5 Modular Guide Series
Control of Vector-Borne Diseases in Dogs and Cats

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Vector-borne diseases (VBDs) can affect dogs and cats. They are caused by a wide range of infectious agents including viruses, bacteria and parasites (protozoa and helminths).

The pathogens are transmitted by arthropod vectors such as ticks, fleas and Diptera (mosquitoes, phlebotomine sand flies, muscid flies).

The following series of modular guides for veterinary practice gives an overview of the most important VBDs and suggests control measures in order to prevent animal and/or human infection[1].

Key companion animal VBDs

5.1. Insect-borne diseases
   a. Canine leishmaniosis
   b. Cardiopulmonary dirofilariosis
   c. Subcutaneous dirofilariosis and other filarioid infections
   d. Bartonellosis (cat scratch disease)

5.2. Tick-borne diseases
   a. Babesiosis (piroplasmosis)
   b. Ehrlichiosis
   c. Anaplasmosis
   d. Lyme borreliosis

5.3. Vector-borne viral diseases
   a. Tick-borne encephalitis
   b. Louping-ill infection
   c. West Nile virus infection

Flea

Mosquito

Sand fly

Tick

Diagnosis of vector-borne diseases

Diagnosis of VBDs is performed considering anamnestic data (e.g. possibility of exposure to vectors and epidemiological information), presence of clinical signs and results of laboratory tests. ESCCAP recommends routine tests for diseases that are prevalent and pose health risks to animals and humans.

Blood samples can be examined microscopically for microfilariae (e.g. *Dirofilaria immitis, D. repens* and agents of other filarioid infections) and for merozoites of *Babesia* spp., for antigens (e.g. *D. immitis*), for specific antibodies (e.g. tick-borne encephalitis virus, *Babesia* spp.*, Leishmania* spp., *Borrelia* spp., *Anaplasma* spp., *Ehrlichia canis*) and DNA (e.g. filarioid infections, *Babesia* spp., *Ehrlichia* spp. and *Bartonella* spp.) The gold standard for the diagnosis of bartonellosis is a blood culture.

Smears obtained from superficial lymph nodes or bone marrow aspirates can be investigated for *Leishmania* sp. amastigotes.

Immunohistochemistry and virus isolation can be used to diagnose West Nile virus infection.

Preventive and control measures

Vector-borne diseases should first be controlled through ectoparasite management. Prevention of insect and tick bites by the application/ administration of repellents/insecticides/acaricides, in the form of impregnated collars, spot-on and spray formulations and tablets, is important. The basic objective is to interrupt transmission of pathogens and thus prevent clinical disease[1].

Animals should be groomed regularly to identify the extent of the flea/tick infestation. Visible ticks can be removed and carefully disposed of. "Hot-spot" areas endemic in sand flies/mosquitoes/ticks should be avoided.

When recommending an ectoparasite management programme, veterinarians should consider the animal's health status, environment, the presence of other animals and any relevant country of origin or travel destination.

[1] For more information see:
    ESCCAP Guideline 03: Control of Ectoparasites in Dogs and Cats.
    ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

Preventing zoonotic infection

Pet owners should be informed about the potential health risks of VBDs. Generally, important preventive measures for pet owners in terms of ectoparasites are:

- Control ectoparasite infestations of pets through treatment with appropriate ectoparasiticides, particularly for ticks and parasitic insects.
- Control of infections through regular diagnostic testing in endemic areas.
- Minimise exposure to environments where vectors may occur.
- Practise good personal hygiene and check for the presence of ticks on clothes and the skin after potential exposure.
- Advise people at risk of exposure to vector-borne zoonotic pathogens of the health risks, particularly during pregnancy, or when there is an existing illness or immunosuppression.
- Give administration recommendations to achieve optimal owner compliance.

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In Europe, canine leishmaniosis is predominantly caused by *Leishmania infantum*. The vectors are blood-sucking females of the genus *Phlebotomus* (sand flies).

Dogs are the main reservoir of *L. infantum* but cats and other wild carnivores can also be hosts. Many other mammalian species can become infected including humans. Human cutaneous and visceral leishmaniosis is an important vector-borne zoonotic disease in southern Europe. Clinical cases of human leishmaniosis are mainly reported in children and immunocompromised patients.

Distribution

The endemic area of sand flies and/or leishmaniosis in Europe extends up to the southern alpine border in Italy and France[1].

Life Cycle

The life cycle starts when an infected sand fly takes a blood meal and injects promastigotes into the skin of the host.

Leishmania spp. may also be transmitted intra-uterine from mother to offspring, via infected blood donors or through venereal transmission.
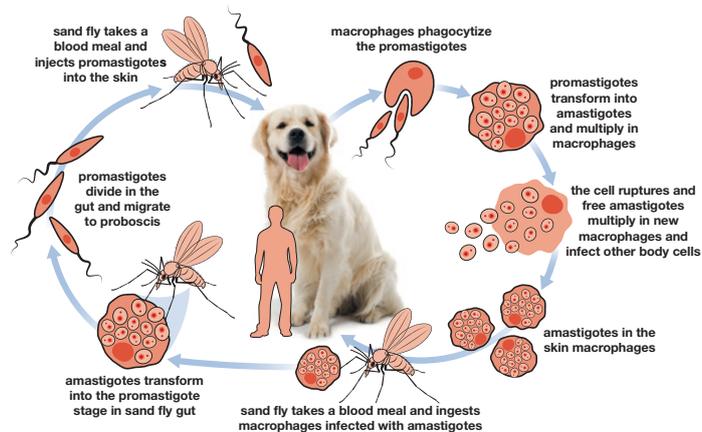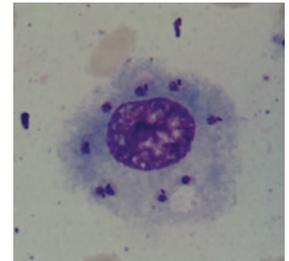
Clinical Signs

The clinical presentation of canine leishmaniosis ranges from asymptomatic infection to life-threatening disease. **Local** cutaneous lesions at the site of the initial phlebotome bites (ear pinnae, nose and abdomen) are often the first signs of infection.

