# Bovine Babesiosis

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INTRODUCTION

Bovine babesiosis or redwater is a tick-borne disease caused by the intra-erythrocytic protozoan parasite *Babesia*. Two species are economically important in tropical and subtropical regions of the world, including southern Africa: *Babesia bovis*, which causes Asiatic redwater, and *Babesia bigemina*, which causes African redwater. *Babesia divergens* causes an economically important disease in the British Isles and northern Europe. The acute disease is characterized by haemolysis and circulatory disorders (in the case of *B. bovis*); death may follow in some instances. European breeds introduced to tropical/subtropical regions are particularly susceptible to Asiatic and African redwater. A clinically inapparent form of the disease is common in young animals, and recovered animals become latent carriers for variable periods. Recovery is followed by a lasting immunity. Cross-immunity between the two organisms is limited. At least two further species, *B. occultans* (transmitted by *Hyalomma marginatum rufipes*) and an unnamed *Babesia* sp. (transmitted by *Hyalomma truncatum*), are known to be present in South Africa. Neither appears to be of economic significance and therefore will not be discussed. The same applies to *B. major* (transmitted by *Haemaphysalis punctata*), which has the same distribution as *B. divergens*.

*Babesia bovis* was probably introduced into southern Africa with the Asian blue tick (*Rhipicephalus (Boophilus) microplus*) during the latter part of the 19th century. *Babesia bigemina* is principally transmitted by the common, indigenous African blue tick (*Rhipicephalus (Boophilus) decoloratus*), as well as by *R. (B.) microplus*. (Fig 1). Other tick vectors may also be involved.

The diseases caused by *B. bovis*, *B. bigemina* and *B. divergens* are clinically very similar but it is important to differentiate between them for a number of reasons. While *B. bovis* is the more virulent of the
two parasites, *B. bigemina* is probably more important in southern African because of its wider distribution.

**Video link:** [http://www.youtube.com/watch?v=7vdE12e_q9g&feature=youtu.be](http://www.youtube.com/watch?v=7vdE12e_q9g&feature=youtu.be)

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**EPIDEMIOLOGY**

**Distribution**

Both *Babesia* species occur in Central and South America, parts of Europe and Asia, Australia and Africa. *Babesia bigemina* has been eradicated from the United States of America. In southern Africa *Babesia bovis* is restricted to areas where *R. (B.) microplus* is prevalent, usually the higher rainfall areas in the eastern parts. Due to its wider vector range, *Babesia bigemina* is much more widespread and is present throughout southern Africa, except for the more arid and some high-lying parts.

**Susceptible hosts / reservoirs**

European, Sanga and Zebu breeds are all susceptible, and all develop latent infections after recovery. European breeds can retain *B. bovis* infections for life and remain infective for ticks for up to two years, while most cattle with a significant Zebu content lose the infection within two years. *Babesia bigemina* infections rarely persist for more than a year, regardless of the host, and infected cattle remain infective for ticks for only four to seven weeks.

It is possible to have *Babesia* spp. and their vectors present in a cattle population without measurable economic losses or clinical disease. This is known as endemic stability, which is defined as the state where the relationship between host, agent, vector and environment is such that clinical disease occurs...
rarely, if at all. An important factor in the establishment of endemic stability is the age of first exposure: Calves have a natural resistance during the first six to nine months of life and rarely show clinical signs, yet develop solid, long-lasting immunity.

**Transmission**

*Rhipicephalus (B.) microplus* is the only known tick vector of *B. bovis* in southern Africa. Transmission is transovarial with engorging adult ticks ingesting the parasites and larval ticks of the next generation transmitting the infection. Ensuing stages are not infected.

Confirmed vectors of *B. bigemina* include *R. (B.) microplus, R. (B.) decoloratus and Rhipicephalus evertsi evertsi* (lesser importance). Transmission by *Rhipicephalus (Boophilus)* spp. is transovarial, with engorging adult ticks becoming infected, but the infection is transmitted to cattle by the nymphal and adult stages of the next generation.

*Babesia bovis* and *B. bigemina* follow similar developmental patterns in adult *Rhipicephalus (Boophilus)* spp. Initial development takes place in epithelial cells of the gut wall where schizogony (multiple fission) occurs with the formation of large merozoites (vermicules, sporokinetes). Successive cycles of schizogony then occur within a variety of cell types and tissues, including the oocytes. Thus, transovarial transmission occurs with further development taking place in the larval stage.

