Lyme Disease and Other Tickborne Diseases
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Lyme disease
Lyme disease is an emerging bacterial disease in many regions that is spread by the black-legged ticks *Ixodes scapularis* (central and eastern North America), *Ixodes pacificus* (west coast) and *Ixodes ricinus* (Europe). The main causative agent, *Borrelia burgdorferi*, is a Gram negative, fastidious spirochete that is maintained in a sylvatic cycle involving ticks, birds, small mammals and large mammals (Figure 1). Both infected nymphs and adult ticks can transmit *B. burgdorferi*. The relative role of each is unclear; however, nymphs are the main source of human exposure, probably in large part because their small size makes them harder to detect and more likely to be attached long enough to infect the host.

Figure 1: *Ixodes scapularis* and *Borrelia burgdorferi* life cycle

There is a wide range of other *Borrelia* species, along with various subspecies of *B. burgdorferi*. To date, only *B. burgdorferi* has been implicated as a cause of disease in dogs.
After an infected tick attaches to a host, the bacterium alters its gene expression, repressing outer surface protein A (OspA), a protein that helps it attach to the tick’s midgut, and increasing expression of OspC, allowing it to migrate into the host. This requires time, and typically 48-72h, if not more.

Most dogs that are exposed to *B. burgdorferi* never develop disease. Many will mount a detectable immune response, which accounts for the baseline seroprevalence that can be found in healthy dogs in endemic regions. The percentage of exposed dogs that develop Lyme disease is unknown, and evaluation of that is complicated by problems definitively diagnosing disease. Cats seem to be refractory to disease, although seroconversion can occur.

In dogs, Lyme arthritis is the most commonly recognized clinical presentation, with typically mild disease that is transient or rapidly responsive to antimicrobials. This is characterized by lameness involving one or more joints, with potential shifting lameness. Fever, lethargy and mild lymphadenopathy may also be present. Lyme nephritis, glomerulitis associated with immune complex deposition, is the other main recognized manifestation and while rare, it can be serious.

Diagnosis of Lyme disease can be challenging and involves a combination of identification of a risk of exposure, detection of antibodies against *B. burgdorferi*, clinical presentation consistent with Lyme disease and exclusion of other likely causes. Detection of the organism by validated PCR or culture, especially from synovial fluid in a dog with synovitis, is strongly supportive but is uncommonly available.

A few different serological tests are available, with most providing a positive/negative indication. A quantitative C6 antibody assay is also available, as is a multiplex ELISA targeting OspA, OspC and OspF. The value of quantitative assays is unclear as there is no evidence that there is a correlation between magnitude of titre and disease. Routine tests targeting C6, VLS E or OspF indicate natural exposure as these are not components of vaccines. OspC and OspA can be false positive results from vaccines, depending on their composition.

While experimental Lyme arthritis in dogs has been self-limiting, antimicrobials are typically used to hopefully speed the course of recovery. Doxycycline (10 mg/kg q24h or 5 mg/kg q12h) is the main drug, although amoxicillin may be used in cases where doxycycline is not an option. Cefovecin can also be effective but is best reserved for cases where doxycycline cannot be used.

Response is rapid and if there is no clinical response within 1-3 days, the diagnosis should be reconsidered. Adjunctive therapy, such as NSAIDs or other forms of pain relief, is often indicated. Treatment options for Lyme nephritis are poorly investigated and depend in large part on the severity of disease. Thorough evaluation is required to identify other causes (e.g. pyelonephritis, neoplasia, other immune mediated causes) and treatment typically involves antimicrobials, combined with measures targeting renal disease (e.g. ACE inhibitor or aldosterone-receptor blocker, antithrombotics, anti-hypertensives, dietary modification). Immunosuppression (e.g. mycophenolate) may be required, and is usually indicated in severe cases (e.g. significant proteinuria, hypoalbuminemia, severe progressive azotemia).
Treatment of healthy seropositive dogs continues to cause controversy. In endemic areas, the rate of seropositivity in the healthy dog population can be high, while the incidence of overt Lyme disease is unknown. There is currently no evidence that seropositivity is a predictor for development of Lyme disease, including Lyme nephritis. Screening of positive dogs for proteinuria has been recommended, to allow for investigation of Lyme nephritis (or other renal diseases), and this is reasonable but low yield. Treatment of seropositive, healthy dogs is hard to justify. Evaluation of C6 titres does not impact decisions for healthy dogs.

