Selected Small Animal Infectious Diseases

The purpose of these very brief description of a few of the more important vaccinatable companion animal infectious diseases is by no means to provide a detailed description of the disease. Rather it is an attempt to present some of the more important facts on a few of the diseases for which we as companion animal practitioners vaccinate regularly.

Canine Distemper Virus

- CDV is a member of the *Morbillivirus* genus, family *Paramyxoviridae*.
- It is an enveloped RNA virus.
- It infects a very wide range of natural hosts – including many free ranigig wildlife species.

- It is shed within 7 days of infection.
- It is most abundant in the secretions of the respiratory tract.
- Trans-placental infection can occur from infected dams.
- Virus may be excreted for up to 90 days following infection although shorter periods are more typical.
- The infection rate is significantly higher than the disease rate with 25-75% of infections being subclinical.

Pathogenesis is best understood by examining the chart below:

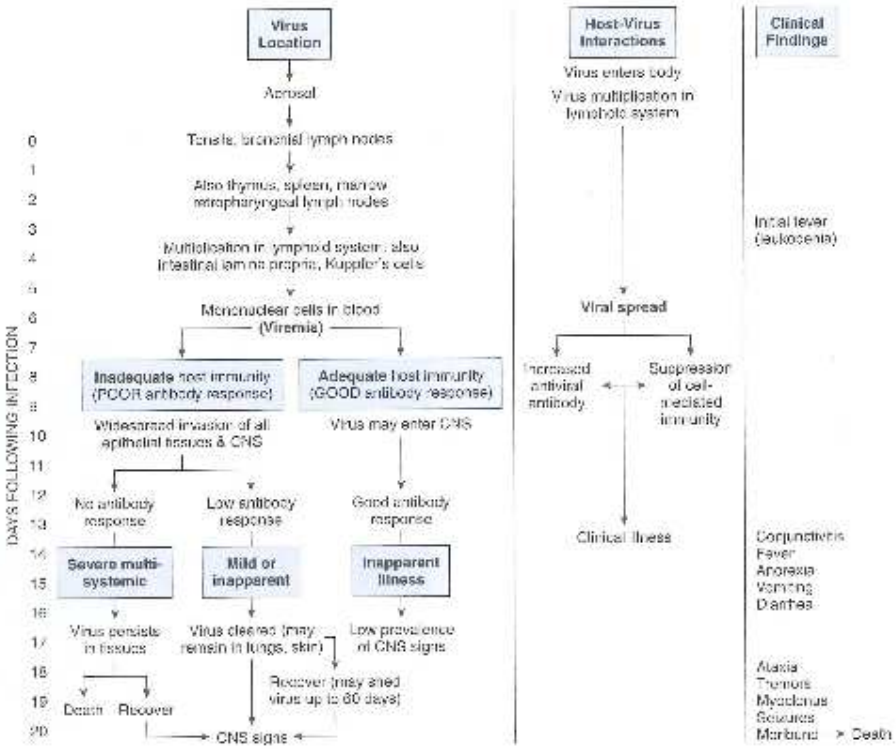

Figure: Sequential pathogenesis of canine distemper

Clinical signs - may vary but include:

Systemic signs: Non-specific malaise, pyrexia, respiratory disease and gastrointestinal disease.

Skin signs:

- Vesicular and pustular dermatitis and naso-digital hyperkeratosis.
- Some evidence exists that CDV may trigger juvenile pyoderma.

Neurological signs:

- These usually develop between 3 and 4 weeks following infection.
- These signs may occur many months after systemic sings have cleared.
- Systemic signs may have been very mild and missed.
- Dogs that never develop systemic signs and only present with neurological disease may well be partially immune due to vaccination.
- Neurological signs are extremely variable and depend on the localization of infection.
- Myoclonus and so called “chewing gum fits” are not pathognomonic but are highly suggestive.
- Transplacental infection can cause neurological disease in puppies under 2 months old.

Neonatal infections:

- These may cause enamel hypoplasia,
- neurological disease and cardiomyopathy.

Bone lesions: Hypertrophic osteopathy has been attributed to CDV infection.
Ocular disease: Mild anterior uveitis and optic neuritis have been associated with CDV.

Combined infections
Immunosuppression : May result in opportunistic infection with *Samonella, Toxplasma, Neospora* and *Pneumocystis.*

Diagnosis

- Clinical signs are non-specific.
- Viral inclusions may be seen on blood smear or in cells of the CSF on occasion in acute disease.
- Immunohistochemisry to detect viral inclusions in the biopsies of the nasal mucosa, foot pads and haired skin of the neck may be useful.
- Dogs shedding virus may well test positive on Snap Tests that detect antigen in ocular or respiratory discharges or urine.
- Antimortal diagnosis may be difficult in the chronic neurological disease.
- In this case CSF titer determination is very helpful. The CSF should never show a positive CDV titer. CSF's titers may be caused by blood contamination of the CSF sample and for this reason it is helpful to measure CPV titers in the CSF at the same time. CPV antibodies can only appear in the CSF by blood contamination.
- Blood IgM titers can be positive for up to 3 months following infection and up to 3 weeks following vaccination.
- Blood IgG titers only prove previous exposure (to vaccine or wild virus) but are not diagnostic for current infection.

Treatment is supportive.

Prevention is by vaccination.

Canine Infectious Tracheobronchitis (ITB)

A common, usually self-limiting cause of upper respiratory disease that is commonly caused by multiple infectious agents. Canine parainfluenza virus and *Bordetella bronchiseptica* are the most commonly isolated organisms. Puppies may be susceptible to *B. bronchiseptica* pneumonia.

- The most common viral aetiology is canine parainfluenza virus (CPiV-2).
- Canine adenovirus -2 (CAV-2) is also implicated and CDV may also play a role
- *B. bronchiseptica* is the principle bacterium involved in ITB.

Disease outbreaks occur usually in dense husbandry environments where aerosol is the means of transmission. Cats are also known to become infected with *B. bronchiseptica* (sometimes in association with their upper respiratory viral infections). Older asymptomatic carrier animals often act as a reservoir of infection for young naïve animals. Whereas the viruses shed for up to 2 weeks post infection, *B. bronchiseptica* may be shed for 3 months or longer.

Clinical findings

- **A**re typical as for "kennel cough" and typically develop 3-10 days following exposure to infection.
- Young puppies with no vaccination may develop life-threatening pneumonia.

Diagnosis is typically based on clinical presentation and history.
Additional testing such as chest radiography, TTA or BAL is usually not indicated unless pneumonia is suspected.
Viral isolation is possible but seldom performed.

Treatment - Refer disucssion with your veterinarian

Prevention in puppies

- Is usually achieved through MDA titers.
- MDA does not appear to interfere with vaccination in CPiV at 6 weeks. This is however not the case for CAV-2. Where MDA's may interfere for as long as 12-16 weeks.
- The duration of natural immunity following infection has not been published although one unpublished study demonstrated CPiV Ab's for up to 2 years following infection.
- Dogs naturally infected with *B. bronchiseptica* have been shown to be highly resistant to reinfection for at least 6 months.
- Vaccinations are available against most pathogens involved in this syndrome (*B. brochiseptica,* CPiV, CAV-2) for both parenteral and IN use.
- The viral antigens are usually incorporated in routine vaccination protocols.
- *Bordetella* bacterins are not included in multivalent vaccines.
- *Bordetella* bacterins have been thoroughly evaluated and have shown significant evidence in reducing the severity of signs. Sequential use of the parenteral and then the IN preparation have also been shown to enhance protection.
- In environment with high levels of exposure it has been recommended to use at least 2 parenteral vaccinations followed by and IN dose.
- Adult dogs exposed to risk should be vaccinated annually and boosted IN before an anticipated risk period. The parenteral vaccine seldom causes local injection site reactions. In vaccination can however result in transient upper respiratory tract reactions for 2-5 days.