**PATHOGENESIS**

The primary mechanism is intravascular haemolysis (leading to haemoglobinaemia and haemoglobinuria), resulting in anaemia, hypoxia and secondary inflammatory lesions in various organs, especially liver and kidneys. The secondary mechanism is electrolyte imbalances, complement activation, coagulation disorders and release of pharmacologically active substances resulting in vascular malfunction and hypotensive shock.

The main sequelae of the disease are:

- Anaemia due to haemolysis; haemoglobinaemia and haemoglobinuria (Figure 2); icterus.
- Pharmacologically active substances such as kinins and catecholamines lead to increased vascular permeability and dilatation of blood vessels resulting in oedema and hypovolaemic shock.
- Centrilobular liver degeneration and degeneration of kidney tubule epithelium are caused by hypoxia and possibly by immunopathologic reactions.

Damage to kidney tubule epithelium impairs ion exchange, resulting in H+ retention (leading to acidosis).
DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Clinical signs and pathology

In natural infections, incubation periods usually vary from 8 to 15 days. In acute manifestations, fever (>40°C) is usually present for several days before the onset of other clinical signs: inappetence, depression, weakness and reluctance to move. Haemoglobinuria is often present especially in B. bigemina infections (hence the common name "redwater"). Anaemia and icterus are especially obvious in more protracted cases. Diarrhoea is common and pregnant cows may abort. Cerebral babesiosis, which occasionally develops in B. bovis infections, is manifested by hyperaesthesia, nystagmus, circling, head pressing, aggression, convulsions and paralysis; these signs may or may not accompany other signs of acute babesiosis.

Necropsy of uncomplicated ("typical") cases of babesiosis is characterised by light red, watery blood and the mucous membranes and carcase are paler than normal (these changes are due to anaemia). In many cases this pallor may be masked by icterus (Figure 3). The spleen is invariably enlarged and has a pulpy consistency (severe congestion) (Figure 4). The liver is swollen, friable and yellowish-brown in colour (degeneration, bile stasis). The hepatic surface may have an evenly mottled appearance, with lighter coloured periacinar areas (fatty degeneration to necrosis). The gall-bladder is distended with viscous bile which often contains dark brown granules up to 1 mm in diameter (bile inspissation). The intestinal content is usually diminished (anorexia) and yellowish in colour (bile-stained). The kidneys are mildly to moderately swollen and dark reddish-brown in colour (haemoglobinuric nephrosis) (Figure 5) or yellowish-brown (cholaemic nephrosis). The lungs are often oedematous, with foam present in the bronchi and trachea (probably due to agonal left heart failure). The heart itself is usually flabby and pale (degeneration, anaemia) and agonal endocardial and epicardial petechiae and ecchymoses may be present. In cases which survive for longer, mild to moderate transudation into the body cavities (hydrothorax, hydropericardium, ascites) may be observed. The urine is discoloured and may be deep yellow to yellow-brown (bilirubinuria) or a clear port-wine colour (haemoglobinuria). It must be emphasised that the above description of the macroscopic lesions applies not only to typical cases of
babesiosis but to any disease in which significant erythroylysis occurs. It can thus be used as a model for most of the other haemolytic diseases.

![Bovine babesiosis: carcass of calf with pronounced icterus](image1)

![Splenomegaly](image2)

![Kidney moderately swollen and dark reddish-brown in colour (haemoglobinuric nephrosis)](image3)

**Laboratory confirmation**

Thin blood films made from capillary blood are preferred; thick blood films are more sensitive, but species differentiation is more difficult. Blood of the general circulation may contain up to 20 times fewer *B. bovis* than capillary blood. In *B. bigemina* infections, parasitized cells are evenly distributed throughout the blood circulation. *Babesia bovis* parasitaemias are often low (<0.1%), even at the peak of the reaction, while *B. bigemina* parasites are usually more numerous and therefore easy to detect.