Enlargement of single or multiple lymph nodes is often observed and may be accompanied by weight loss, anorexia and weakness.

Phlebotomus perniciosus[A]

sand fly takes a blood meal and injects promastigotes into the skin

macrophages phagocytize the promastigotes

promastigotes transform into amastigotes and multiply in macrophages

promastigotes divide in the gut and migrate to proboscis

the cell ruptures and free amastigotes multiply in new macrophages and infect other body cells

amastigotes in the skin macrophages

amastigotes transform into the promastigote stage in sand fly gut

sand fly takes a blood meal and ingests macrophages infected with amastigotes

Leishmania infantum
amastigotes in a macrophage from a lymph node aspirate

[A] Photo courtesy of Dr. Rosa Gálvez, Veterinary Faculty, UCM.

General skin lesions may develop. These are normally non-pruritic and keratoseborrhoeic but may become ulcerative, papular, pustular or nodular.

Other clinical signs might include gastrointestinal disorders, lameness, vasculitis, glomerulonephritis and ocular lesions. Cardiorespiratory and neurological disorders may occur in rare cases.

Common laboratory findings include anaemia, thrombocytopenia, leucopenia, hyperglobulinaemia and hypoalbuminaemia. Proteinuria and variable azotaemia with an increase in the urine protein/creatinine ratio can be seen in some sick dogs and are considered an indicator of a poor clinical prognosis.

[1] See www.esccap.org for maps of the distribution of canine leishmaniosis in Europe.

[2] See www.esccap.org for therapy tables by country or region.

[3] See LeishVet Guidelines on www.leishvet.org

[4] See ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

Diagnosis

Diagnosis is based upon typical clinical signs, epidemiological information and laboratory test results. Detection of antibody titres, CBC, biochemical profile and urinalysis including a urine protein/creatinine ratio should always be performed.

Direct diagnosis is possible by detecting amastigote stages in stained smears obtained from superficial lymph node or bone marrow aspirates. PCR is more sensitive using, in descending order of sensitivity: bone marrow or lymph node aspirates, skin, conjunctival swabs, buffy coat, and peripheral whole blood.

Serology is the first step towards detecting a specific antibody response in dogs from 12 weeks after initial infection. Commercial kits are available for a qualitative diagnosis. To confirm infections and for clinical management post chemotherapy, indirect fluorescent antibody test (IFAT) or enzyme-linked immunosorbent assay (ELISA) are required.

Treatment

Treatment is only indicated in symptomatic cases and does not eliminate infection. Meglumine antimoniate or miltefosine in combination with allopurinol are effective in the treatment of symptomatic dogs. Allopurinol should be continued after completion of the combination therapy to reduce the risk of relapses[2,3]. Immunomodulatory therapy has been proposed to support treatment success.

Dogs on allopurinol treatment should be fed a purine-restricted diet. *L. infantum* infection is not eliminated by the currently available treatment regimens and long-time to life-long follow-up and repeated treatment cycles are often necessary.

Dogs with concurrent renal disease have a poor prognosis and should be supported by standard care.

Control

Preventing bites during the phlebotome season, by the application of repellents/insecticides, is the most promising strategy.

Depending on the area, it may be necessary to protect dogs year round (e.g.: Spain, southern Italy).

Keeping dogs indoors at dusk and dawn and using insecticidal room sprays and mosquito window/door/bed nets (with <0.4 mm mesh) treated with pyrethroids can also reduce phlebotome bites. Keeping the environment clear of garbage and organic matter may reduce the breeding sites of phlebotomes[4].

Vaccines against *Leishmania* infection are available which are effective in the population to reduce the occurrence of the disease, if combined with a repellent.

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Cardiopulmonary dirofilariosis is caused by the filarioid nematode species *Dirofilaria immitis* which infects the vascular system of dogs and cats.

Mosquitoes act as vectors for the heartworm *Dirofilaria immitis* which is the most pathogenic filarioid species.

D. immitis is zoonotic. Visceral localisation in the human such as 1–4 cm pulmonary nodules may mimic neoplasia.

Distribution

D. immitis is endemic across southern Europe, such as Italy and Spain, and in Hungary, Slovakia, Romania and Bulgaria.

Life Cycle of *D. immitis*

Infective larvae enter the host when mosquitoes feed. *D. immitis* larvae (L4, L5) migrate via the connective tissue and bloodstream to the pulmonary arteries and the right heart.

Mature females release microfilariae in dogs 6–7 months post infection, which become available to blood-sucking mosquitoes. The cat is considered unlikely to infect mosquitoes
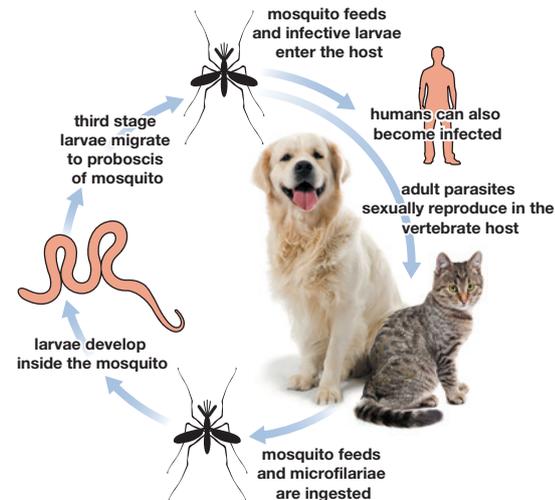
Clinical Signs

Heartworm disease is essentially a pulmonary disease and the right heart is affected only in the latter stages. It is severe and potentially fatal.