Canine Viral Enteritis

The most important agent involved in canine **parvovirus 1 and 2**.
Canine caronavirus and **canine rotavirus** are also involved but at much lower levels.

Canine Parvoviral Enteritis

- This is a non-encapsulated DNA virus that requires rapidly dividing host cells for replication.
- This virus is very resistant to environmental degradation.
- CPV-2 has been shown to persist in the environment for up to 5 months.
- It is the most common cause of canine viral diarrhoea and carries a high morbidity and mortality in dogs under 6 months of age.
- Infection does not necessarily translate into clinical disease.
- When disease does occur it is worse in young rapidly growing pups that are co-infected with helminths, protozoa, and certain enteric bacteria.
- It is highly infectious and transmission is through contact with contaminated faeces or environment.
- Incubation can be as brief as 4-6 days.

Clinical signs - *are most commonly predominantly gastrointestinal.*

- Vomiting, diarrhoea (often very haemorrhagic) with collapse are typical.
- Profound leukopaenia is common and prognostic.
- Rapidly progessive panhypoproteinaemia is due to gut loss.
- Anaemia developes due to blood loss and bone marrow necrosis.
- Complications may involve neuroglycopaenia and secondary infections due to immunosuppression (catheter tip sepsis, gram negative septicaemia, polyarthritis)
- Acute myocarditis is no longer common (seen following in-utero infections and in infections of puppies under 8 weeks of age).

Diagnosis is frequently circumstantial.

Rapid snap tests are in frequent use. These tests are positive only as long as virus is shed in faeces (a period of around 5-7 days from onset of sings).
False positive tests can be seen for 5-12 days following the use of MLV vaccines.
Faecal electronmicroscopy is sensitive and specific but not widely available.

Treatment is supportive and should always involve the use of intravenous fluids to replace lost body water and electrolytes (especially K^+). See us or discuss this further with your veterinarian as the therapy is rigorous and intensively in it application.

Prevention

- Puppies recovering from infection are solidly immune, possibly for life.
- The most important means of protection is vaccination.
- MLV CPV-2 vaccines are in wide use.
- Serum Ab titers begin rising within 3 days of vaccination and rise rapidly to protective levels.
- The most important cause of so called vaccine failure remains MDA and consequent failure to sero-convert following vaccination.
- Unfortunately there is a window of between 2-3 weeks during which time MDA titers are high enough to neutralize an attenuated live CPV-2 vaccine but not high enough to protect against wild viral infection.

Poor hygiene & husbandry remain common causes of infection & outbreaks of disease.

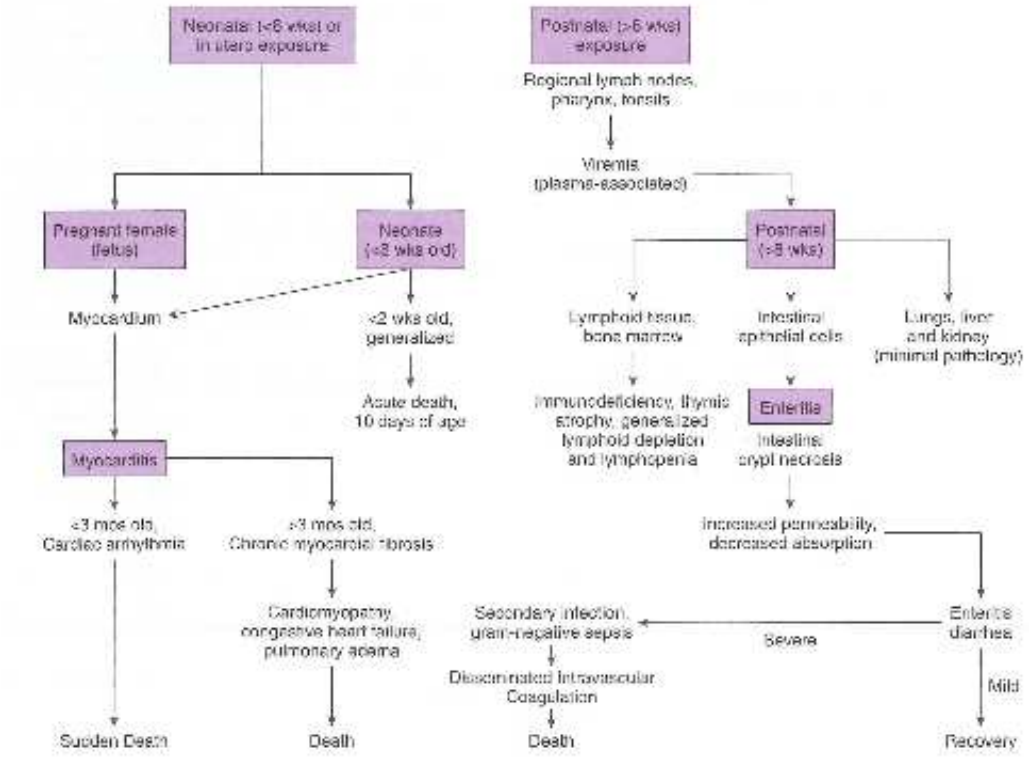

Figure: Sequential pathogenesis of canine parvovirus infection

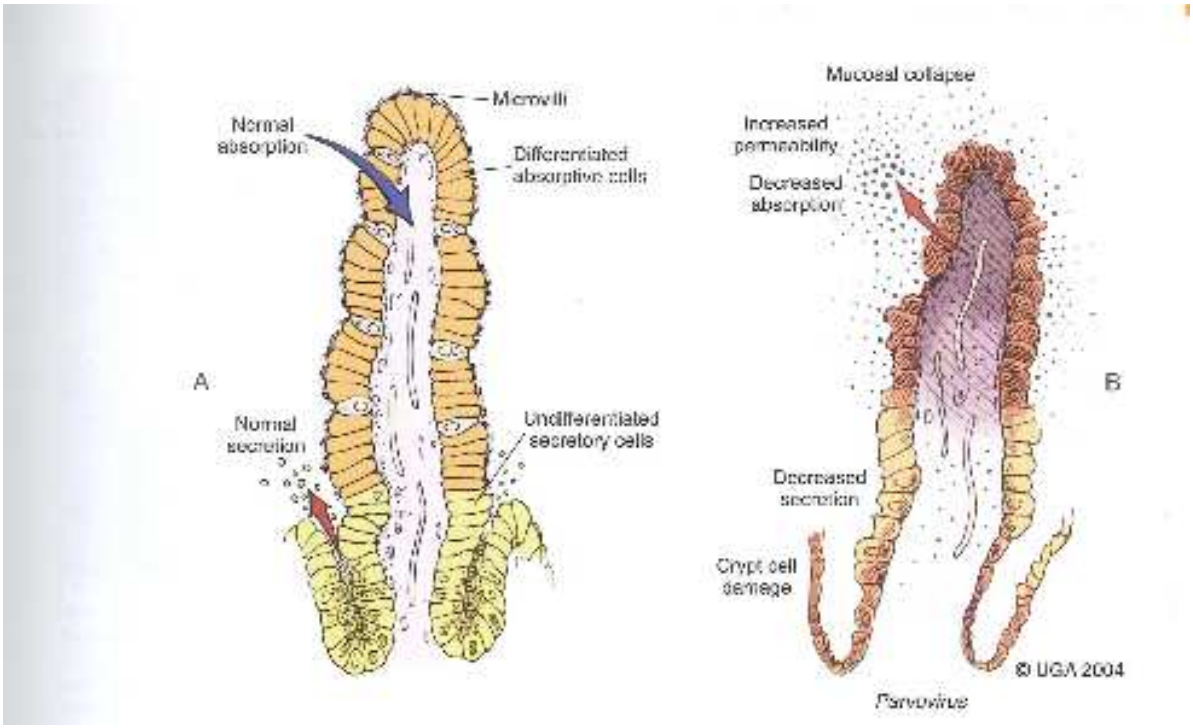

Figure: A. Normal intestinal villus showing cellular differentiation along the villus. B. Parvovirus infected villus showing necrosis of the villus.