*Babesia* spp. develop only in the erythrocytes. Merozoites (Figure 6) penetrate the cell membrane with the aid of the apical complex, and transform into trophozoites that undergo merogony to give rise to two new merozoites. Cells containing more than two parasites are rare. Parasitaemia can exceed 20% at the peak of the clinical reaction.
Babesia bovis is a “small” Babesia measuring up to 2 µm in diameter, while B. bigemina is larger and can extend to the full diameter of an erythrocyte. Both species show considerable morphological variation, however, making it difficult to distinguish one from the other. Large forms of B. bovis are quite common.

Single B. bovis organisms are round, oval or irregular in shape while paired forms are piriform or club-shaped (Figure 7). The angle between the paired organisms is often, but not invariably, obtuse (“bow-tie” appearance).

Single forms of B. bigemina are round, elongated or amoeboid in shape (Figure 8). Paired forms are typically piriform with an acute angle between them (Figure 5 & 8).

Diagnosis can also be confirmed by examination of brain smears. If post mortem changes have resulted in parasites no longer resembling typical Babesia parasites, comparison of parasitaemias in brain smears
and peripheral blood smears can indicate the *Babesia* species involved. *B. bigemina* parasitaemias in peripheral and brain smears will resemble one another, while *B. bovis* parasitemia in brain capillaries will tend to be substantially higher than that in peripheral smears.

For histopathological examination, specimens of brain, spleen, liver and lung should be submitted.

The indirect fluorescent antibody (IFA) test is specific for *B. bovis*, but cross-reactions with antibodies to *B. bovis* in the *B. bigemina* IFA are a particular problem. Unfortunately, in the standard IFA test the degree of serological cross-reaction that occurs between the four *Babesia* spp. present in southern Africa is such that accurate differentiation is sometimes difficult. Internationally validated enzyme-linked immunosorbent assays (ELISAs) for the diagnosis of *B. bovis* infection have been developed. There is still no similarly validated ELISA for *B. bigemina*.

Sequestration of parasitized red blood cells in the peripheral circulation and evidence of vascular stasis are striking in acute infections of *Babesia bovis* (Figure 9). Accumulations of haemosiderin and phagocytosed red blood cells are common in cells of the reticuloendothelial system, especially in the spleen, liver and lymph nodes. Other lesions include:

- degeneration and necrosis of the epithelium of the convoluted tubules in the kidneys and an accumulation of hyaline or granular casts in the tubular lumens;
- centrilobular hydropic or fatty degeneration to extensive centrilobular and midzonal hepatic necrosis and bile stasis;
- marked congestion of the sinusoids of the spleen and a reduced ratio of white to red pulp with the germinal centres containing few cells;
- oedematous and congested sinuses in the lymph nodes and depletion of lymphocytes in the germinal centres;
- oedema of the lungs in some cases;
- marked distension of the capillaries of the brain by parasitized red blood cells (perivascular haemorrhages are uncommon) (Figures 10 & 11);
- haemorrhages in the myocardium and hyaline degeneration of some myocytes; and
- degeneration of skeletal muscle fibres in the hind limbs.
Figure 9: Erythrocytes parasitised by *Babesia bovis* adhering to each other

Figure 10: Brain capillaries packed with erythrocytes parasitised by *Babesia bovis*
Figure 11: Cerebral babesiosis: cherry-red discoloration of the brain due to distension of blood vessels with parasites erythrocytes

In the kidneys, the capillaries are not packed as tightly with parasitized red blood cells as are those in the brain. Capillaries in the lungs are packed with red blood cells, but only a small proportion of the cells are parasitized.

In *Babesia bigemina* infections, histological changes are less pronounced than in *B. bovis* infections, and sequestration of infected red blood cells and vascular stasis are not features of the infection. Changes in the kidneys and liver are similar to those caused by *B. bovis*. Extensive necrosis of the red pulp of the spleen is common and large thrombi may be present.

Haemolytic anaemia, which is characteristically macrocytic and hypochromic, is a feature of *B. bovis* infections. Packed cell volumes (PCV) may fall to less than 0.10, total erythrocyte counts to less than 3.0 x 10⁶/µℓ, and total haemoglobin to less than 50 g/ℓ. Severe anaemia is particularly evident in protracted cases, while peracute cases may die with little evidence of anaemia. In cattle that survive, parasitaemia levels start decreasing –5 days after the onset of patency and evidence of erythrocytic regeneration can be detected 2 to 4 days later: anisocytosis, polychromasia, punctate basophilia, macrocytosis and reticulocytosis. Leukocytic changes are variable, ranging from leukopenia to leukocytosis.