Dog

Clinical signs begin with weakness, moderate weight loss and chronic cough followed by dyspnoea and sometimes syncope after exercise/excitement. As right congestive heart failure develops, ascites, oedema, anorexia and severe weight loss may be observed. Migration of adult worms from the pulmonary arteries to the right heart causes "caval syndrome" typified by dyspnoea, a tricuspid cardiac murmur and haemoglobinuria; the outcome is usually fatal unless worms are surgically removed.

Cat

Most cats infected by *D. immitis* show no clinical signs. Others may develop acute respiratory signs (heartworm-associated respiratory disease or HARD), such as coughing, dyspnoea and haemoptysis, frequently accompanied by vomiting, around 10 weeks post infection. Sudden death in apparently healthy cats may occur.

mosquito feeds and infective larvae enter the host

humans can also become infected

adult parasites sexually reproduce in the vertebrate host

mosquito feeds and microfilariae are ingested

larvae develop inside the mosquito

third stage larvae migrate to proboscis of mosquito

D. immitis life cycle

Diagnosis

Dog

Heartworm infection can be detected by blood tests to demonstrate circulating microfilariae or adult antigens in serum or plasma.

Antigens from adult female heartworms are present approximately 6–8 months post infection. PCR and other techniques can be used to differentiate species based on isolated microfilariae. Thoracic radiography and echocardiography are useful for staging the severity of the heartworm infection.

Cat

Cats are considered to be rarely microfilaremic and detection of microfilariae in the blood has therefore very low sensitivity. Blood/serological antigen tests for adult female heartworm have a high specificity and may provide proof of infection; however, tests often return negative results due to immature parasite stages, because only males are present or because the worm burden is low. A negative test does not rule out infection. Antigen testing can also be performed in cats.

Thoracic radiographic alterations compatible with echocardiographic findings are needed for diagnosis in cats. Cardiac ultrasound should always be performed if feline heartworm infection is suspected.

Treatment

Macrocyclic lactones can be used as **microfilarial therapy** for 8 weeks prior to adulticidal therapy in dogs. They should be used in combination with doxycycline for the first 4 weeks to reduce *Wolbachia* bacteria (these are obligate endosymbionts of *D. immitis*).

Melarsomine dihydrochloride is an effective **adulticide** in dogs. Complications, due to pulmonary thromboembolism, should be reduced by rest and corticosteroid administration.

Surgical intervention is advised when multiple worms have been displaced into the right cardiac chambers causing caval syndrome.

There is no safe and effective adulticide for cats. Decreasing doses of prednisolone can relieve respiratory distress.

Control

Oral or spot-on macrocyclic lactones administered monthly to dogs or cats (or an extended duration spot-on macrocyclic lactone administered every 12 weeks to cats) throughout the transmission season[1] or an annual treatment with an injectable, sustained-release formulation[2] in dogs are effective against *D. immitis* L3 and L4 and can prevent disease caused by adult worms. Dogs and cats should be given macrocyclic lactones within 30 days of travelling into an endemic area, and monthly or 12-weekly thereafter with the last dose given after return to a non-endemic area.

Topical administration of synthetic pyrethroids such as permethrin or deltamethrin can be used on dogs as mosquito repellents. In endemic areas, dogs should be tested annually for circulating antigens and blood microfilariae.
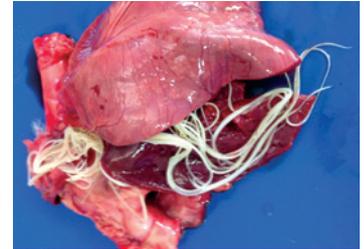
Adult heartworms in a canine heart

Heartworm infection causes pulmonary disease

[1] See www.esccap.org for links to therapy tables by country or region.

[2] See ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

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Subcutaneous dirofilariosis is caused by filarioid nematodes of the species *Dirofilaria repens*. In Europe, *D. repens* is the most important cause of human filarioid infection.

Dirofilaria repens is the most frequent species associated with subcutaneous filariosis of dogs and cats.

Most human cases are asymptomatic and are often diagnosed after the surgical removal of a 1–6 cm nodule containing worms. Pre-adult worms are often observed in subconjunctival or subcutaneous swellings that may mimic neoplasia.

Acanthocheilonema reconditum is found in subcutaneous tissues and fasciae, the peritoneal cavity and the kidneys of canids. *Acanthocheilonema dracunculoides* is found in the peritoneal cavity and may be transmitted by fleas, louse flies or *R. sanguineus*. *Cercopithifilaria* spp. are parasites of the subcutaneous tissue and fasciae of canids and transmitted by the tick *R. sanguineus*.
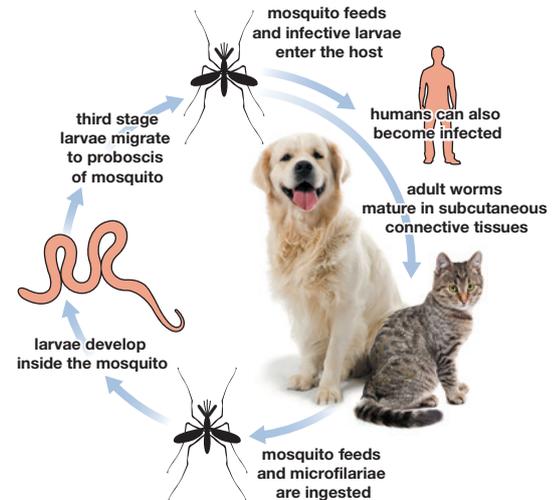
Distribution

Autochthonous *D. repens* infections have been reported in Germany, Austria, Italy, Czech Republic and Poland[1]. Endemic areas of *D. repens* and *D. immitis* overlap in many regions

Acanthocheilonema reconditum and *A. dracunculoides* infections have been found in hunting dogs and dogs living outdoors in some countries such as Spain and southern Italy. *Cercopithifilaria spp.* have been found in southern Europe.