Prevention of Lyme disease involves tick control and vaccination. Tick control is the most important aspect, since prevention of ticks from attaching (or attaching long enough to transmit *B. burgdorferi*) will prevent disease. Vaccination is best positioned as a secondary level of protection for situations where tick control is ineffective. Vaccination should not be used in lieu of tick control, but as an adjunct method to provide extra protection. Tick control includes behavioural modifications (e.g. avoiding high risk sites), environmental modifications (e.g. keeping lawns cut short, clearing of brush and debris), tick checks and tick removal, as well as tick preventives. Tick preventives, should be used during the risk period, which is anytime the temperature rises about ~4°C. Tick preventives that kill quickly or prevent attachment and feeding are preferable to those that require a longer time to work, mainly from the standpoint of potential transmission of other (currently uncommon) tickborne diseases that are transmitted much more quickly. From a Lyme disease standpoint, any product that prevents tick attachment or eliminates ticks before 24-36h should be effective.

Current vaccines target OspA, attempting to eliminate *B. burgdorferi* in the tick’s midgut before it can be transmitted. OspC may also be targeted, to eliminate transmitted bacteria. The relative efficacy of different vaccines is unknown. Since there is no (or limited) natural immunity against OspA from natural infection, vaccination of dogs that have previously been diagnosed with Lyme disease is reasonable.

**Other tickborne diseases**

A range of other tickborne diseases can be of concern, depending on the geographic region and the types of ticks that are present.

**Ehrlichiosis**

Canine ehrlichiosis is caused by a range of *Ehrlichia* species, mainly *E. canis*, *E. ewingii* and *E. chaffeensis*. The brown dog tick (*Rhipicephalus sanguineus*) is the vector for *E. canis*, while *Amblyomma americanum* (Lone Star tick) is the main vector for *E. chaffeensis* and *E. ewingii*. The acute phase is characterized by signs such as fever, depression, anorexia, weight loss, hepatomegaly, splenomegaly, ocular and nasal discharge and lymphadenopathy. Hematologic changes (thrombocytopenia, non-regenerative anemia and leukopenia) typically develop subsequently, within 10-20 days of initial infection. CNS disease may be evident in a subset of cases. Bone marrow suppression can occur with chronic disease.

Diagnosis is based on clinical signs, likelihood of exposure and antibodies against *Ehrlichia* spp. Most available tests cross-react between different *Ehrlichia*
species. Detection of *Ehrlichia* in blood by PCR is diagnostic but of relatively low sensitivity.

Doxycycline (5 mg/kg q12h or 10 mg/kg q24h) is the treatment of choice. Four weeks is usually recommended, although data are lacking. There is usually a rapid (24-48h) and dramatic clinical response. Poor response should lead to reconsideration of the diagnosis. Prevention should focus on tick prevention.