Changes in blood chemistry reflect the consequences of circulatory stasis and hypotension, including renal and liver damage, and muscle degeneration. Characteristically, the following occur:

- significant increases in blood urea nitrogen and plasma creatinine levels,
- marked increases in unconjugated bilirubin levels, plasma creatine kinase and lactate dehydrogenase levels,
- the presence of haemoglobin in the serum to a level that may be as high as 5 g/L,
Livestock Health, Management and Production › High Impact Diseases › Vector-borne Diseases › Bovine Babesiosis

- increased plasma concentrations of fibrinogen and soluble fibrin,
- proteinuria during the acute phase of the infection of which 15 to 20 per cent is haemoglobin and 70 to 75 per cent albumin,
- metabolic and respiratory alkalosis as shown by elevated bicarbonate, excess base levels and a lowered pCO₂, and
- a terminal increase in lactate and pyruvate levels.

Haemolytic anaemia, the outstanding feature in *Babesia bigemina* infection, is very similar to that seen in *B. bovis* infections. Red blood cell destruction occurs more rapidly in severe cases however, and is accompanied by precipitous falls in PCVs, red blood cell counts and haemoglobin values. Osmotic fragility of the red blood cells increases during the acute phase of the infection, and serum haemoglobin levels are high in acute cases. Evidence of kidney and liver damage is similar to that seen in *B. bovis* infections.

**Differential diagnosis**

Babesiosis in bovines could be confused with anaplasmosis, but the latter generally leads to rumen stasis and constipation. Other causes of haematuria or haemoglobinuria may lead to a suspicion of babesiosis. Cerebral babesiosis could be confused with heartwater.

**CONTROL / PREVENTION**

**Vector control**

Eradication of the tick vectors (the so-called minimum disease situation) is the most desirable, permanent solution to the problem but is rarely considered practical or economical. The alternative approach, allowing natural endemic stability to develop by practicing limited or no tick control, is similarly unrealistic in areas where *R. appendiculatus* and *Amblyomma* spp. are well established. These regions are also endemic for other *Rhipicephalus (Boophilus)* spp. and essential control of other tick species will inevitably affect the epidemiology of redwater. In the long-term, this approach can be achieved by integrating the strategic use of acaricides, the application of vaccines in endemically unstable conditions and the use of tick-resistant breeds of cattle.

**Vaccination**

Cattle develop a durable immunity after a single infection with *B. bigemina* and *B. bovis*. This feature has been exploited with the use of live attenuated vaccines to immunize cattle.

The *Babesia* strains used in vaccines are of reduced virulence, but are not entirely safe. Reactions to *B. bigemina* may occur within seven days and to *B. bovis* within 10–14 days after vaccination. A practical recommendation is therefore to limit the use of vaccine to calves aged 3–9 months when non-specific
immunity will minimize the risk of reactions. When older animals have to be vaccinated, there is a risk of severe vaccine reactions, and they should be observed daily for three weeks after vaccination. Ideally, rectal temperatures of vaccinated cattle should be taken and animals treated when significant fever develops. Because of the risk of abortions, vaccination of pregnant cows is rarely advised.

The immunity lasts for several years in the case of *B. bovis*, but in the absence of natural challenge, it may break down in the case of *B. bigemina*. Redwater vaccines can be given at the same time as anaplasmosis and other vaccines, with the exception of heartwater vaccine. The incubation period after vaccination against babesiosis and heartwater is the same. Treatment is required in animals that react, i.e. showing an increased temperature reaction. At this stage, the owner will not be able to determine against which of the two pathogens the animal is reacting, and will therefore not be able to administer appropriate treatment. The incubation period after vaccination against anaplasmosis is longer than in the case of babesiosis, and reactions should not overlap; treatment of the animal reacting to the babesiosis vaccine will not influence development of immunity against anaplasmosis.

Vaccination against *B. divergens* is not commonly done. A formalin-inactivated vaccine has been used with some success in Austria since 1988, while an experimental live vaccine has been successfully used in Ireland.

