Feline Panleukopenia
updated September 17, 2015


Virus

Feline panleukopenia virus (FPV) is the prototype of closely related parvoviruses isolated from dogs, mink, raccoons, raccoon dogs, foxes and other canids (Parrish, 1990). They were initially named after the hosts from which they had been isolated. Current taxonomy defines canine parvovirus and feline panleukopenia virus as a single entity (Tattersall, 2006), but in the present guidelines, FPV refers the parvovirus in cats.
Virions contain single-stranded genomic DNA, are not enveloped (naked), sometimes penetrated by stain; they are 18-22 nm in diameter, of icosahedral symmetry, but appear to be round and hexagonal in electron micrographs (Fig. 1).

Unlike most other DNA viruses, parvoviruses are unable to activate DNA synthesis in host cells. Their replication is in the nucleus (Fig. 2, 3), and the infected cells must be actively mitotic.

This explains why tissue damage occurs predominantly in rapidly dividing cells, like those in the intestine, bone marrow, and in embryonic tissue.

FPV infects cats and other members of the Felidae, as well as raccoons, mink, and foxes (Steinel et al., 2001). It also infects dogs, where FPV replication was seen in lymphoid tissues (thymus, spleen, etc.).
In 1978, a new parvovirus, closely related to FPV, was first described in dogs (Carmichael, 2005). It was named canine parvovirus type 2 (CPV-2), to distinguish it from another parvovirus isolated from dogs in 1970, which is now called "canine minute virus". CPV-2 has evolved from FPV by acquiring 5 or 6 amino acid changes in the capsid protein gene (Parrish, 1990; Truyen, 1999) and is no longer able to infect cats. However, during further adaptation to the canine host, which most likely occurred in the raccoon, the raccoon virus acquired the amino acid changes that had enabled the new virus to better bind to the canine cellular receptor but also retained its ability to infect cats (Hueffer and Parrish, 2003; Allison et al., 2012). This led to the generation of the new type CPV-2a, that acquired further mutations including those at amino acid 426 of the VP2, which determine the different antigenic types 2a, 2b, and 2c. The parvoviruses now circulating in the dog populations worldwide - genetically and antigenically defined as types CPV-2a, -2b, and -2c - can infect cats and may even cause disease (Truyen et al., 1995, 1996; Mochizuki et al., 1996). However, CPV infections of cats are rare in Europe and the USA, and the virus has only sporadically been found in diagnostic material (Truyen et al., 1996). CPV was isolated from feline peripheral blood lymphocytes after numerous blind passages, and viral DNA was demonstrated by PCR (Ikeda et al., 2000). Recently, however, a case of CPV-2c infection in a cat with severe clinical signs was described in Portugal (Miranda et al., 2014).

During the evolution from FPV to CPV-2 with its various antigenic types, neutralizing epitopes have been affected such that cross-neutralization by FPV antisera is markedly lower against the new virus (Truyen and Parrish, 1993).

Epidemiology

FPV is non-enveloped and highly resistant to physical factors and chemical substances. In contaminated environments, it may remain infectious for weeks or even months (Uttenthal et al., 1999). Diseased carnivores shed virus at high titres (up to 10^9 TCID50 per gram of faeces), and virus quickly accumulates in affected shelters and catteries. As it is highly contagious, susceptible animals may still become infected, even after a seemingly thorough disinfection of the premises. It is therefore recommended that only successfully vaccinated kittens and cats should enter such an environment.

Although few data on FPV prevalence are available, particularly breeding catteries and rescue shelters are at risk (Addie et al., 1998; Cave et al., 2002). Persistent infections and persistent viral shedding are rare; using PCR, healthy cats have been found positive in faeces over weeks; it is unknown whether this is of epidemiological significance (Jakel et al., 2012). Interestingly, CPV-2 viruses could be isolated from feces of healthy cats in the UK in two shelters. It is unclear if this is of epidemiological importance (Clegg et al., 2012).

After intrauterine infection, FPV antigen is present in the cerebellum of kittens for weeks (Csiza et al., 1971). The analysis of parvovirus sequences recovered from wild carnivores (pumas, coyotes, raccoons, and others) revealed a broad range of virus types. This implicates the infection of predators by their prey, if the latter was infected with parvoviruses, and thus a new route of infection (Allison et al., 2013).