Canine Caronaviral Enteritis

- Caronaviruses are fairly resistant and can persist in a contaminated environment for short periods (40-60 hours depending on moisture and temperature).
- The true importance of this virus in canine enteritis is not known; it does however appear to be of minimal importance.
- **CCV and CPV can co-exist and CCV may enhance the severity of CPV infection.**

Clinical signs seem to be less severe than those caused by CPV.

- In contrast to CPV (which usually only affects dogs under a year of age), CCV is able to infect and probably cause disease in dogs of any age.
- Vomiting and diarrhoea are common but haemorrhagic diarrhoea is not a feature.
- Fever and leukopaenia are not clinical features.
- Spontaneous recovery within 8-10 days is common.

Diagnosis without electronmicroscopy is difficult.

Treatment is supportive.

Prevention can be provided by vaccination.

- Protection is not complete in that virus replication is not completely prevented in a vaccinated host.
- Assessing the role of vaccination against this disease is difficult as the disease is usually self-limiting and mild.

Infectious Canine Hepatitis (ICH)

- ICH is caused by canine adenovirus 1. (CAV-1).This is antigenically distinct from CAV-2 which causes upper respiratory tract disease in dogs.
- It is a resistant virus and persists in the environment for months.
- Virus is excreted in the urine for 6 – 9 months following renal infection.

Pathogenesis is best understood by examining the chart below:

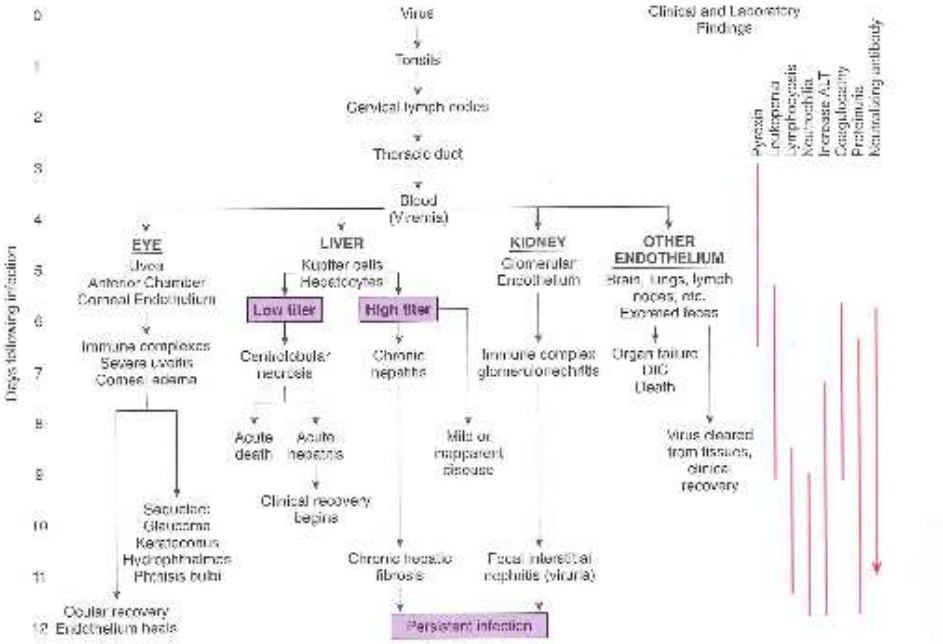

Figure: Sequential pathogenesis of infectious canine hepatitis. Solid vertical bars on the right correspond to chronologic occurrence and duration of respective clinical or laboratory findings.

Clinical findings

- The disease is most commonly seen in dogs under a year of age but can occur at any age in unvaccinated dogs.
- Death is commonly peracute leading to the suspicion of a toxin.
- The acute stage is characterised by pyrexia, abdominal pain, vomiting and bloody diarrhoea. Pneumonia may be present and upper respiratory signs are often found.
- Hepatomegaly may be palpable
- Subcutaneous oedema may be seen.
- A haemorrhagic diathesis is common with widespread bleeding (petechial and ecchymotic). Haemorrhagic body cavity effusions may be present.
- Icterus may occur in dogs that survive the acute phase.
- Uncomplicated disease passes in 5-7 days.
- Corneal oedema and anterior uveitis may occur as recovery begins and ma be the only signs seen in some dogs.

Diagnosis is usually based on clinical findings, laboratory evidence of an acute liver insult, a consumption coagulopathy and proteinuria.
Definitive diagnosis is seldom pursued but virus isolation, immunoflouresence and serology may be helpful.
CAV-1 viral inclusions may be seen within nuclei on FNA of the liver.

Treatment is symptomatic and supportive.
This would include management of acute hepatic failure and support for the coagulopathy.

Prevention *in the pup will depend on the dams anti-CAV-1 Ab titer.*

- The disease is rare today because of widespread use of vaccines.
- Vaccination is still regarded as core because the virus is environmentally resistant and wildlife hosts act as reservoirs.
- The most commonly used vaccine against this disease is a CAV-2 MLV which provides years of protection.

Feline Immunodeficiency Virus

Epidemiological

- Studies have shown that this virus has a **worldwide** distribution.
- Seroprevalence of the disease varies between a low of around 4% of the cat population to highest incidence in small pockets reaching close to 50%.
- Males are over represented due to their biting and fighting habits.
- Adults have higher rates of infection.
- The earliest date of infection that can be proven is 1966.
- It is a disease that affects a wide range of wild felids.
- Transmission is primarily through saliva and blood transfer and hence the role of fighting in male cats.
- In-utero infection and infection through milk have been shown experimentally.
- Semen has been shown experimentally at least to transmit virus.
- Horizontal transmission between cats in a multiple cat household is probably rare.

Pathogenesis:

- Clinical findings progress through several stages.
- Recognized stages include the acute stage, a clinically asymptomatic phase of variable duration and a terminal phase of infection referred to as feline acquired immunodeficiency syndrome (AIDS).
- Clinical signs are non-specific.
- The acute phase clinical signs may go undetected. Some will exhibit fever, malaise, acute enteritis, stomatitis, dermatitis, conjunctivitis, and respiratory tract infection.
- This phase may last a few days to a few weeks.
- After this they enter a clinically silent phase that may last years.
- The AIDS phase is characterized by neoplasia, opportunistic infections, myelosuppression, and neurological disease.
- Numerous opportunistic organisms have been described as complicating infections in FIV infected cats but despite this, little work has been done to compare the incidence of these infections in FIV negative cats. It may be that there is little difference between the rate of infection with what has been described as opportunistic organisms and the same organisms in FIV negative cats is not different. It has been shown that *Cryptococcus neoformans* and *Cryptosporidium* is no more common in FIV positive cats.
- Statistically FIV positive cats are more likely to develop lymphoma and leukaemia. Most lymphomas are B-cell.
- It has been postulated that the reduce CMI in FIV results in ineffective tumour surveillance by the immune system.
- It is also possible that the FIV virus has a direct oncogenic effect through an oncogene.