Anaplasmosis

Anaplasmosis is caused primarily by the tickborne bacterium *A. phagocytophilum*. This bacterium is transmitted by *Ixodes* ticks and the distribution of anaplasmosis mimics that of Lyme disease (which is much more common). After 24h of more of feeding, *A. phagocytophilum* migrates from the tick to the host, and probably most often causes no overt disease. In some individuals, clinical infection will develop, characterized by vague signs such as lethargy, inappetance and fever. Signs similar to Lyme disease may be observed, and co-infection with both is possible since their vectors are the same. Thrombocytopenia is the most common hematology abnormality. Morulae, accumulations of vacuole-bound bacteria, may be evidence cytologically. Diagnosis is based on clinical signs, tick exposure and detection of morulae and/or antibodies against *Anasplasma*. Doxycycline is the main treatment.
Introduction
Respiratory tract infections are not uncommon in dogs and cats, but can be difficult to manage for many reasons. A variety of different pathogens, including viruses and bacteria can be involved, sometimes in co-infections. The upper respiratory tract harbours a diverse bacterial microbiota and many (or most) potential causes of respiratory disease can be found in some healthy individuals. Definitive diagnosis of the presence of a bacterial infection can be difficult. Treatment options have not been well studied, including drug choices and duration of treatment. These factors mean that determining when and how to treat may be a challenge. There is also a wide spectrum of disease that may require markedly different approaches. Examples are provided below, along with a table of antimicrobial options (Table 1).

Feline upper respiratory tract disease (URTD)
This multifactorial syndrome can be caused by a range of bacterial and viral pathogens, and typically causes ocular and nasal discharge, epistaxis, sneezing and conjunctivitis. Disease may be acute or chronic, with chronic disease being a potential frustrating problem to manage.

Most cats with URTD have viral infections (FHV-1, calicivirus), but secondary bacterial infections can develop, particularly with staphylococci, streptococci, E. coli and Pasteurella. Chlamydia felis, Bordetella bronchiseptica and Mycoplasma may also be involved. Diagnosis is not often attempted because of the potential difficulty identifying the underlying cause and the limited impact a diagnosis has on treatment decisions.

Treatment recommendations
For acute disease, monitoring without antibiotics is recommended in most cases, particularly those that are acute and not severe. A 10 day observation period is reasonable if the cat has no evidence of fever, lethargy or anorexia accompanying the signs of URTD, signs that might indicate the progression to bacterial pneumonia.

When treatment is required, doxycycline is recommended for 7-10d as the first line choice. A moxicillin would be an acceptable choice if C. felis and Mycoplasma are not highly suspected.

If there is poor response to antimicrobials, further diagnostic testing is indicated, not simply selection of a new drug.

For chronic disease, diagnostic testing is required to identify underlying causes that might need to be addressed. First line options for acute disease still apply. Use of drugs such as 3rd generation cephalosporins, fluoroquinolones or azithromycin should be reserved for situations where first line options are not viable.

If disease is recurrent, underlying problems cannot be identified or addressed, and there is a history of response to treatment, use of the same drug to which there was a positive response the last time is reasonable.
Canine upper respiratory disease complex (CIDRC)

This common problem (often referred to as kennel cough) can be caused by many different viruses and bacteria. It is highly contagious and usually minimally virulent, and is most often self-limiting. Testing is not often performed because of limited impact on treatment in acute, sporadic cases. Therefore, empirical treatment options are usually required.

Treatment recommendations

Observation for 10 days without antibiotics is recommended as disease will usually resolve without antimicrobials. Antimicrobials should be recommended in dogs with fever, lethargy or inappetance plus mucopurulent nasal discharge. If there is no evidence of bacterial pneumonia, doxycycline for 7-10 days is recommended. Amoxicillin or amoxicillin/clavulanic acid are considerations if treatment with doxycycline fails or is not possible (e.g. not well tolerated), but is suboptimal if *B. bronchiseptica* is a likely cause.

Pneumonia

Various bacteria may be involved, with *B. bronchiseptica*, *Mycoplasma* spp., *Streptococcus equi zoopneumoniae* and *Streptococcus canis* being potential primary pathogens and *Escherichia coli*, *Pasteurella* spp., *Streptococcus* spp., *B. bronchiseptica*, *Enterococcus* spp., *Mycoplasma* spp., *S. pseudintermedius* and other coagulase-positive *Staphylococcus* spp., and *Pseudomonas* spp. being opportunistic agents. Treatment is ideally based on culture of lower airway specimens, but empirical treatment is needed while awaiting culture results.