Pathogenesis

FPV causes a systemic infection. The virus is transmitted via the faecal-oral route, initially replicates in tissues of the oropharynx and is then distributed via cell-free viraemia to virtually all tissues. Replication of the parvoviral single-stranded DNA requires cells in the S-phase of division and is therefore restricted to mitotically active tissues; in the gut, this results in enteritis (Figs. 4, 5). Paroviruses require cellular DNA polymerases to synthesize the complementary DNA strand, which is the first step in replication and a prerequisite for transcription.
The virus infects lymphoid tissues where it may cause cellular depletion and a functional immunosuppression. Lymphopenia may arise as a result of lymphocytolysis but also indirectly, from lymphocyte emigration into tissues. The bone marrow is affected, and virus replication has been described in early progenitor cells, with dramatic effects on virtually all myeloid cell populations (Parrish, 1995). "Panleukopenia", i.e. the deficiency of all white cell populations is the result (Truyen and Parrish, 2000).

The hallmark of FPV replication is the shortening of the intestinal villi due to a sometimes complete loss of epithelial cells in the gut (Parrish, 2006). The virus replicates in the rapidly dividing cells in the crypts of Lieberkühn, which impairs regeneration of the epithelium and results in the lesions described above (Fig. 6). Their severity correlates with the epithelial turnover rate, and co-infection with enteric viruses - like feline coronavirus - may enhance the severity of disease.
Intrauterine transmission or perinatal infection may affect central nervous system development. "Feline ataxia syndrome" results from an impaired development of the cerebellum due to lytic infection of the Purkinje cells in the kitten (Figs. 7, 8) (Csiza et al., 1971; Kilham et al., 1971). An FPV-like virus has been described as the cause of reproductive disorders in pregnant foxes (Veijalainen and Smeds, 1988).

Foetal infection may induce immunological tolerance, so that kittens continue shedding virus for extended periods of time (Pedersen, 1987).

Foetuses infected between the 35th and 45th days of gestation have depressed T-lymphocyte mediated immunity. In adult cats, infection leads to a transient decrease in the immune response: neutrophil counts decrease severely, and lymphocytes disappear from the circulation, lymph nodes, bone marrow and thymus (Pedersen, 1987; Ikeda et al., 1998).

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Table 1

<table>
<thead>
<tr>
<th>Affected cells</th>
<th>Consequences</th>
<th>Clinical manifestation</th>
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<tbody>
<tr>
<td>Intestinal crypt epithelium</td>
<td>Villous collapse, enteritis</td>
<td>Diarrhoea</td>
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<tr>
<td>Lymph node, thymus</td>
<td>Germinal centre depletion, apoptosis of lymphocytes, thymic atrophy</td>
<td>Lymphopenia</td>
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</table>
Immunity

Passive immunity acquired via colostrum

In the kitten, maternal antibodies have a biological half-life of about ten days (Scott et al., 1970; Pedersen 1987). When antibodies have waned below a titre of 40 to 80 (as measured by haemagglutination inhibition) they do not reliably protect against infection but may interfere with active immunisation (Fig.9). Most cats have maternal antibodies at protective titres until weeks 6 to 8. Later immunisations are effective (Dawson et al., 2001; EBM grade I), which makes ABCD recommend vaccinations at 15 to 16 weeks of age, as explained in the present Guidelines.

Active immune response against FPV

Antibodies play an important role in the immune response to FPV. Maternally-derived antibodies (MDA) efficiently protect kittens from fatal infection. This passively acquired immunity is later replaced by an active immune response obtained by vaccination or as a consequence of a natural infection.

Acquired immunity is solid and long lasting (Thiry, 2002a) and can be induced by both inactivated and modified live virus (MLV) vaccines. FPV antiserum can be used for passive immunisation when unvaccinated animal are likely to be exposed to virus before the initiation of a vaccine-induced, active response (Barlough et al., 1997).