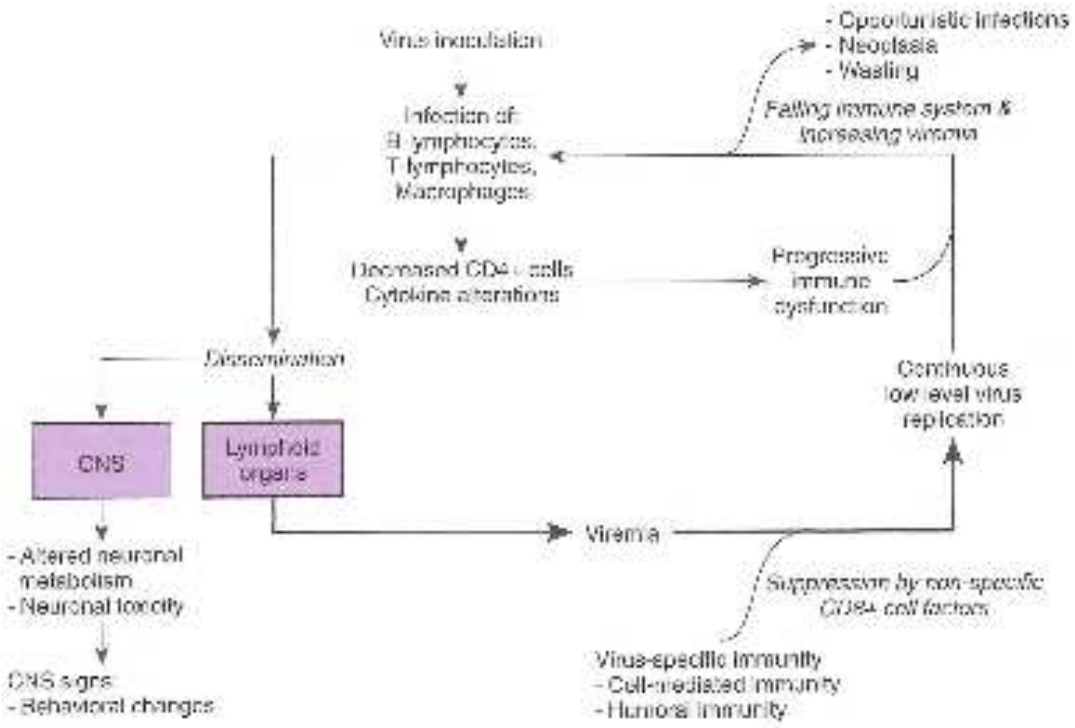

Figure: Pathogenesis of feline immunodeficiency virus infection.

Diagnosis

- Depends on the serological detection of FIV antibodies.
- In house tests are reasonably sensitive and specific.
- Infected cats develop high viral loads and subsequent high Ab titers and thus traditionally a positive Ab titer has been interpreted as synonymous with infection.
- This has been complicated by the introduction of a vaccine.
- <u>It is impossible to distinguish between a vaccine induced Ab titer and an infection induced Ab titer.</u>
- Unless one is certain that a cat has not been vaccinated, care should be taken in interpreting a positive test.

- Kittens born to vaccinated queens will also test positive without having the infection and this may lead inadvertent and mistaken euthanasia of a healthy animal.
- Antibody tests must also be interpreted with care in kitten less than months of age.
- Passive Ab transfer from mother to kitten from an infected or vaccinated queen can occur.
- Kittens very rarely become infected with FIV from their mothers.
- Most kittens testing positive will eventually test negative once MDA titers wane.
- A kitten that tests positive should be retested in 6 months time.
- A second positive test indicates active FIV infection.
- A kitten from an unknown background may initially test negative if it has not had sufficient time to develop and Ab titer. These kittens should be retested 2-3 months later to confirm an initial negative result.
- Cats in the acute phase of the disease may test negative and if reasonable suspicion of infection exists, a retest in 8-12 weeks should be performed.
- Most cats in the asymptomatic phase of the disease will test positive.
- Detectable Ab titers may disappear in terminal disease as the immune system collapses.

Guidelines for testing for FIV has been published:

Guidelines for FIV Testing in Cats
The following circumstances warrant testing: • All sick cats • All cats to be adopted • All cats with an unknown status • All cats that have risk factors for recent exposure (retesting may be indicated to give time for cats to sero-convert) • Bite or fight wounds • Cats that live largely outdoors • A negative cat living with a positive cat should be tested yearly • All cats vaccinated for FIV should be tested negative before vaccination

Treatment

- Naturally infected cats may live out a full life and eventually die of FIV unrelated causes.
- FIV itself is a rare direct cause of clinical disease..... However this statement needs to be interpreted in the light of secondary infecting agents ► see below....

Antiviral chemotherapy

Most antivirals used to treat FIV have been developed as treatments for HIV. Few well designed controlled studies have evaluated the various treatment options have been performed.
The nucleoside analogue zidovudine (AZT) alone or in combination with phosphonylmethoxyethylademine (PEMA) have been used.
AZT prevents virus from infecting new cells but does nothing for already infected cells.
AZT thus reduces viral load, improves immunocompetence and improves quality of life, prolonging life.
AZT does improve neurological function in cats with FIV induced neurological disease.
Resistance in the virus to the drug has been demonstrated.
Continuous haematological monitoring (weekly for the first month then monthly) during the use of AZT is important as it can cause anaemia.
If Hct drops below 20% treatment should be stopped.
If myelosuppression is present, the drug should not be used.
Doses should be reduced if renal failure is present.

Human recombinant IFNα has been used.
High dose parenteral dose regimes are most effective but can only use for 6-7 weeks before antibodies to the cytokine develop.
Oral low dose usage stimulates local lymphoid tissues (it is not absorbed systemically). No placebo controlled studies have evaluated this use in FIV.

General Management of FIV Infected Cats

- It is best if all cats status is known because this affects long term management.

- **The most effective strategy for prolonging life is to restrict the cat to indoor life as this prevents exposure to secondary infecting agents.**

- **At least yearly health checks are recommended to detect early changes in health status.**
- Any infections (bacterial, protozoal or fungal) should be treated aggressively and early.
- Itraconazole is an antifungal effective against systemic mycoses (like Cryptococcus) and dermatophytosis. Griseofulvin must be avoided because of its potential bone marrow suppressive effects.
- Treatment of FIV associated stomatitis is challenging. Repeated dentals and antibiotics may be effective in the short term. Glucocorticoids should be avoided. AZT has shown benefit. Complete dental extraction has also been an effective strategy.
- Underlying causes of anaemia must be sought but where they are not found, rhEPO should be considered.
- Rh-GM-CSF may also be considered in leukopaenic cats.
- Glucocorticoids may be beneficial in cases where neurological function is impaired.
- Intact males should be castrated.

Opinions regarding general vaccination of FIV infected cats differ.

It has been shown that infected cats can mount an immune response to injected antigens although it may be blunted – especially in the terminal stage of the disease.
As far as possible **inactivated vaccines** should be used. Live FPV vaccination has been reported to result in disease.
Lymphocyte stimulation (as occurs with vaccination) has been shown to stimulate an increased viral load and thus vaccination may be seen as a trade off between vaccine- induced protection and potentiation of an immunosuppressive state: **It is for this reason that it is suggested that FIV infected cats be kept indoors as far as possible.**
One should use only core vaccines (panleukopaenia and upper respiratory infection aetiologies) and inactivated products should be used as far as possible.
The duration of immunity following vaccination is unknown.

Prevention

- A controversial vaccine is available.
- **The biggest controversy has been caused by the way the vaccine induces FIV antibodies which then make it impossible to use traditional FIV test methods to diagnose the disease.**
- **Five strains of FIV exist and the vaccine will only protect against a homologous strain challenge.**
- **The available vaccine contains subtypes A and D with an adjuvant.**
- Some concerns also exist around the possibility of vaccination actually enhancing disease through increasing virus load.

Feline Leukaemia Virus Infection

This infection was first described in 1964 when virus particles were seen budding from the surface of lymphoblast cells in a cat with lymphoma.
The tumour could be transmitted to a naïve cat with the transmission of the virus.
The virus occurs globally and has historically been blamed for the majority of the disease burden in domestic cats.
These perceptions of the infection are gradually changing as the incidence of FeLV decrease.

The virus is a single stranded RNA enveloped retrovirus.
The ability of the virus to include itself in the hosts DNA (DNA copies of the viral RNA are made and randomly inserted into the hosts genome) is the reason infection persists life long following bone marrow infection. To cure a cat, every infected cell would need to be recognised and killed.