Treatment recommendations

If infection is suspected to have been caused by *Bordetella* or *Mycoplasma* and there is no evidence of systemic disease (fever, dehydration, lethargy, respiratory distress), doxycycline is recommended.

If aspiration pneumonia is suspected, a parenteral beta-lactam (e.g. ampicillin, ampicillin/sulbactam, first generation cephalosporin such as cefazolin) may be adequate.

If evidence of sepsis is present, broader spectrum coverage, including good activity against gram negative bacteria, is indicated. This should consist of a drug with good gram positive activity (e.g. ampicillin, ampicillin/sulbactam, first generation cephalosporin such as cefazolin) plus a fluoroquinolone. Treatment can be refined based on culture results, when they become available.

Table 1: Selected antimicrobials for the treatment of respiratory infections in dogs and cats. From The International Society of Companion Animal Infectious Diseases guidelines (Lappin et al, in press).

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Amikacin</td>
<td>Dogs: 15 mg/kg</td>
<td>Not recommended for routine use but</td>
</tr>
<tr>
<td>Drug</td>
<td>Dosage</td>
<td>Details</td>
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<tr>
<td>IV/IM/SC, q24h</td>
<td>Cats: 10 mg/kg, IV/IM/SC, q24h</td>
<td>may be useful for treatment of multidrug resistant organisms or if parenteral enrofloxacin or ciprofloxacin are contraindicated. Potentially nephrotoxic. Avoid in dehydrated animals and those with renal insufficiency.</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>22 mg/kg, PO, q8-12h</td>
<td>May be useful for treatment of secondary bacterial URI caused by <em>Pasteurella</em> spp. and <em>Streptococcus</em> spp., some <em>Staphylococcus</em> spp. and many anaerobic bacteria. Ineffective against beta-lactamase producing bacteria, most <em>Bordetella bronchiseptica</em> isolates, all <em>Mycoplasma</em> spp., and <em>Chlamydia felis</em> in cats.</td>
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<tr>
<td>Amoxicillin-clavulanate</td>
<td>Dogs: 11 mg/kg, PO, q8-12h</td>
<td>Used as a first-line option for secondary bacterial URI from <em>Pasteurella</em> spp., <em>Streptococcus</em> spp., methicillin-susceptible <em>Staphylococcus</em> spp. (including penicillase-producing strains) and many anaerobic bacteria. Resistance is common in <em>B. bronchiseptica</em>. Ineffective against all <em>Mycoplasma</em> spp., and inferior to other drugs for <em>C. felis</em> in cats.</td>
</tr>
<tr>
<td>Amoxicillin-sulbactam</td>
<td>20 mg/kg, IV, IM, q6-8h</td>
<td>Used alone parenterally for cases with uncomplicated secondary bacterial pneumonia (gram-positive and anaerobic bacteria). Used concurrently with another drug with wider gram-negative activity if life-threatening disease exists.</td>
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<tr>
<td>Ampicillin sodium</td>
<td>22-30 mg/kg, IV, SQ, q8h</td>
<td>Used parenterally for cases with uncomplicated secondary bacterial pneumonia (gram-positive and anaerobic bacteria). Used concurrently with another drug with gram-negative activity if life-threatening disease exists.</td>
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<tr>
<td>Azithromycin</td>
<td>5-10 mg/kg, PO, q12h day 1 and then q3 days (Longer intervals are not indicated)</td>
<td>Used for primary bacterial diseases (in particular <em>Mycoplasma</em> spp.) and for pneumonia of undetermined etiology because the spectrum includes <em>Toxoplasma gondii</em> and <em>Neospora caninum</em>.</td>
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<tr>
<td>Cefazolin</td>
<td>25 mg/kg, SQ, IM, IV, q6h</td>
<td>Used parenterally for cases with uncomplicated secondary bacterial pneumonia (gram-positive and anaerobic bacteria). Used concurrently with another</td>
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<tr>
<td>Drug</td>
<td>Dosage/Route</td>
<td>Description</td>
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<tr>
<td>Drug with wider gram-negative activity if life-threatening disease exists. Ineffective against <em>B. bronchiseptica</em>, <em>Mycoplasma</em> spp., and <em>C. felis</em> in cats, and enterococci.</td>
<td></td>
<td></td>
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<tr>
<td>Cefovecin</td>
<td>8 mg/kg, SC, once. Can be repeated once after 7-14 days.</td>