Parvoviruses induce a range of immune responses including T-helper CD4+ lymphocytes and CD8+ cytotoxic T lymphocytes. Parvovirus uptake occurs by phagocytosis but also by other non-phagocytic mechanisms such as fluid pinocytosis or receptor-mediated endocytosis (Sedlik et al., 2000).
Diagnosis

In practice, FPV antigen detection in faeces is usually carried out using commercially available latex agglutination or immunochromatographic tests (Veijalainen et al., 1986; Addie et al., 1998). These tests have a good specificity and acceptable sensitivity when compared to reference methods (Neuerer et al., 2008; Schmitz et al., 2009; EBM grade I). Tests marketed for the detection of FPV antigen as well as those for detecting canine parvovirus antigen may be used to diagnose FPV in faeces.

Diagnosis by electron microscopy has lost its importance due to more rapid and automated alternatives. Specialised laboratories offer PCR-based test on whole blood or faeces. By PCR, healthy cats tested positive in faeces over weeks, but the epidemiological significance of this finding is unknown. Clinicians need to bear this in mind when interpreting diagnostic data.

Disease management

A cat showing clinical signs of feline panleukopenia, substantiated by laboratory evidence should be kept in isolation. Supportive therapy and good nursing care significantly decrease s mortality caused by FPV. The severe dehydration accelerates disease progression (Fig. 10, 11). Restoration of fluid and electrolyte, and of the acid-base balance preferably by intravenous drip is most important in symptomatic treatment (Fig. 12).
As the gut barrier often is destroyed in FPV-infected cats, intestinal bacteria may invade the blood stream. Bacteraemia may ensue, facilitated by the existing neutropenia, and leading to sepsis in these immunocompromised patients. Prevention of sepsis is essential, and a broad-spectrum antibiotic with a proven efficacy against gram-negative and anaerobic bacteria is recommended. Examples are amoxicillin/clavulanic acid or piperacillin in combination with aminoglycosides, fluoroquinolones, cephapirin or piperacillin/tazobactam. The potential side effects of these drugs should be taken into consideration. Antibiotics should be administered parenterally (preferentially intravenously).

Oral intake of water and food should only be restricted if vomiting persists, and feeding should be continued as long as possible, and restarted as soon as possible. Beneficial effects of early enteral nutrition have been reported in canine parvovirus (Mohr et al., 2003). A highly digestible diet is preferred, but if the cat does not accept it, any diet is better than no food intake at all. If vomiting persists, anti-emetics should be considered. Vitamin supplements, particularly of the B vitamin complex can be given to prevent development of thiamine deficiency, an infrequent sequel, which occurs infrequently.

Cats that develop hypoproteinaemia may require plasma or whole blood transfusions to restore oncotic pressure. Plasma transfusion in combination with heparin may control disseminated intravascular coagulation (DIC), as it supplements anti-thrombin III and other important plasma proteins. In cats that are anorexic or show severe vomiting and/or diarrhoea, or in patients with persisting hypoproteinaemia, full or partial parenteral nutrition is required, preferably via a central venous catheter in the jugular vein (Hartmann and Hein, 2002).

