculosis compared to other breeds.

REFERENCES