Epidemiologically the virus is really only important in domestic cats. Wild felid infections are rare.
There is evidence that the rate of infection is steadily dropping.
Whereas around 75% of all cats with lymphoma were FeLV positive decades ago, this is no longer true.
This is attributable to the testing and removal strategies that have been adopted as well as vaccination.
Outdoor cats have a higher incidence than indoor cats.
Whereas FIV infection rates are highest in fighting males, FeLV is highest in social cats and this reflects the mode of transmission.

Transmission is through close contact between cats shedding the virus and naïve cats.
Saliva is the most important means of transmission.
This has implications for multiple cat households where food bowls and water bowls are shared and where cats groom each other.
The virus is easily inactivated because of its lipid envelope and hence it does not persist in the environment.
Indirect transmission via humans is this not possible.

Queens may infect kitten in-utero or during nursing.
Queens may test negative and still have a latent infection that reactivates with gestation and parturition.
Kittens may also initially test negative despite being infected. They will become test positive only when the virus begins replicating. Hence if one kitten in a litter should test positive, all kittens in that litter should be regarded as positive and isolated.

Susceptibility is highest amongst young kittens.
Experimental infection of healthy adult cats is almost impossible.
So, although exposure increases with age, susceptibility to develop viraemic infection decreases with age.

Disease pathogenesis varies widely and depends on viral pathogenicity, infection pressure, the age of the cat and the cat's immune status.

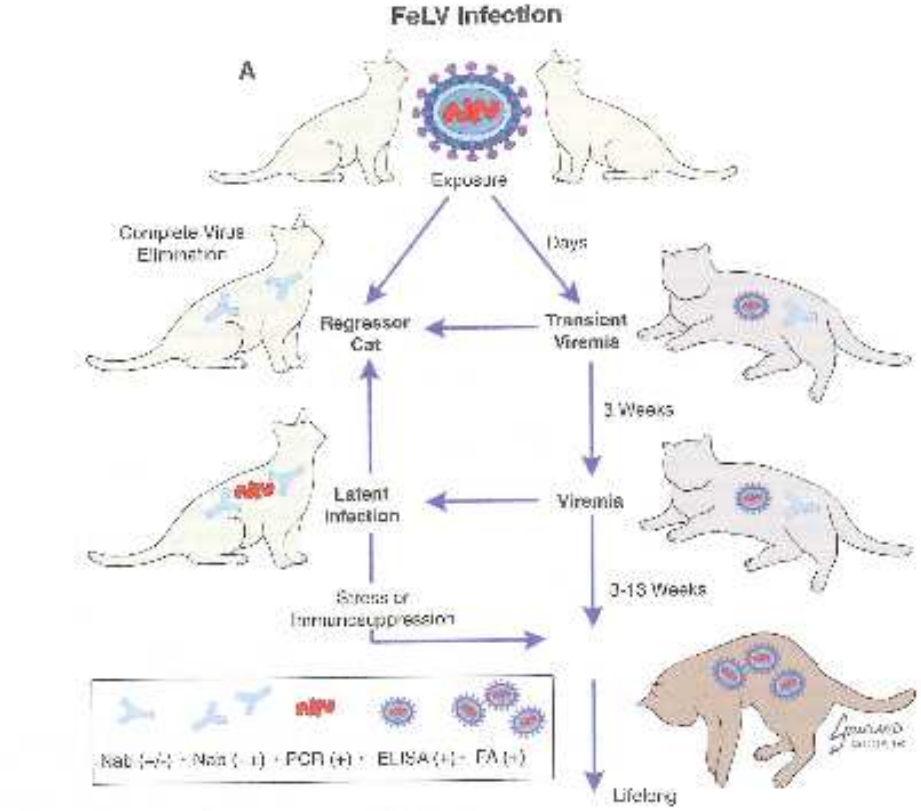

Figure: a time course of FeLV infection.

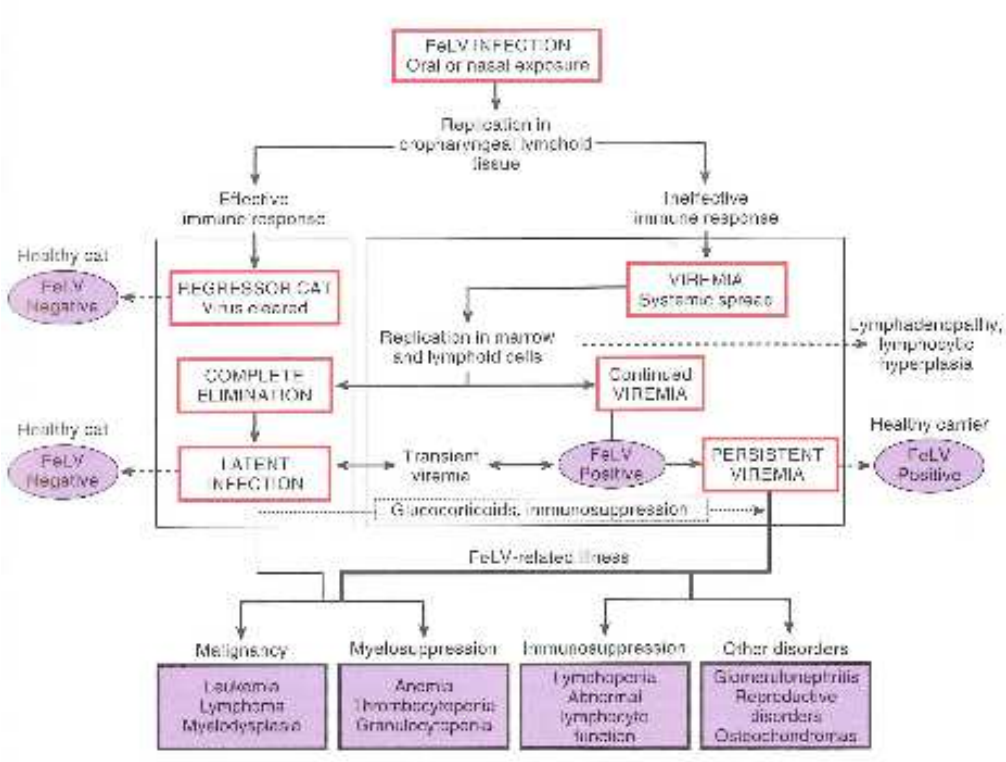

Figure: Interactions of FeLV with host cells and immune system leading to various clinical problems in cats with ineffective immune responses.

Clinical signs - vary greatly.

- Infection in multiple cat households carries a poorer prognosis than the infection in the general population does.
- The death rate in persistently viraemic cats in multiple cat households is 50% by 2 years and 80% by 3 years.
- Survival for persistently viraemic cats in single cat households is better.
- The reason for presentation is most commonly anaemia and clinical manifestations of immuosuppression.
- In one study that evaluated 8642 FeLV infected cats various co-infections (FIP, FIV, respiratory infections, haemotropic mycoplasmosis and stomatitis) were the most common reason for presentation (15%), followed by anaemia (11%), lymphoma (6%), leukopaenia or thrombocytopaenia (5%), or leukopaenia or myeloproliferative disease (4%).
- The reasons for the varied clinical presentation of persistent viraemia are unclear.
- It is clear that the single most important factor determining outcome however is the age at which the cat becomes infected. The younger they are when infected the more rapid the course of disease and the earlier death, the older, the slower the course of the disease and the longer the period of health before clinical disease and ultimately death.