Table 2. Overview of treatment in cats with FPV (all measures are EBM level 4)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Comment</th>
<th>ABCD recommendation</th>
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<tbody>
<tr>
<td><strong>Antiviral Therapy</strong></td>
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<tr>
<td>anti-FPV serum</td>
<td>anti-CPV serum effective in dogs</td>
<td>beneficial effects in cats expected</td>
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<tr>
<td>feline recombinant Interferon-omega</td>
<td>effective in dogs</td>
<td>beneficial effects in cats expected</td>
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<tr>
<td><strong>Symptomatic Therapy</strong></td>
<td></td>
<td></td>
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<tr>
<td>fluid therapy</td>
<td>to control dehydration and restore electrolyte and acid base balance</td>
<td>necessary in every cat with vomiting and diarrhea</td>
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<tr>
<td>antibiotics</td>
<td>prevention of sepsis</td>
<td>broad-spectrum antibiotic with proven efficacy against</td>
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<td>(amoxicillin/clavulanic acid</td>
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<td>gram-negative and</td>
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<td>in combination with</td>
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<td>anaerobic bacteria recommended</td>
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<td>- aminoglycosides or</td>
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<td>- fluoroquinolones* or</td>
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<td></td>
<td>restarted as soon as possible</td>
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<tr>
<td>Antiemetics</td>
<td>to control vomiting</td>
<td>recommended for vomiting animals</td>
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<tr>
<td>B vitamin complex</td>
<td>prevention of thiamine deficiency</td>
<td>recommended</td>
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<tr>
<td>plasma or whole blood transfusion</td>
<td>to restore oncotic pressure</td>
<td>recommended in cats with hypoproteinaemia</td>
</tr>
<tr>
<td>full or partial parenteral</td>
<td>to restore oncotic pressure and meet energy requirements</td>
<td>recommended in cats with anorexia, severe vomiting/diarrhea or persisting hypoproteinaemia</td>
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<tr>
<td>nutrition</td>
<td></td>
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<tr>
<td>Low-molecular heparin (fragmin)</td>
<td>to control DIC</td>
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Anti-FPV serum can be used to prevent infection of susceptible animals following after exposure. The therapeutic efficacy of immune serum has been demonstrated in dogs (Meunier et al., 1985), and
similar beneficial effects may be expected in cats.

Feline recombinant interferon-omega is effective in the treatment of parvoviral enteritis in dogs (Minagawa et al., 1999; Martin et al., 2002; De Mari et al., 2003) and also inhibits replication of FPV in cell culture. So far no data are available on its efficacy in FPV-infected cats.

Due to the extreme physicochemical stability of FPV, contaminated cages, litter trays, food dishes, water bowls, shoes and clothing can play an important role in transmission. Therefore attention to hygiene is of utmost importance. The virus is resistant to many common disinfectants, but can be inactivated by products that contain peracetic acid, formaldehyde, sodium hypochlorite, or sodium hypochlorite hydroxide. Commercially available chemical disinfectants proven effective against non-enveloped viruses or a disinfectant (solution or dry fog) based on formaldehyde or peracetic acid can be used in room disinfection.

Susceptible kittens and unvaccinated older animals should not be in contact with other cats until they are properly immunized. Once a disease outbreak occurs, passive immunization can be used to protect susceptible cats (young kittens with an incomplete vaccination history, colostrum-deprived kittens or unvaccinated cats). Anti-FPV serum can be given subcutaneously or intraperitoneally and may protect for 2-4 weeks. If a commercial product of equine origin is used, repeated administration is not recommended as this may lead to anaphylactic reactions. Since the administered immunoglobulins will bind to parvoviral epitopes, these animals should not be vaccinated within the first three weeks after passive immunization.

**General recommendations on vaccine type and vaccination protocol**

Both MLV and adjuvanted inactivated FPV vaccines are available for administration by injection, and both provide solid immunity against disease. In an immune-competent cat capable of mounting an appropriate response, MLV vaccines generally result in a more rapid protection (Ley et al. 2006; EBM grade II). However, even a single dose of an inactivated FPV vaccine may rapidly induce good antibody responses in naïve cats within a short time span (Fischer et al., 2007). There are no data to suggest that particular vaccine brands are more efficacious than others.

In most situations, there is no reason to prefer one vaccine type to the other in an individual cat; MLV products are being used more generally, because of the more rapid onset of protection and a better resistance to MDA. There may be considerations affecting this decision:

MLV FPV vaccines should not be used in pregnant queens because of the risk of placental virus passage to the foetus and damage, especially to the developing cerebellum (Pollock and Postorino, 1994). In some countries, inactivated FPV vaccines are licensed for use in pregnant queens, but their vaccination should generally be avoided.

MLV FPV vaccines should never be administered to kittens under 4 weeks of age for the same reason: to avoid damage to the cerebellum, which is still developing in young neonates (Pollock and Postorino, 1994).

Because of the ubiquity of the virus and the serious consequences of an infection, vaccination is recommended for every cat: the FPV vaccine is de

**Primary vaccination course**

Most kittens are protected by MDA in the first weeks of life. However, without serological testing, the level of protection and the point at which a kitten will become susceptible to infection and/or can respond immunologically to vaccination is unknown; also, there is considerable variation between individuals.