<td>May be effective for treatment of secondary bacterial URI caused by <em>Pasteurella</em> spp., some <em>Staphylococcus pseudintermedius</em> and <em>Streptococcus</em> spp.. Ineffective for <em>B. bronchiseptica</em>, <em>Mycoplasma</em> spp., and <em>C. felis</em> in cats and <em>Enterococcus</em> spp.. Pharmacokinetic data are available to support the use in dogs and cats, with a duration of 14 days (dogs) and 21 days (cats).</td>
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<tr>
<td>Chloramphenicol</td>
<td>Dogs: 50 mg/kg, PO, q8h Cats: 50 mg/cat, PO q12h</td>
<td>Reserved for multidrug resistant infections with few other options. Effective for the primary bacterial pathogens, penetrates tissues well, and has an excellent spectrum against anaerobes and so may be considered for treatment of pneumonia when the owner cannot afford dual antimicrobial agent therapy. Myelosuppression can occur, particularly with long term therapy. Owners should be instructed to wear gloves when handling the drug because of rare idiosyncratic aplastic anemia in humans.</td>
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<tr>
<td>Clindamycin</td>
<td>Dogs: 10 mg/kg, PO, SC, q12h Cats: 10-15 mg/kg, PO, SC, q12h</td>
<td>Activity against most anaerobic bacteria, many gram-positive bacteria and some mycoplasmas. Not effective for most gram-negative bacteria and some <em>Bacterioides</em> spp..</td>
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<tr>
<td>Doxycycline</td>
<td>5 mg/kg, PO, q12h Or 10 mg/kg, PO, q24h</td>
<td>Used for dogs or cats with URI, CIRDC, or bronchitis that is likely to be associated with <em>B. bronchiseptica</em>, <em>Mycoplasma</em> spp., and <em>C. felis</em> (cats). An injectable formulation is available if parenteral administration is needed. Either the hyclate or monohydrate salts can be used. Can be used in kittens and puppies &gt; 4 weeks of age without teeth browning.</td>
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<tr>
<td>Fluoroquinolones</td>
<td>Various</td>
<td>Active against most isolates of <em>B. bronchiseptica</em>, <em>Mycoplasma</em> spp., and <em>C. felis</em> (cats) as well as many secondary</td>
</tr>
<tr>
<td>Drug</td>
<td>Doses and Administration</td>
<td>Notes</td>
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| Gentamicin                    | Dogs: 9-14 mg/kg, IV, q24h  
Cats: 5-8 mg/kg, IV, q24h                      | Not recommended for routine use but may be useful for treatment of multidrug resistant organisms or if parenteral enrofloxacin is contraindicated. Potentially nephrotoxic. Avoid in dehydrated animals and those with renal insufficiency. |
| Imipenem-cilastatin           | 3-10 mg/kg, IV, IM q8h                                                                  | Reserve for treatment of multidrug resistant infections, particularly those caused by Enterobacteriaceae or Pseudomonas aeruginosa. Recommend consultation with a respiratory or infectious disease veterinary specialist or veterinary pharmacologist prior to use. |
| Meropenem                     | Dogs: 8.5 mg/kg SC 12h  
 Or 24 mg/kg IV q12h  
Cats: 10 mg/kg q12h, SC, IM, IV       | Reserve for treatment of multidrug resistant infections, particularly those caused by Enterobacteriaceae or Pseudomonas aeruginosa. Recommend consultation with an infectious disease veterinary specialist or veterinary pharmacologist prior to use. |
| Minocycline                   | Dogs: 5 mg/kg, PO, q12h  
Cats: 8.8 mg/kg PO q24h or 50 mg/patient PO q24h | Similar to doxycycline and can be used for dogs or cats with URI, CIRDC, or bronchitis that is likely to be associated with B. bronchiseptica, Mycoplasma spp., and C. felis (cats). |
| Trimethoprim-sulfamethoxazole or trimethoprim-sulfadiazine | 15 mg/kg PO q12h  
Note: dosing is based on total trimethoprim + sulfadiazine | Generally avoided in respiratory tract infections that may involve anaerobic bacteria (particularly pyothorax). Ineffective for primary bacterial pathogens other than Streptococcus spp. |
| concentration | Concerns regarding adverse effects in some dogs, especially with prolonged therapy. If prolonged (>7 d) therapy is anticipated, baseline Schirmer's tear testing is recommended, with periodic re-evaluation and owner monitoring for ocular discharge. Avoid in dogs that may be sensitive to potential adverse effects such as KCS, hepatopathy, hypersensitivity and skin eruptions. |
Tickborne Diseases in Horses
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As ticks are increasingly expanding their ranges, the risk of tickborne disease increases correspondingly. Ticks (and pathogens that accompany them) are commonly found in some regions of Canada, yet diagnosis and management of tickborne disease remains somewhat enigmatic.