Life Cycle of *D. repens*

Infective larvae enter the host when mosquitoes feed. *D. repens* larvae (L4, L5) migrate into the subcutaneous connective tissues where they reach maturity. Adult worms are found between subcutaneous and deep connective tissue layers in most parts of the body.

mosquito feeds and infective larvae enter the host

humans can also become infected

third stage larvae migrate to proboscis of mosquito

adult worms mature in subcutaneous connective tissues

larvae develop inside the mosquito

mosquito feeds and microfilariae are ingested

D. repens life cycle

[1] See www.esccap.org for maps of the distribution of *Dirofilaria* spp. in Europe.

Clinical Signs

Dirofilaria repens causes subcutaneous non-inflammatory nodules in dogs and cats containing adult parasites or immature stages. During surgery, the parasite may also be found in the perimuscular fasciae, perirenal fat or abdominal cavity or may migrate to the ocular conjunctiva or other tissues.

Infections with *A. reconditum*, *A. dracunculoides* and *Cercopithifilaria* spp. are mostly subclinical.

Diagnosis

Adult worms are found between subcutaneous and deep connective tissue layers in most parts of the body.

D. repens infection can be detected after the surgical removal of a subconjunctival or subcutaneous nodule containing worms.

Treatment

Spot-on moxidectin is licenced in all EU countries as adulticide therapy for *D. repens* infection and reduction of microfilaria in dogs.

Topical administration of synthetic pyrethroids such as permethrin and deltamethrin can be used as mosquito repellents in dogs. Dogs should be checked annually for circulating antigens and blood microfilariae.
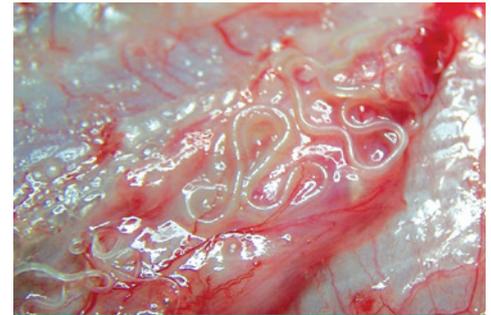
The control strategies for *D. immitis* have also been found to be effective in preventing subcutaneous infection by *D. repens* in dogs and cats.

Culex pipiens [B]

The worm may cause skin nodules and swelling

D. repens resides in the subcutaneous tissue

[B] Photo courtesy of Adolfo Ibanez Justicia, Centre Monitoring Vectors, NVWA, The Netherlands.

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The bacterium *Bartonella henselae* is the causative agent of bartonellosis or cat scratch disease (CSD) in humans. Cats are considered the main reservoirs and the vectors between cats are fleas and their faeces.

Transmission to humans occurs through contact with subclinically-infected cats usually by being scratched or bitten. It is unclear whether humans can become infected directly by being bitten by the cat flea.

Distribution

Cat fleas and *Bartonella* are distributed worldwide[2]. Infection occurs most frequently in cats less than 2 years of age, stray cats, cats with outdoor access, and animals in multi-cat households. Indoor cats, however, can be also affected.

Clinical Signs

Most *Bartonella* spp. infections in cats remain subclinical. Bacteraemia develops within 1–3 weeks after initial infection, with chronic recrudescence for up to 21 months. Clinical signs are only observed in immunosuppressed cats.

Infection has also been associated with urinary tract disease and reduced reproductive performance.

In dogs, *Bartonella* spp. have been associated with endocarditis, myocarditis, hepatitis and rhinitis.

In humans, infection with *B. henselae* does not always cause bartonellosis or cat scratch disease (CSD). Immunocompetent patients with CSD usually experience pustule formation at the infection site, regional lymphadenopathy, abscess formation and sometimes fever. Uncomplicated CSD is usually self-limiting but may last for months until complete resolution.

The course of the disease is much more complicated in immunocompromised patients as bacillary peliosis, bacillary angiomatosis, endocarditis, retinitis and encephalopathies can develop.

Ctenocephalides felis

Infected flea dirt may be detected between cats' teeth and under their claws

Diagnosis

Diagnosis is based upon the identification of clinical signs (if other causes can be excluded), response to treatment and blood culture. It is also possible to detect *Bartonella* spp. DNA in samples of blood, tissue, cerebrospinal fluid or aqueous humour. Antibodies can be detected serologically from day 10–14 after infection. It only shows cat or dog exposure to *Bartonella* spp. For diagnosis of clinical bartonellosis, repeated testing of serum samples should show a rising antibody titre.

Response to antibiotic treatment effective against *Bartonella* spp. may support a tentative diagnosis.

Treatment

Treatment is recommended for animals showing clinical signs where other differential diagnoses have been ruled out and/or for those in contact with immunocompromised persons. Antibiotics reduce bacteraemia but do not eliminate the pathogen. Possible therapies include amoxicillin/clavulanic acid, doxycycline, and fluoroquinolones[1].

In cats and dogs that respond to therapy, treatment should be continued for at least 28 days or 2 weeks after remission of the clinical signs. If the animal still shows clinical signs after 7 days, azithromycin may be recommended until 2 weeks after signs subside.

Control

Flea control is the primary measure for the prevention of *Bartonella* spp. infection. Insecticide treatments and strict hygiene are required to minimise the presence of flea faeces on the animal and in its environment[2].

New cats should only be introduced into a household with immunocompromised persons if the cats are are older than 1 year, free of fleas and have tested with PCR negative for *Bartonella* spp. These cats should also be kept indoors.

Wounds caused by cats should be immediately washed and disinfected.