In general, MDA will have waned by 8 to 12 weeks of kitten age to a level that allows an active immunological response, and an initial vaccination at 8 to 9 weeks of age followed by a second vaccination 3 to 4 weeks later is commonly recommended. The data sheets of many vaccines contain recommendations to this effect. However, kittens with poor MDA may be vulnerable (and capable of responding to vaccination) at an earlier age, while others may possess MDA at such high titres that they are incapable of responding until some time after 12 weeks of age.

No single primary vaccination policy will therefore cover all potential situations. These are ABCD's recommendations:

- **All kittens should receive FPV vaccines**
- A minimum of two doses – one at 8 to 9 weeks of age and a second 3 to 4 weeks later (at a minimum of 12 weeks of age) should be administered to cats living in low risk situations.

In higher risk situations, a third vaccination at 16 weeks is recommended. Maternal antibodies may persist beyond week 12 in some cats, as recent data suggest (Jäkel et al., 2012; Dawson et al., 2001), such that vaccination at 12 weeks may fail to induce protection (Kruse et al., 2010). Therefore, a third kitten vaccination at 16 weeks of life should be given to kittens in e.g. breeding catteries or cat shelters. A 16-week-vaccination should also be considered for kittens born to queens with high antibody titres, as these are likely to transmit high levels of MDA that may persist for more than 12 weeks in their kittens (e.g. queens that have recovered from disease, that have lived in a high-exposure environment, or have received vaccination close before or during pregnancy).

If prophylactic administration of immunoglobulins is not possible, additional earlier vaccinations should be considered, especially if MDA is known or suspected to be poor and/or if the kitten is in a high risk situation (EBM grade II). If a kitten is vaccinated at or before 4 weeks of age, this should only be done using an inactivated product, and repeat vaccinations can be done at 3 to 4 week intervals up to 12 weeks of age.

Adult cats of unknown vaccination status should receive a single initial FPV vaccine injection (MLV) followed by a booster vaccination one year later.

**Booster vaccinations**

Cats that respond to FPV vaccination maintain a solid immunity for at least seven years – the latest point in time tested - in the absence of any repeat vaccination or natural challenge (Scott and Geissinger, 1999; Lappin et al., 2002; EBM grade II). Nevertheless, the ABCD recommends the following revaccination protocol:

- All cats receive a first booster 12 months after completion of the primary vaccination course (this will ensure adequate vaccine-induced immunity for cats that may not have adequately responded to the primary course)
- Following this first booster, subsequent revaccinations are recommended at intervals of three years or longer, unless special conditions apply (EBM grade II).

While most cases of feline parvovirus are caused by infection with FPV, variants of canine parvovirus (CPV-2a, CPV-2b, CPV-2c) have emerged that infect cats and may cause disease. Current FPV vaccines appear to afford protection against these new CPV variants (Chalmers et al., 1999; Nakamura et al. 2001; EBM grade II).

**Control in specific situations**
due to increased virus production (EBM grade III). Thus only FIV cats at high risk of exposure to infectious agents should be vaccinated, and only with killed products. (Dawson et al., 1991; Reubel et al., 1994; Foley et al., 2003; EBM grade III). In one study, cats experimentally infected with FIV developed vaccine-induced panleukopenia when given MLV FPV vaccines. Therefore, more frequent vaccination should be considered in these cats.

FIV-infected cats are capable of mounting immune responses to administered antigens except during the terminal phase of infection; also primary immune responses may be delayed or diminished in immunocompromised animals only. Nonetheless, cats with stable chronic conditions such as chronic renal disease, diabetes mellitus or hyperthyroidism should receive vaccines at the same frequency as healthy cats. In contrast, cats with acute illness, debilitation, or high fever should not be vaccinated, unless there are compelling reasons to do so. In these cases, inactivated preparations should be used. Reticuloendothelial cells in immunocompromised cats may not be able to mount an adequate immune response against antigens present in live vaccines. Thus only MLV vaccines should be used in immunocompromised cats. However, the use of corticosteroids at the time of vaccination should generally be avoided. In immunocompromised individuals, inactivated FPV vaccines are recommended. Modified live FPV vaccines should be used with caution in severely immunocompromised individuals, as the failure to control viral replication could potentially lead to clinical signs.