**Treatment**

A number of drugs have been used (see Table 1). Recovery is the rule if specific treatment is given early in the course of the infection. If treatment is delayed, however, supportive therapy may be essential if the animal is to survive. Non-specific support includes the use of haematinics, vitamins, intravenous administration of fluids, good nutrition and provision of shade. Blood transfusions may be indicated in cattle with heavy parasitaemias and low PCVs (<0.10); histo-incompatibility seldom occurs at the first transfusion. In acute *B. bovis* infections, use of antioxidants such as vitamin E, and high doses of corticosteroids may help to offset the hypotensive and hypercoagulable state of the animal. In cases of cerebral babesiosis, intravenous use of hypertonic solutions of mannitol or glucose may provide temporary relief.

**Chemoprophylaxis**

Imidocarb and diminazene are the only babesicides with useful prophylactic properties for the short-term control or prevention of babesiosis. Treatment with imidocarb (3 mg/kg) will prevent overt *B. bovis* infections for at least four weeks and *B. bigemina* infections for at least eight weeks. Diminazene (3.5 mg/kg) will protect cattle against the two diseases for one and two weeks, respectively. Unfortunately, the prophylactic use of imidocarb may interfere with the development of immunity following vaccinations because the residual effect of the drug may eliminate or suppress the infection. The interval between the use of imidocarb and vaccination should be at least eight weeks if immunity to *B. bovis* is required and 16 weeks in the case of *B.
bigemina. If diminazene is used, the intervals for the two parasites should be about four and eight weeks, respectively.

Control of outbreaks

Procedures to be followed during an outbreak will depend largely on the number and manageability of the animals concerned, and the availability and cost of labour, drugs, vaccine and acaricides. One or more of the following actions can be taken to limit losses:

- Treat sick animals and separate them, if possible, from the rest of the herd.
- Have the diagnosis confirmed at a reputable laboratory.
- Treat unaffected cattle for ticks to prevent exposure.
- Consider immediate vaccination of all unaffected cattle.
- Consider use of a prophylactic treatment programme as mentioned above.

Table 1: Treatment

<table>
<thead>
<tr>
<th>Drug</th>
<th>Trade Name</th>
<th>Dosage and Route of Administration*</th>
<th>Uses and Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diamidine Derivatives</strong></td>
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<tr>
<td>Diminazene</td>
<td>Berenil</td>
<td>3.5 mg/kg i/m</td>
<td>Rapid activity against <em>B. bovis</em> and <em>B. bigemina</em>; well tolerated</td>
<td>-</td>
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<td></td>
<td>Ganaseg</td>
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<td></td>
<td>Trypazen</td>
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<td></td>
<td>Veriban</td>
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<tr>
<td></td>
<td>Babezene</td>
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<td></td>
<td>Dimisol</td>
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<tr>
<td>Amicarbalide</td>
<td>Diampron</td>
<td>5–10 mg/kg s/c, i/m</td>
<td>Rapid activity against <em>B. bovis</em> and <em>B. bigemina</em>; well tolerated</td>
<td>-</td>
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<tr>
<td></td>
<td>Pirodia</td>
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<tr>
<td>Imidocarb</td>
<td>Imizol</td>
<td>1.2–3.0 mg/kg s/c or i/m</td>
<td>Rapid activity against <em>B. bovis</em> and <em>B. bigemina</em>; well tolerated</td>
<td>Nephro- and hepatotoxic at high doses</td>
</tr>
<tr>
<td></td>
<td>Forray 65</td>
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<td></td>
<td></td>
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<tr>
<td>Phenamidine</td>
<td>Phenamidine</td>
<td>12 mg/kg s/c or i/m</td>
<td>Greatest activity against <em>B. bigemina</em></td>
<td>Cholinesterase inhibition</td>
</tr>
</tbody>
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* i/m = intramuscular; s/c = subcutaneous; i/v = intravenous

Quinoline Derivatives
### Drug Trade Name Dosage and Route of Administration* Uses and Advantages Disadvantages