Wounds caused by cats should be immediately washed and disinfected

Persistent scratching due to irritation caused by fleas or their bites

[1] See ESCCAP Guideline 05:
    Control of Vector-Borne Diseases in Dogs and Cats.

[2] See ESCCAP Guideline 03:
    Control of Ectoparasites in Dogs and Cats.

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Babesiosis is caused by haemoprotozoa of the genus *Babesia* which infect erythrocytes and are transmitted by ticks. The parasites are host-specific with regard to both the transmitting tick species and the mammalian host. *Babesia* spp. infections of dogs and cats have not been reported in humans.
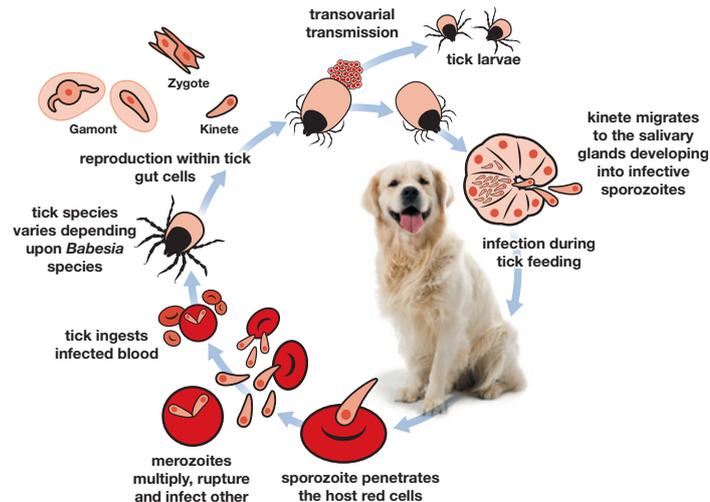
Distribution

Endemic areas of canine babesiosis in Europe are related to the distribution of the tick vector[1].

In central Europe, canine babesiosis is a frequently imported disease with the endemic area of the *B. canis* vector (*Dermacentor reticulatus*) expanding up to the Baltic region. Small *Babesia* spp. sporadically occur. Babesiosis in cats has occasionally been observed but the species and vectors are still unknown.
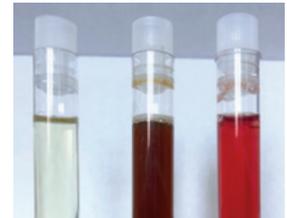
Life Cycle

Female Ixodidae generally require 24–48 hours of initial feeding before *Babesia* sporozoites are transmissible within their saliva to the dog. Transmission may be more rapid in male ticks because they repeatedly feed taking only small amounts of blood, they co-feed with females and they possibly feed from several different hosts.

Gamont
Zygote
Kinete

reproduction within tick gut cells

tick species varies depending upon *Babesia* species

tick ingests infected blood

merozoites multiply, rupture and infect other red cells

sporozoite penetrates the host red cells

transovarial transmission

tick larvae

kinete migrates to the salivary glands developing into infective sporozoites

infection during tick feeding

Babesia canis life cycle

Babesia canis is transmitted by *Dermacentor reticulatus*

Normal urine compared to dark–brown and bloody urine due to haemoglobinuria

Clinical Signs

Babesiosis in dogs may be subclinical or may follow a peracute, acute or chronic course[2]. Acute disease causes fever, lethargy, anorexia, jaundice, vomiting and, in some cases, red-coloured urine and faeces.

Dog with jaundice[C]

Diagnosis

Acute babesiosis can often be confirmed by the examination of freshly-prepared thin blood smears to detect Babesia spp. (Giemsa-stain or Diff-Quik). Peripheral capillary blood from the ear pinna or tip of the tail may yield higher numbers of parasitised erythrocytes. Diagnosis of chronic infections or carrier dogs is a challenge due to very low and often intermittent parasitaemia in these cases.

Specific antibodies can be detected from two weeks after initial infection using ELISA or IFAT.

Clinicopathological findings include haemolytic anaemia, thrombocytopenia, neutropenia and sporadic haemoglobinuria.

Signs of chronic disease include moderate depression, intermittent fever, anaemia, myositis and arthritis.

Clinical signs of feline babesiosis in the latter stages of disease include lethargy, anorexia, weakness and diarrhoea. Fever with icterus is uncommon in cats.

With serology alone, acute infections may therefore be missed. Also seropositivity may not be synonymous with disease and may be detected in chronically infected dogs without clinical signs which have been in contact with the parasite but did not become ill.

Highly sensitive and species-specific PCRs are used for routine molecular diagnosis in blood and to aid the differentiation of Babesia spp.

Treatment

For B. canis chemotherapy with imidocarb dipropionate, and in some countries phenamidine, should be initiated immediately after diagnosis. This treatment improves clinical status, although parasitological cure is unlikely to be achieved[2]. Treated dogs do not develop an immune response capable of protecting against re-infection. Supportive therapy, including blood transfusion and rehydration, is strongly recommended.

Chemotherapy can reduce the clinical severity and mortality rate of babesiosis caused by small Babesia spp. in dogs and Babesia spp. in cats[2].

Control

Effective **tick control**[1] significantly reduces the risk of Babesia spp. infection in dogs living in, or travelling to/through, endemic areas. Regular application of acaricides is advised, using water resistant products with a long duration of efficacy. It is best to avoid or limit access to areas of known high tick density, especially at times of the year when ticks are most active. Ticks found after outdoor activity should be removed.

Vaccination can prevent severe disease, but not infection. The level of protection may vary so re-vaccination every six months in highly endemic areas is advised. Vaccination of pregnant or lactating bitches is not recommended.

[1] See ESCCAP Guideline 03: Control of Ectoparasites in Dogs and Cats.

[2] See ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

[C] Photo courtesy Dr Els Acke.