Lyme disease
Lyme disease is a bacterial infection caused by the spirochete Borrelia burgdorferi. This bacterium is transmitted by black legged ticks (Ixodes scapularis and I. pacificus), as part of a multi-year life cycle that includes birds, small mammals (e.g. mice) and large mammals (deer) as hosts. Horses are incidental hosts for these ticks but can be exposed to B. burgdorferi, with the potential for disease. Transmission requires attachment of an infected tick for a prolonged period of time, likely at least 48 hours, if not 72h or greater.

Lyme disease in horses
Lyme disease is poorly characterized in horses. After exposure to an infected tick, the bacterium can be found in skin near the tick bite, as well as in various tissues. However, experiment infection did not result in abnormalities that are commonly attributed to Lyme disease. Mild suppurative synovitis, perineuritis or meningitis were observed histologically, consistent with the implication of Lyme disease as a potential cause of lameness and neurological disease. Yet, the best (and probably only well) described clinical manifestation of Lyme disease is neuroborreliosis. Fever, shifting lameness and uveitis are commonly associated with Lyme disease clinically, but with limited evidence.

Prevalence and incidence
The incidence of Lyme disease is unknown, in large part because of the lack of good diagnostic criteria. It is also highly variable regionally, mimicking the distribution and density of ticks. Various prevalence studies have been performed in horses, with rates of up to 59% reported in highly endemic areas. The high seroprevalence and low apparent incidence of disease supports the hypothesis that disease, at least disease that is severe enough to be detected, is rare after exposure to an infected tick.

Clinical Signs
Neuroborreliosis: This can be manifested as a wide range of neurological abnormalities, typically without fever. Cranial nerve dysfunction and meningitis signs are most common.

Uveitis: Signs typical for uveitis of other origins are present. Neurological disease may be present concurrently.
Pseudocutaneous lymphoma: While rare, cutaneous lymphoma with *Borrelia* DNA in affected tissue has been identified. Whether this truly means *B. burgdorferi* is the cause or whether there can concurrent dormant presence of the bacterium in areas with lymphoma is unclear. However, this type of lesion has been found in experimentally infected ponies.

Other: The above described manifestations are the best proven types of Lyme disease in horses, yet Lyme disease is often associated anecdotally with stiffness, shifting lameness and vague performance or lameness problems. *B. burgdorferi*-associated synovitis has been identified and this type of disease is better proven in some other species (e.g. humans, dogs) so it is likely that Lyme disease truly does manifest as these vague disorders.