FeLV induced tumours:

- FeLV is a major oncogene causing lymphoma and leukaemia most commonly.
- The association between FeLV and these malignancies has been clearly established in many ways but the association is now on the decrease as the number of non-FeLV associated lymphoid malignancies is increasing.
- It must however be remembered that although detecting an FeLV antigen positive test in a cats blood that has lymphoid malignancy may be less common, it is clear that latent infections (where provirus is present in bone marrow cells) are known to be oncogenic.
- FeLV associated lymphomas are T-cell in origin. B-cell tumours are usually not FeLV associated.
- Mediastinal lymphomas are most frequently FeLV induced.
- Gastro-intestinal lymphoma is less frequently FeLV associated.
- About half of the cats with multicentric lymphoma are FeLV positive.

Haematopeoitic Neoplasia (Myeloproliferative disorders)

- More than half of the cats with non-lymphoid leukaemia are FeLV positive.
- Chronic neoplastic bone marrow stimulation may result in myelofibrosis.
- Fibrosarcoma may be caused by FeSV – a recombinant virus that develop from FeLV combining with cell-derived oncogenes.

Non neoplastic haematological dysfunction

- Cytopaenias caused by bone marrow suppression are a common sequel to FeLV infection.
- Non-regenerative anaemia is the most common (more than 60% of non-regenerative anaemias in cats are caused by FeLV).
- Haemolytic anaemia may also be a feature of FeLV and these anaemias are strongly regenerative. This is a secondary IMHA and may go together with *Mycoplasma haemofelis* infection.
- Thrombocytopaenic bleeding tendencies can also be caused by FeLV as a form of secondary IMTP.

Immune Suppression

- Diseases associated with immunosuppression account for a high proportion of the FeLV associated morbidity and mortality.
- The immunosuppression is more severe than what is seen with FIV.
- Antibody responses are delayed and blunted in infected cats and hence response to vaccinations is not optimal and more frequent vaccination has been suggested.
- The most common infections associated with FeLV are FIP, haemobartenellosis, coccidiosis and upper respiratory tract infections.
- Most of these secondary infections are treatable.

Diagnosis

- **Testing cats and preventing exposure of uninfected cats to infected cats is the most effective way of controlling the disease.**
- FeLV vaccination should NOT be considered a substitute for testing.
- Retesting 90 days after a first test is recommended to be absolutely sure that the cat is negative following and exposure.

Testing guidelines are given below:

General Considerations
Indications for testing • General health screen for all cats • Any illness regardless of previous testing or vaccination • Presence of FeLV associated diseases (haematologic, neoplastic, immunosuppressive) • Adoption (regardless of age) • Potential exposure (at least 28 days since exposure)
Test selection • ELISA for screening (serum better than whole blood) • Direct FA tests for cell associated Ag • PCR detects latent infection and is only indicated if other tests are negative

1. Remember the ELISA test detects the **antigen (p27)** and not antibodies.
2. Tests thus indicate a viraemia and this may be reflective of a either a transient (before bone marrow infection) or persistent state.
3. ELISA tests are best performed on serum or plasma (this reduces false negative and positive results).
4. It is always advisable to retest a positive test result (false positives are more likely as the disease incidence decreases as a disease incidence drops, predictive values of a test do as well).
5. Retesting of cases in which the disease is less likely is especially important. A cat with a positive test that has a thymic lymphoma is more believable than a positive result in a cat that comes a single cat household with minimal or no exposure.

Treatment is indicated despite the decreased life expectancy in most of these cats as many of them will live for years from the time of diagnosis. Indeed many of these cats will live out full lives and eventually die from causes unrelated to their retroviral infection if well cared for.
Always remember that euthanasia should never be based on the presence of a positive test result alone. FeLV infected cats are subject to all the same diseases non-FeLV infected cats are and owners should always be given the option of treatment.

Management of infected households requires care around the points of virus transmission (feed bowls, water bowls, grooming behaviour).

- All cats in an infected household should be tested so that their status is known.
- Infected cats always endanger the uninfected.
- Isolation of the infected cats should be recommended.
- The risk of viral transmission to uninfected cats that have been living with infected cats for a time is not very high as they have most likely been infected and developed an immunity.
- Studies have however shown that virus neutralizing Ab in immunes may not be life-long and previously immune cats may become viraemic.
- An adult FeLV cat that has been living with FeLV positive cats for 3 months or more has been shown to be around 10-15%.
- Where owners refuse or cannot separate test negative from test positive cats, the test negative cats should be vaccinated in an attempt to boost their immunity.
- Vaccination in these environments does however not provide high levels of protection.
- Where no new cats are introduced to these environments, the test negative cats will probably outlive the positive cats and hence eventually the household should revert to being negative.

Individual cats that are test positive should be confined to indoors to protect other cats in the environment and to protect the infected immunosuppressed cat from opportunistic infections.

- Increased levels of husbandry will prolong life.
- Regular veterinary health checks will help identify diseases that can be manage early.
- It is especially important to evaluate oral health, palpate lymph nodes, skin, eyes and urinary tract should be evaluated as should a haematology and biochemistry profile.
- Vaccination programs should not be suspended.
- There is no evidence that MLV vaccines pose any greater risk to FeLV infected cats – but if an inactivated vaccine can be chosen it may be preferable to make use of these products.
- There is no point to vaccinating against FeLV in cats already infected. This has no effect on viraemia, carrier state or virus elimination.
- It has been shown that FeLV infected cats will mount weaker immune responses to vaccines than will healthy cats.
- For this reason it has been suggested than more frequent vaccination may be considered – especially against rabies in rabies endemic areas.

Treatment of FeLV associated diseases

1. Secondary diseases that result from FeLV are treated as they would be if FeLV were not present as an underlying cause.
2. Treatment may need to be prolonged or more intensive because of the FeLV status.
3. FeLV alone does not cause pyrexia and hence any FeLV positive cat with a fever should be investigated.
4. Antibiotic unresponsive pyrexia should prompt a search for a concurrent virus, fungus, protozoa or neoplasia.
5. Glucocorticoids should be avoided until there is a very clear therapeutic indication for their use.

Bone marrow suppressive syndromes:

FeLV induced anaemia is common and blood transfusion is crucial.
Only if there is an IMHA present should glucocorticoids be used.
A thorough search for Mycoplasma should always be instituted.
Using human recombinant erythropoietin has proven successful in cases of non-regenerative anaemia.
Severe neutropaenia may be as a result of immune mediated arrest of neutrophil precursors. Glucocorticoids are indicated in this case. Bone marrow cytology or biopsy is the diagnostic test of choice in these cases.
In cases of panleukopaenia as a result of myeloid hypoplasia, FeLV is the likely cause and glucorticoids are contraindicated. In these cases granulocyte colony stimulating factor may be useful.

Lymphoma and leukaemia:

In my cases chemotherapy results in remission that may last years.
Although the prognosis for a lymphoma that is FeLV positive is worse, treatment can nevertheless make a significant difference and benefit the cat.
Alimentary neoplasia carries a worse prognosis than neoplasia in other sites.
Normal chemotherapy protocols are followed. These typically include a combination of cyclophosphamide, vincristine, prednisilone and doxorubicin. The median remission time for this combination is around 240 days.
Leukaemia is more difficult to treat as the bone marrow is usually crowded out by neoplastic cells and it may take several weeks depopulate the marrow and allow repopulation with non-neoplastic stem cells. The prognosis for leukaemia is very poor.

Antiviral chemotherapy

Although much has been published in this area, little of the published data is of much use as much of it has not been properly conducted.
No treatment has been found that clears the virus from the body.
Zidovudine (AZT) seems to have little effect and is less effective against FeLV than it is against FIV. It should not be used in cats with myelosuppressive syndromes since the drug itself is myelosuppressive.
Various other antivirals have been used but none of them have shown much promise.

Immune modulating therapy

INF-α has immunomodulatory effects.
High dose SC injection and low dose PO regimes have been used.
The low dose PO regime has shown the most promising results as it does reduce the incidence of clinical signs (30IU daily, one week on, one week off continued until the cat is regarded as clinically healthy and then stopped).
Feline INF-γ has been used with some success
Many immunomodulators have been used to try and treat FeLV. None have shown significant benefit.