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Ehrlichia spp. are vector-transmitted, obligate intracellular bacteria. *Ehrlichia canis* is the aetiological agent of canine monocytic ehrlichiosis (CME) and infects mainly lymphocytes and monocytes wherein the typical, microscopically visible microcolonies (morulae) develop.

The main host of *E. canis* is the dog and the vector is the tick *Rhipicephalus sanguineus*. *E. canis*, or a closely-related species, has been described in cats but the clinical relevance is unclear.

Concurrent vbd-infections are common in the same dog, also because some species are transmitted in the same arthropod vector.

E. canis is not considered a zoonotic agent.

Distribution

E. canis prevalence corresponds to the geographical distribution of its vector *R. sanguineus*. Infections in dogs have been reported in France, Italy, Portugal, Greece, Switzerland, Germany, UK and Spain.

Life Cycle

R. sanguineus preferentially feed on canids and may acquire *E. canis* from bacteraemic animals. During the incubation period of 8–20 days, the pathogens multiply in leucocytes, forming morulae within circulating mononuclear cells.

Ehrlichia spp. spread via the mononuclear phagocytic system to the liver, spleen and lymph nodes. This can cause impaired platelet function, sequestration and destruction.

Transmission by blood transfusion has also been described, therefore screening of canine blood products is strongly recommended in endemic areas.

Engorged *Rhipicephalus* tick [D]
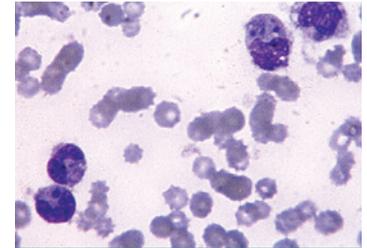
Ehrlichia canis [E]

[D] Photo courtesy Frederic Beugnet, Boehringer Ingelheim.
[E] Photo courtesy of Erik Teske, Utrecht University.

Clinical Signs

During the **acute phase** of canine monocytic ehrlichiosis, which lasts around 1–3 weeks, dogs may show weakness, lethargy, anorexia, dyspnoea, fever, lymphadenopathy, splenomegaly, weight loss and vomiting[2].

Clinical signs associated with haematological abnormalities include pale mucous membranes and bleedings such as petechiae, ecchymosis, epistaxis, prolonged bleeding during oestrus, haematuria and melaena. Clinicopathological abnormalities include thrombocytopenia, leucopenia and normocytic, normochromic, and non-regenerative anaemia.

During the **subclinical phase**, which may last for weeks or months, the dogs appear clinically normal.

Chronic disease is characterised by weakness, apathy, sustained weight loss, fever, pancytopenia, lymphadenopathy, splenomegaly, peripheral oedema in the hind limbs and scrotum, pale mucous membranes, a predisposition for bleedings as well as mucopurulent ocular and nasal discharges. In addition, interstitial pneumonia with dyspnoea, renal dysfunction, glomerulonephritis, arthritis, polymyositis and lameness may occur. Cases of chronic severe CME in dogs have a poor prognosis.

Reports of *E. canis* infections in cats are rare and clinical manifestations are not well described.

Diagnosis

Diagnosis is based upon **anamnesis** to determine tick exposure, clinical signs, laboratory abnormalities and quantitative serology and/or PCR[2].

A **morphological diagnosis** is confirmed if microscopic examination of blood smears reveals morulae in mainly monocytes but also lymphocytes (not granulocytes). However, these are rarely infected (4% in the acute phase). To increase diagnostic sensitivity, buffy coat smears or thin blood smears of blood or lymph node aspirates should be examined.

In **serology**, antibodies may be detected by an indirect immunofluorescence assay (IFA). Seroconversion may occur 1–4 weeks after exposure. Point-of-care immunoassay tests have been developed for use in general practice.

A positive PCR result generally confirms the presence of an infection. However, a negative PCR result does not exclude it[2].

Treatment

Antirickettsial agents such as doxycycline are used daily over 4 weeks to treat ehrlichiosis along with supportive therapy.

German Shepherd with epistaxis

Control

Effective protection against ticks is the primary measure for the prevention of *Ehrlichia* infections[1].

[1] See ESCCAP Guideline 03:
    Control of Ectoparasites in Dogs and Cats.

[2] See ESCCAP Guideline 05:
    Control of Vector-Borne Diseases in Dogs and Cats.

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Anaplasmosis is caused by *Anaplasma* spp. which are vector-transmitted obligate intracellular bacteria. In Europe, *A. phagocytophilum* and *A. platys* have been reported in dogs.

A. phagocytophilum is transmitted by *Ixodes ricinus* ticks, starting after 24–48 hours of feeding. *A. platys* are likely transmitted by ticks such as *Rhipicephalus sanguineus* and *Dermacentor* spp.

Humans can be infected with *A. phagocytophilum*. Direct transmission from infected dogs to humans has not been reported. There is a risk that dogs may carry infected *Ixodes* spp. ticks into the human environment. Blood from infected dogs should be handled with caution.

Distribution

The geographical distribution of *A. phagocytophilum* and *A. platys* infections generally corresponds to the distribution of their tick vectors, *Ixodes ricinus* and *Rhipicephalus sanguineus* respectively[1].

Life Cycle

After infection by a blood-sucking tick, the incubation period is 1–2 weeks. *A. phagocytophilum* develops typical microcolonies (morulae) in predominantly neutrophils and rarely eosinophils. Infected cells are found in the circulating blood and the spleen, liver and bone marrow.

Clinical Signs

A. phagocytophilum infection is often asymptomatic and clinical signs may be non-specific, such as sudden onset lethargy, anorexia, fever, bleeding (petechiae, melaena, epistaxis), lameness, pale mucous membranes, diarrhoea, vomiting, tachypnoea, splenomegaly and enlarged lymph nodes. Rare signs include cough, uveitis, limb oedema and polydipsia/polyuria[1].