**Diagnosis**

Diagnosis is challenging, particularly diagnosis of cases with stiffness and lameness. Seropositivity along with clinical signs of Lyme disease is suggestive; however, in areas where the seroprevalence is high, the positive predictive value is probably low. Identification of spirochetes in affected areas (e.g. CSF, aqueous humour, synovial fluid) is supportive of a diagnosis of Lyme disease, although sensitivity is low. Diagnosis of Lyme-associated lameness and stiffness is challenging and can be suggestive, at best, when the affected horse is seropositive and other likely causes have been excluded. Negative results probably have a high negative predictive value in areas where the disease is endemic and when infection has not likely occurred within the last month, given the time that is typically required for antibodies to be detectable.

Two main serological tests are available. One is the use of an ELISA that detects the C6 protein, and which is marketed predominantly for use in dogs, alongside testing for *Amaplasma, Ehrlichia* and *Dirofilaria immitis* (SNAP 4DX Plus). A multiplex ELISA is available from Cornell University that detects three bacterial outer surface proteins, OspA (predominantly associated with vaccination), OspC (associated with acute infection) and OspF (associated with chronic infection). There is currently no consensus as to the best testing approach. The potential role of PCR and other antigen tests is unclear. A positive response to a treatment course is also suggestive, but far from definitive.

Testing of healthy horses is not indicated as there is no reason to act on a positive result.

**Treatment**

Objective information about treatment of Lyme disease in horses is lacking and anecdotal information is clouded by problems with diagnosis. Treatment is largely based on experiences from other species and the known susceptibility of *Borrelia* to tetracyclines and beta-lactams. Doxycycline is the most widely used drug in horses, typically at a dose of 10 mg/kg PO q12 for 28d. Minocycline is an alternative option, with a dose of 4 mg/kg PO q12h. Intravenous oxytetracycline can be used but the route of administration and availability of oral doxycycline and minocycline make intravenous treatment hard to justify. Similarly, while pencillin
and cephalosporins are likely effective, the route of administration and duration of treatment are problematic.

Systemic treatment is likely indicated for neuroborreliosis, with penicillin (44,000 IU/kg IV q4-6h) or cefotaxime (25-50 mg/kg IV q6-8h) being options

Prevention

Tick avoidance is the basis of Lyme disease prevention; however, it is challenging given the lack of approved tick control products for horses and difficulty modifying environments and behaviours (e.g. trail rides) that foster exposure. Regular tick checks should be performed on horses with outdoor access in endemic areas, with ticks carefully and promptly removed. “Tickscaping” involves making an environment less amenable to ticks, such as through improving light exposure and drainage, removing debris and mowing, although the overall impact of these is probably limited. Various compounds can be used as tick repellents, although licensed products are lacking in horses. Vaccines are available for dogs and these are anecdotally used in horses; however, efficacy and safety are not known.

Anaplasmosis

While rare, anaplasmosis is a potential concern in areas where Lyme disease is present. This disease is caused by *Anaplasma phagocytophilum*, a bacterium that is also transmitted by the black legged tick. Tick infection rates tend to be much lower compared to *B. burgdorferi* and the incidence of anaplasmosis is dwarfed by Lyme disease in endemic regions; however, it is still a relevant concern.

Clinical signs of anaplasmosis may be vague, consisting initially of fever and varying degrees of depression and inappetance. Icterus is a common initial complaint, and other signs such as ventral edema and ataxia may also be noted.

Decreases in white blood cell, red blood cell and platelet counts may be noted, and sometimes morulae (inclusion bodies) can be seen within white blood cells. This finding, in an endemic region, is strongly supportive of anaplasmosis, but cytology has low sensitivity, especially after the first few days of disease. PCR can be used to detect the bacterium in blood. Serological testing can be performed using a canine multiplex ELISA. While this just indicates exposure, the presence of antibodies against *Anaplasma* in a horse with consistent clinical signs is supportive of a diagnosis.