Prevention

Viraemic cats shed virus in saliva especially.
The virus is very labile and hence close social contact is required for transmission.
Viraemic cats should be separated from non-viraemic ones.
Positive cats can quite safely be hospitalized with negative cats in the same hospital as long as they are in separate cages and basic hygiene is followed between contacts.
FeLV positive cats should never be hospitalized in an isolation facility holding other cats with contagious infectious disease.

Testing and removal strategies have been highly effective in reducing the rate of FeLV infection.
This strategy has probably had more of an influence in reducing the infection rate than vaccination.

Vaccine development has taken a long and tortuous route and has proven to much more difficult than was initially thought and more difficult than for other feline viral diseases.
Naturally exposed cats produce antibodies to the virus and become immune and hence it was reasoned that vaccination was theoretically possible.
Vaccination does not interfere with testing.
No vaccine is 100% effective and hence irrespective of vaccine status it is ill advised to put naïve cats with viraemic virus shedding cats and think the vaccine is all that is required for protection.
Immune correlates of vaccine induced protection are difficult to find. Ab titer is not helpful.
measures of CMI are more accurate but technically difficult to perform.

Feline Panleukopaenia (Parvo) Virus

- Like CPV, FPV is a very resistant virus, surviving for as long as a year in organic material, resisting heating to 56°C for 30 min and even surviving 70% alcohol. It does however succumb to bleach disinfection (6% hypochlorite).
- The virus is ubiquitous and virtually all susceptible cats are infected by a year of age.
- MDA can prevent infection and vaccine uptake for as long as 20 weeks (although 3 months is more typical).
- Most infections are sub-clinical.

1. The virus persistence in the environment means that most infections occur indirectly.
2. Viral shedding by infected cats is of short duration.

Pathogenesis is typical for a parvovirus in that it requires rapidly multiplying cells for successful infection.
Bone marrow, intestinal epithelium and thymus are thus target tissues.

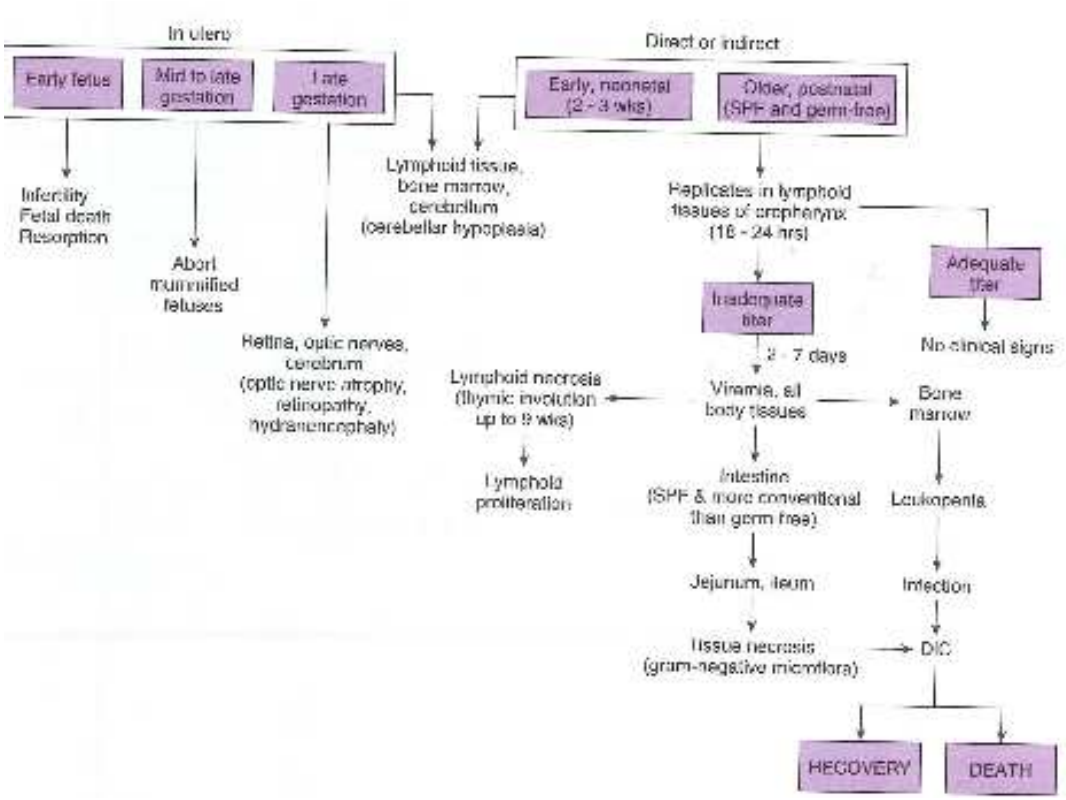

Figure: Pathogenesis of feline panleukopaenia infection.

Clinically, the disease is usually only significant in unvaccinated kittens between 3 and 5 months of age.

- It may however be a cause of fading kittens and it may cause significant mortality in young kittens born to unvaccinated queens.
- Clinical signs include pyrexia, depression, anorexia, vomiting and dehydration. Diarrhoea is less common.
- Death is usually due to septic shock.

Diagnosis is often based on clinical signs and profound leukopaenia.
Serology is not a widely used clinical tool for diagnosing this disease.
An ELISA snap test is available for in-clinic diagnosis. One must however remember that faecal shedding time is short and hence the test may be negative in cases older than three days.

Treatment is as for a dog with parvovirus infection with the one additional treatment required being combination vitamin B given parenterally.

Prevention

- Depends heavily on vaccination once MDA titers have dropped sufficiently (which may only be by 12-14 weeks).
- Pregnant queens and kittens under 4 weeks of age should never be vaccinated with a MLV. Inactivated products are however safe in these animals and are especially helpful in protecting colostrum deprived kittens.
- MLV products provide far better protection earlier following vaccination.
- Although annual vaccination is practiced by most, it is probably unnecessary.
- An initial series of vaccinations have been shown to be effective for as long as 6 years.

Feline Respiratory Disease

- This complex of disease is usually seen in cats that are grouped together in multiple cat households, boarding facilities, humane societies and the like.
- The disease is multifactorial.
- The majority of disease is caused by Feline Herpes Virus-1 (FHV-1, also called feline rhinotracheitis virus) and feline calici virus (FCV).
- FHV seems to induce more severe disease but FCV appears to be more prevalent.
- It is increasingly clear that *Bordetella bronchiseptica* is also a primary pathogen in cats. Interestingly it seems this organism can be transmitted between dogs and cats.
- *Chlamydophila felis* is also involved in feline respiratory disease although it is considered to be a predominantly conjunctival infection.

- FHV is enveloped and does not persist in the environment.
- FCV is not enveloped and hence is more resistant in the environment surviving for up to a week or even longer in damp.

Epidemiology

FHV and FCV are wide spread with a higher prevalence in multiple cat households.
They are shed in ocular, nasal and oral secretions and spread is by contact.
Acutely infected cats are the most important source of infection but recovered cats can become carriers and shed virus. Despite vaccination, chronic carriers remain the most important source of infection.
Aerosol are not believed to be important in transmission.

FHV-1 carrier state:

- Almost all infected cats become latently infected carriers.
- Stress may reactivate viral shedding which endangers cats in close proximity.
- The stressor is followed by shedding about a week later which then lasts about 2 weeks.
- Mild clinical signs may recur during these shedding episodes.
- Parturition can induce shedding but MDA usually protects kittens.

FCV carrier state:

- FCV carriers usually shed virus almost continuously.
- The virus persists in lymphoid tissue in the upper respiratory tract.
- Most animals will eventually clear their infections; some will remain life long carriers.
- Despite the introduction of a vaccine, the carrier state remains common.
- Some studies have shown that between 20 and 30% of a cat population are shedding at any one time.
- FIV may potentiate shedding.
- Vaccination protects against the disease but not the carrier state.