Laboratory abnormalities may include thrombocytopenia, non-regenerative normocytic normochromic anaemia, lymphopenia, monocytosis, leucopenia, leucocytosis, hyperglobulinaemia, hypoalbuminaemia and increased liver enzymes (ALP). Rare findings include hyperbilirubinaemia and renal azotaemia.

Typical morulae of *Anaplasma phagocytophilum* in a neutrophilic granulocyte[E]
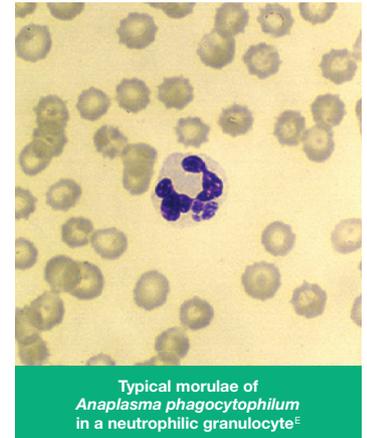
[E] Photo courtesy of Erik Teske, Utrecht University.

The clinical manifestations of **A. platys** infection vary from subclinical to distinct clinical syndromes such as cyclic thrombocytopenia depending on the geographical region (USA vs. Europe)[1]. Concurrent infections with *E. canis* or *Babesia* spp., have been reported which makes it difficult to attribute specific clinical signs to a single pathogen.

Clinical cases of feline anaplasmosis are rare and patients may suffer from lethargy, anorexia, fever, lymphadenopathy, anaemia and conspicuous thrombocytopenia.

Diagnosis

Diagnosis is based upon a thorough **anamnesis** to determine possible tick exposure[2], clinical signs, laboratory abnormalities and serology and/or PCR[1].

A **morphological diagnosis** is confirmed if microscopic examination of blood smears reveals morulae in neutrophil granulocytes and more rarely eosinophils (*A. phagocytophilum*) or platelets (*A. platys*). To increase diagnostic sensitivity, buffy coat smears should be examined.

In **serology**, antibodies may be detected by an indirect immunofluorescence assay (IFA).

Seroconversion may occur 1–4 weeks after exposure. Point-of-care immunoassay tests have been developed for use in general practice.

Two serological tests at 3–4 week intervals are required to confirm seroconversion. A single positive result combined with clinical signs is not sufficient evidence for a diagnosis. It is important to note that *A. phagocytophilum* might cross react with *A. platys*.

A positive PCR result generally confirms the presence of an infection. However, a negative PCR result does not exclude it[1].

Treatment

Antirickettsial agents such as doxycycline are used to treat anaplasmosis along with supportive therapy. The prognosis of *A. phagocytophilum* infections is fairly good.

Control

Effective protection against ticks is the primary measure for the prevention of *Anaplasma* spp. infections[2].

Anaplasmosis can cause sudden onset lethargy in dogs

Anaemia indicated by pale mucous membranes[c]

[1] See ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

[2] See ESCCAP Guideline 03: Control of Ectoparasites in Dogs and Cats.

[c] Photo courtesy Dr Els Acke.

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Lyme borreliosis is caused by *Borrelia burgdorferi* sensu lato spirochaetes. They infect many mammals and birds and are transmitted by ticks (*Ixodes ricinus*, *I. hexagonus*, and *I. persulcatus*).

Infections have been demonstrated in dogs but disease in cats is poorly understood. Human infections are of major public health importance.

Distribution

Endemic areas are directly related to the distribution of the tick vectors. Lyme borreliosis is present all over Europe, except in extremely hot southern or cold northern areas.

Life Cycle

Ixodidae ticks are recognised vectors of *B. burgdorferi* sensu lato. Tick vectors can acquire *Borrelia* spp. when feeding on an infected "reservoir host". Several animal species have been identified as reservoirs in Europe, including many wild mammals and birds.

Borrelia organisms accumulate in the salivary glands of ticks and are transmitted transstadially. There is no transovarial transmission. The tick must be attached for at least 16–24 hours before pathogen transmission to a new host occurs. Bacteria remain in the skin of the host before disseminating to other tissues.

Clinical Signs

Clinical borreliosis is not clearly defined in dogs. Most infected dogs are subclinical and it is difficult to correlate naturally acquired *B. burgdorferi* infection with clinical signs such as fever, lameness, myalgia and lethargy.

Puppies may be at higher risk of "Lyme arthropathy" which is lameness in one or more joints. The term "Lyme nephropathy" has been adopted for a syndrome of protein losing immune complex nephropathy that occurs in 2% of seropositive dogs.

Clinical manifestations in naturally-infected cats are uncommon.

Ixodes hexagonus [F]

[F] Photo courtesy of Faculty of Veterinary Medicine, Utrecht University.

Diagnosis

Direct diagnosis of *Borrelia* by culture, cytology or PCR may be difficult because some studies suggest that *B. burgdorferi* invades the soft tissues of dogs after transmission by ticks but does not appear to be present in the bloodstream or urine. That is why the organism is rarely found in blood, urine or CSF but can be detected in skin and/or synoviae.

In **serology**, *Borrelia* antibodies usually appear 3–5 weeks after infection and can be detected using immunochromatographic tests. However, positive results indicate exposure to the pathogen rather than clinical disease. If dogs suspected of having Lyme borreliosis are seropositive, a Western Blot immunoassay or line immunoassay (LIA) can be carried out to check for specific antibody signals. Additionally, specific anti-C6 peptide antibody reactions are highly sensitive and specific in the blood, serum or plasma of naturally infected dogs. Serologic cross-reactivity among *Borrelia* spp. as well as *Leptospira* spp. may occur with a conventional IFAT.

Treatment

Doxycycline is the antibiotic of choice. A response to therapy should be evident within 1–4 days in the case of polyarthritis but does not clear the infection in all dogs.