Tetracyclines are the main treatment options, with intravenous oxytetracycline (7 mg/kg IV q24h for 3-8d) used most often. The short duration of treatment makes intravenous administration more feasible, compared to Lyme disease, although oral doxycycline or minocycline are options.
Equine herpesvirus
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The equine herpesvirus group contains 9 known viruses (EHV1-9) that vary greatly in their epidemiology and clinical relevance. Most attention is paid to EHV-1 because of the potential for sporadic and epidemic abortion and neurological disease, but other types have clinical relevance.

**EHV 1**

EHV1 is the most commonly recognized cause of EHV-associated disease. It is endemic in the horse population, lying dormant in ganglia or lymphocytes of a large percentage of healthy horses. It continually circulates in the horse population internationally through transmission of actively infected horses, as well as periodic reactivation of latent infection.

EHV1 infection is associated with various clinical problems, including mild systemic disease (e.g. fever), respiratory disease, neurological disease (equine herpesvirus myeloencephalopathy (EHM)), abortion and neonatal infection.

Abortion and EHM are the most important problems, and outbreaks can occur. Abortion usually occurs in the last 4 months of gestation and whether this is from new exposure or reactivation of latent infection is hard to discern.

EHM is a potentially devastating neurological disease that can be associated with outbreaks. It is a result of vasculitis and thrombosis in the CNS. Much attention has been paid to ‘neuropathogenic’ strains that have a mutation that is detectable by PCR. An association between this mutation and EHM has been made, yet it is far from absolute and wild-type (non-neuropathogenic) strains can cause neurological disease.

Diagnosis is usually based on PCR of nasal swabs or blood (buffy coat). Detection of the neuropathogenic mutation can be performed, although the clinical relevance of this is limited since those results do not impact management of a case or farm. Serological testing can also be used. Detection of a 4 fold increase in samples collected 10-28 days apart is diagnostic for EHV infection, although it does not differentiate between EHV1 and EHV4.

A variety of antiviral drugs can be tried, although efficacy is unclear, in part because active viral infection may not be the main problem by the time clinical signs are identified. Antiviral options include acyclovir, valacyclovir, famcyclovir, ganciclovir and cidovir.

Vaccination is a key component for prevention of EHV1 abortion. Whether it helps against EHM is unclear.

**EHV 2**

This EHV type can be commonly found in horses. Conjunctivitis, epiphora and keratopathy have been reported associated with EHV-2 infection, particularly in
weanlings, but the role of this type in disease is not well understood. This virus is likely of very limited consequence to the horse population.

**EHV 3**

EHV 3 is the cause of coital exanthema, a disease characterized by vesicular lesions on the penis of males and the perineum and vulva of females. Disease is self-limiting and rarely causes severe or long term problems. It is not associated with abortion.

Transmission can be through direct contact (breeding or nose-nose), as well as through contaminated fomites. Both natural breeding and artificial insemination pose a risk since EHV 3 can be passed in semen.

Control of EHV 3 mainly revolves around general infection control and hygiene practices meant to reduce the risk of EHV 1 transmission, as well as breeding practices to prevent breeding-associated infections.

**EHV 4**

Closely related to EHV1, EHV4 is a common cause of mild respiratory disease in young horses. It is transmitted through direct horse-horse contact and indirect contact with nasal secretions. Most horses are infected as weanlings or yearlings, developing mild, self-limiting infections.

**EHV-5**

This type is associated with equine pulmonary multinodular fibrosis (EMPF), a potentially severe disease of adult horses. While EHV-5 is widespread in the equine population, understanding of the epidemiology and pathophysiology of EHV-5 infection is poor. Disease is characterized by vague and often insidious initial signs of low-grade fever, weight loss and progressive exercise intolerance. Radiographically, nodular interstitial fibrosis is evident. EHV-5 can be isolated from the lungs of infected horses, via lung biopsy or BAL, and disease is typically progressive, poorly responsive to treatment or ultimately debilitating. Treatments often include corticosteroids and antiviral drugs (e.g. acyclovir, valacyclovir), although little is known about specific drug efficacies.