<table>
<thead>
<tr>
<th>Drug</th>
<th>Trade Name</th>
<th>Dosage and Route of Administration*</th>
<th>Uses and Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinuronium Sulphate</td>
<td>Babesan</td>
<td>1 mg/kg s/c</td>
<td>Greatest activity against <em>B. bigemina</em></td>
<td>Slow effect on <em>B. bovis</em>. Cholinesterase inhibition: dose rate should not be exceeded (atropine counteracts toxic effects)</td>
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<tr>
<td></td>
<td>Ludobal</td>
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<td></td>
<td>Acaprin</td>
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<td></td>
<td>Pirevan</td>
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<tr>
<td>Acridine Derivatives</td>
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<tr>
<td>Euflavine</td>
<td>Gonacrine</td>
<td>2–4 mg/kg i/v</td>
<td>Rapid activity against <em>B. bovis</em> and <em>B. bigemina</em> spp.</td>
<td>Highly irritant if not given strictly i/v</td>
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<tr>
<td></td>
<td>Euflavine</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Trypan blue</td>
<td>Trypan blue</td>
<td>0.1 mg/kg i/v</td>
<td>Active against <em>B. bigemina</em></td>
<td>Little effect against <em>B. bovis</em>, irritant if not given i/v; discoloration of milk and carcass</td>
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<td>Antibiotics</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Tetracycline</td>
<td>Terramycin LA</td>
<td>20 mg/kg i/m</td>
<td>Mitigates <em>Babesia</em> vaccine reactions</td>
<td>Doubtful efficacy in clinical disease</td>
</tr>
</tbody>
</table>

### MARKETING AND TRADE / SOCIO-ECONOMICS

Bovine babesiosis is an OIE-listed disease, requiring the regulatory authorities of all infected countries to report outbreaks to the OIE twice annually. In South Africa, where bovine babesiosis occurs commonly, there are no statutory control measures in force and outbreaks do not have to be reported to the local veterinary authorities. According to the OIE Terrestrial Animal Health Code, countries that import cattle from countries infected with bovine babesiosis, should require the presentation of an international veterinary certificate attesting to a number of provisos. There are no trade bans associated with bovine babesiosis.
Severe losses may occur when susceptible cattle are brought into an area where *Rhipicephalus* (*Boophilus*) spp. are prevalent. Mortality rates of 5–10% under these conditions are quite common.

### IMPORTANT OUTBREAKS

In southern Africa, outbreaks are usually attributed to *Babesia bovis*. This is because the vector, *Rhipicephalus (Boophilus) microplus*, which was probably introduced from Asia, is apparently adapting to local conditions and is still expanding its distribution. When the vector enters a new area, the local cattle population is naive to *Babesia bovis* infection, and outbreaks occur. If the tick population becomes established, endemic stability will eventually ensue.

Outbreaks also occur in endemic areas after droughts are broken by good rains. During the drought environmental conditions are unfavourable to ticks and the tick population therefore decreases. The upshot is that a cohort of calves may grow up without being exposed at a young age and are therefore fully susceptible. Once the drought is broken and conditions become favourable for ticks, the expanding tick population leads to a high level of transmission of *Babesia bovis* and *Babesia bigemina* and outbreaks of disease may occur.

### FAQS

1. **Can bovine babesiosis be cured?**  
   Yes; various drugs effect clinical cure.

2. **Does treatment sterilise the infection?**  
   This depends on the species (*B. bovis* or *B. bigemina*) and the drug used, e.g. while treating with imidocarb will tend to sterilise both species, administration of diminazene will not sterilise *B. bovis* infection.

3. **Can one vaccinate cattle as a preventative measure?**  
   Yes. The vaccines consist of blood infected with live, but attenuated, parasites.

4. **Can cattle react to the vaccination by showing clinical signs?**  
   Yes. Although the vaccine strains are attenuated, cattle can react clinically, and then have to be treated.

5. **Does the vaccination have to be repeated?**  
   No. As this is a live vaccine, one vaccination should be sufficient. As one would only vaccinate in areas where there is natural challenge, infection through tick transmission will act as a booster.

6. **Is bovine babesiosis a notifiable disease in southern Africa?**
No. Babesiosis is widespread in southern Africa, and there are no statutory requirements for controlling or eradicating the disease. However, it is notifiable to the OIE (World Organisation for Animal Health), and each infected member country’s regulatory authority must submit a report twice yearly of outbreaks in the country.

7. **Can other species of livestock or wildlife harbour the infection?**

No. Both *Babesia bovis* and *Babesia bigemina* are specific to cattle. Artificial infections of African buffaloes were transitory in nature.

8. **Is eradication of ticks required to control the disease?**

No. An endemically stable situation is the ideal.

**REFERENCES**