Control

Tick control is currently the preferred method of disease prevention[1]. Reducing infestation by infected ticks prevents transmission of the pathogen, reducing the risk of infection and clinical disease.

The use of Lyme borreliosis vaccines is controversial.

Dogs and cats are not reservoirs of *B. burgdorferi* and thus do not present a public health concern. However, ticks collected from dogs or cats may carry the pathogen and therefore need to be carefully disposed following removal to prevent the possible transmission of *Borrelia* spp. to new hosts, including humans.

Fluid aspiration from a joint with polyarthritis[C]

[1] See ESCCAP Guideline 03: Control of Ectoparasites in Dogs and Cats.

[C] Photo courtesy Dr Els Acke.

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Vector-borne viral infections are usually initiated by the bite of an infected tick or mosquito.

Ixodes ricinus ticks can carry the European **tick-borne encephalitis virus** (TBEV) and transmit it to a wide range of vertebrates including humans.

Louping-ill virus (LIV) is a tick-borne, zoonotic virus mainly detected in sheep and red grouse. It is transmitted by the *Ixodes ricinus* tick. Transmission may also occur after exposure to the tissues or unpasteurised milk of infected animals and via aerosols. Stockmen, abattoir workers, butchers and veterinarians who have close contact with sheep or other potentially infected species, are most at risk and can develop flu-like symptoms or neurological signs after exposure. However, the illness is rarely fatal.

West Nile virus (WNV) is transmitted by mosquitoes with wild and domestic birds acting as the main hosts. Humans and other mammalian species (mainly horses) are dead-end hosts. Virus transmission in humans may also occur via blood transfusion and organ transplantations.

Distribution

European **TBEV** can occur in areas where its tick vector, *I. ricinus*, is present. Larvae, nymphs and adult ticks can be infected and transstadial and occasionally transovarial transmission occurs[1]. Endemic regions include Austria, Czech Republic, Denmark (eastern), France (eastern), Germany, Greece, Hungary, Italy (northeastern), Netherlands, Norway, Slovakia, Slovenia, Russia, Sweden, the Baltic States, Switzerland, UK[2].

LIV has mainly been reported in the British Isles and Scandinavia. Transstadial and overwintering of the virus occurs but usually not transovarial transmission. The virus is closely related to TBEV.

In Europe, **WNV** is mainly present in Mediterranean countries (Italy, France, Greece, Spain and Portugal) and eastern European countries (Bulgaria, Romania, Hungary)[2].

Clinical Signs

Clinical manifestations of vector-borne viral diseases are diverse and those reported in pets are almost exclusively from canine clinical cases[3].

Ixodes ricinus [G]

Culex pipiens [B]

[G] Photo courtesy Ireneusz Irass Walędzik.
[B] Photo courtesy of Adolfo Ibanez Justicia, Centre Monitoring Vectors, NVWA, The Netherlands.

TBEV may cause peracute lethal, acute and chronic subclinical disease. Rottweiler dogs seem to be over-represented in reported cases. Fever, apathy, weakness, anorexia and severe encephalitis have been observed.

Acute or subclinical viral encephalomyelitis may be caused by **LIV** infection in sheep, cattle, humans and horses in LIV areas.

WNV in dogs is rare with few reported cases. Fever, apathy, anorexia, and progressive neurological signs have been described.

Diagnosis

Diagnosis of **TBEV** is based upon clinical evidence, and on exposure to *I. ricinus* ticks in endemic regions. A rise in specific antibody titres in paired sera, or specific antibodies in the CSF, may confirm a diagnosis. Viraemia in TBEV is usually short-lived and not present at the time of clinical presentation. In viral CNS infections, mononuclear pleocytosis is found in the CSF of infected dogs.

In **LIV** infection, an increase in serum antibody titres can be measured by the haemagglutination inhibition test.

Immunohistochemistry, virus isolation, RT-PCR and serology are used to detect infection with **WNV**.

Treatment

Clinically-apparent **TBEV** infections are treated with non-steroidal anti-inflammatory drugs (NSAIDs) along with adequate supportive therapy, including rehydration. Treatment with glucocorticoids is controversial.

[1] See ESCCAP Guideline 03: Control of Ectoparasites in Dogs and Cats.
[2] See www.ecdc.eu
[3] See ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

main hosts

PRIMARY TRANSMISSION CYCLE OF WNV

dead-end hosts

vector
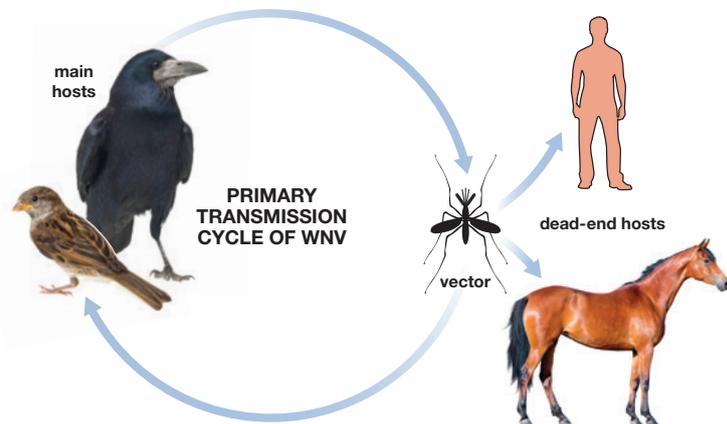
Control

Safe and effective **TBEV** vaccines are available for humans at risk of exposure but no vaccines are available for dogs. Effective protection against ticks is the primary control measure.

Animals that survive a **LIV** infection and eliminate the virus by an effective humoral immune response remain seropositive and the protection is probably lifelong.

Avoiding mosquito bites by the use of repellents is the most important way to prevent **WNV** infection. Vaccines are available for horses at risk and an experimental vaccine for dogs and cats is being evaluated[1].

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