Bordetella bronchiseptica

- Seroprevalence varies between 24 and 79% and organisms can be isolated from around 50% of cats.
- It is a cause of disease in stressed animals and crowded facilities.
- The organisms can be transmitted between dogs and cats. This has implications for where dogs and cats are housed together.
- A carrier state for this organisms also exists.
- Shedding may persist for nearly 5 months after a clinical infection.

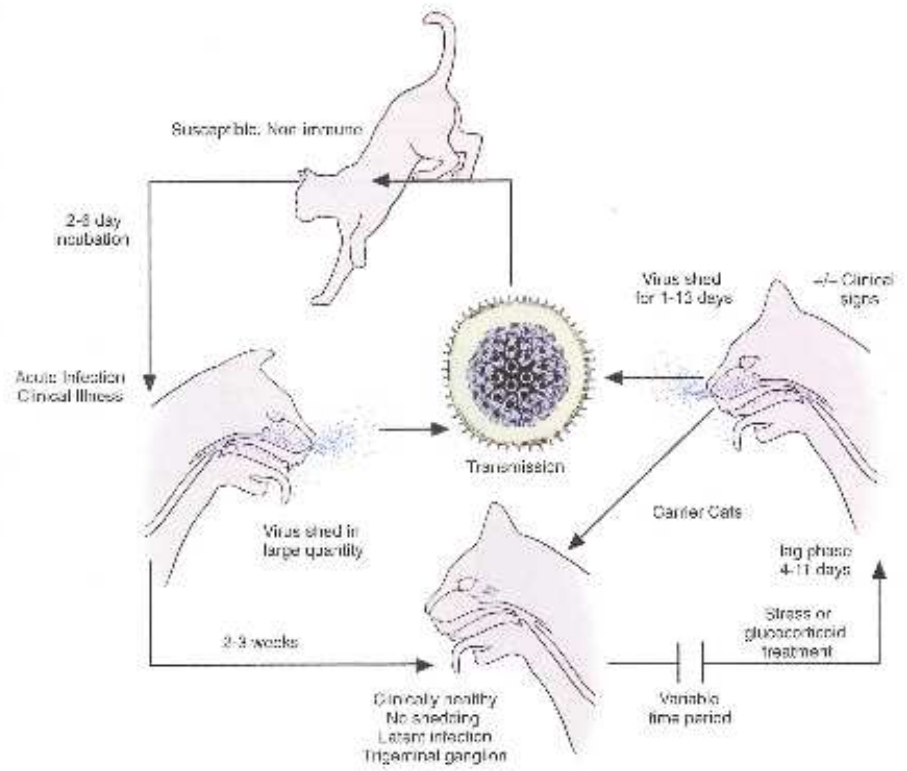

Figure: FHV-1 carrier state epidemiology.

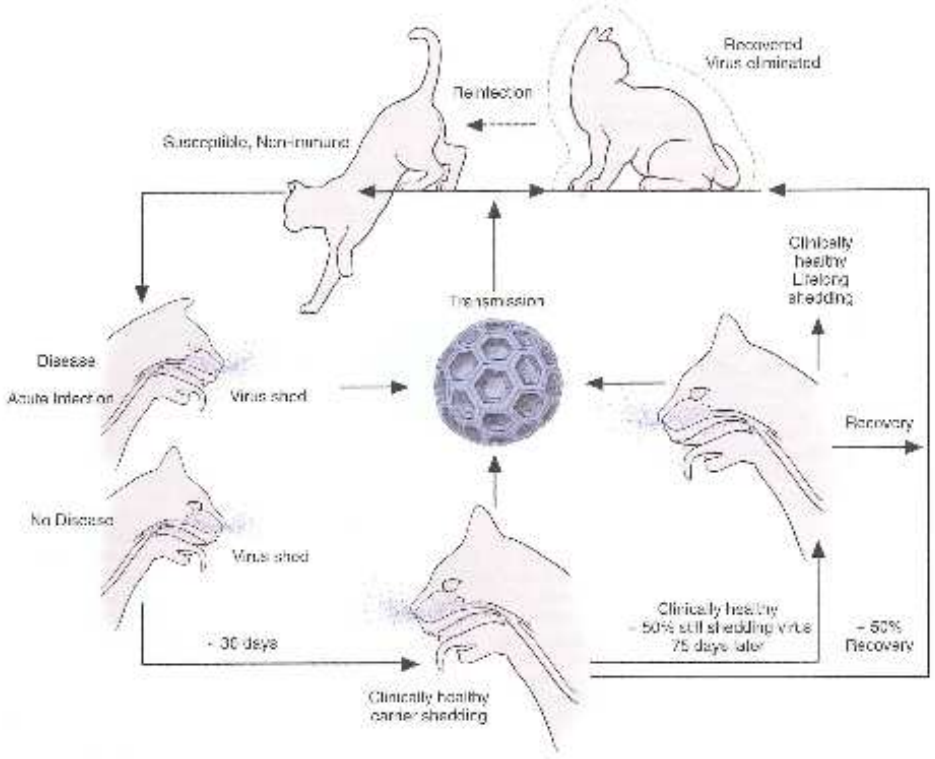

Figure: FCV carrier state epidemiology.

Pathogenesis and pathology

FHV-1 infection leads to multifocal epithelial necrosis with neutrophilic infiltration and fibrin exudation.
Turbinates may show lytic changes.
Lesions usually take 2-3 weeks to resolve.
Lung involvement is rare.
Secondary bacterial infection frequently complicates the clinical picture.

FCV usually causes oral ulceration.
Ulcers take 2-3 weeks to heal.
Pulmonary lesions are initially and alveolitis followed by pneumonia.
FCV can also cause acute sinovitis causing joint pain.

Bordetella bronchiseptica causes upper respiratory tract disease in cats (in dogs it is usually tracheal and bronchial). The release of bacterial toxins cause acute inflammation.

Clinical findings

- FHV-1 causes depression, sneezing, inappetance, pyrexia with ocular nasal discharge.
- Conjunctivitis may be severe. Keratitis, corneal ulceration and uveitis may occur.
- Discharges become mucopurulent and dry.
- Primary viral pneumonia may occur.
- In the very young and very old mortality rate may be high but generally it is low with signs resolving in around 3 weeks.
- Severe infection may lead to permanent remodelling of the turbinates and respiratory epithelial metaplasia which predisposes to lifelong secondary bacterial infection.
- FCV induces a more mild syndrome in which oral ulceration is common.
- Acute lameness may be a feature but it is short lived and fully reverses.
- Lymphoplasmacytic gingivitis stomatitis syndrome may be caused by FCV infection.

Bordetella bronchiseptica **causes a broad range of upper respiratory signs ad may also cause pneumonia.**

Diagnosis frequently rests on clinical signs alone.

- Viral isolation can be performed on oropharyngeal swabs.
- ELISA and PCR techniques are also available.
- Swabs for *Bordetella bronchiseptica* isolation must be collected in a special transport medium (charcoal Amies transport medium).

Treatment: Consult with your veterinarian: Fastidious nursing care is essential.

Prevention

- Most cats are protected following the use of a MLV vaccine for FHV but immunity is not necessarily complete.
- Immunity usually lasts beyond a year but one study showed that by 7,5 years it had declined by 50%.
- FCV titers are generally higher than with FHV and immunity is usually longer lived (usually extending beyond a year but declining to beneath detection by 7 years).
- MDA titers protect kittens for between 9 and 14 weeks.
- An effective IN vaccine against *Bordetella bronchiseptica* is in use.

Disease control:

- Routine vaccination is essential.
- *Bordetella bronchiseptica* vaccination can probably be reserved for high risk environments.
- Household cats should probably be vaccinated yearly (especially if they board for annual holidays).
- Boarding catteries should insist on current vaccination and consideration can be given to the use of a Bordatella vaccine.