Vaccination schedules used recommended for privately owned cats are appropriate in for most breeding catteries. Queens not up-to-date on vaccinations may receive booster vaccines injections before prior to breeding, to maximize delivery of MDA to kittens (Lawler and Evans, 1997; EBM grade I). As a consequence, their kittens from such queens may need an extra primary vaccination at 16 to 20 weeks, because of a longer in case of persisterpersistance of anti-MDA. As stated before, routine vaccination of pregnant cats should be avoided.

Lactation is not known to interfere with the immune response. However, administration of any vaccine may stress the queen and may result in a temporary deterioration of mothering ability and milk production. Vaccination of lactating queens should therefore be avoided.

Breeding catteries

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Vaccination of immunocompromised cats

Vaccines cannot generate optimum protection in animals with conditions that compromise their immune function. Such conditions include genetic immunodeficiencies, but also deficient nutrition, genetic immunodeficiencies, systemic disease, concurrent administration of immunosuppressive drugs, and environmental stress. Efforts should be made to correct the latter conditions before vaccination, and to protect cats from exposure to infectious agents and to correct these conditions if possible prior to vaccination; if this cannot be assured, vaccination should be performed nevertheless and repeated after the animal is fully recovered.

In immunocompromised individuals, inactivated FPV vaccines are recommended. Modified live FPV vaccines should be used with caution in severely immunocompromised individuals, as the failure to control viral replication could potentially lead to clinical signs.

In cats receiving corticosteroids, vaccination should be considered carefully. Depending on dosage and duration of treatment, corticosteroids may cause functional suppression of particularly cell-mediated immune responses, but pertinent studies are lacking. In dogs, corticosteroids do not hamper effective immunization if given for short periods of time at low to moderate doses (Nara et al., 1979; EBM grade IV). However, the use of corticosteroids at the time of vaccination should generally be avoided.

In cats with chronic illness vaccination may sometimes be necessary. Manufacturers evaluate vaccine safety and efficacy in healthy animals and accordingly label their vaccines for use in healthy animals only. Nonetheless, cats with stable chronic conditions such as chronic renal disease, diabetes mellitus or hyperthyroidism should receive vaccines at the same frequency as healthy cats. In contrast, cats with acute illness, debilitation, or high fever should not be vaccinated, unless there are compelling reasons to do so. In these cases, inactivated preparations should be used.

Retrovirus-infected cats should be kept indoors and isolated, to diminish the likelihood of infecting other cats and to reduce exposure to other infectious agents. FeLV-infected cats should be vaccinated against FPV. Although there is no evidence that FeLV-infected cats are at an increased risk of vaccine-induced disease from residual virulence of MLV vaccines, noninactivated (“killed”) infectious vaccines products are preferred if available/recommended. FeLV-infected cats may not be able to mount adequate immune responses to rabies vaccines, and perhaps also not to other vaccines. Therefore, more frequent vaccination should be considered in these cats.

FIV-infected cats are capable of mounting immune responses to administered antigens except during the terminal phase of infection; also primary immune responses may be delayed or diminished (Dawson et al., 1991; Reubel et al., 1994; Foley et al., 2003; EBM grade III). In one study, cats experimentally infected with FIV developed vaccine-induced panleukopenia when given MLV FPV vaccines (Buonavoglia et al., 1993; EBM grade III). Immune stimulation of FIV-infected lymphocytes in vivo promotes virus production, and in vivo, vaccination of chronically infected cats with a synthetic peptide was associated with a decrease in the CD4+/CD8+ ratios (Lehmann et al., 1992; Reubel et al., 1994). Therefore, a potential trade-off to protection from secondary disease is the progression of FIV infection due to increased virus production (EBM grade III). Thus only FIV cats at high risk of exposure to infectious agents should be vaccinated, and only with killed products.
infected with feline immunodeficiency virus. Vet Immunol Immunopathol 35:199-214


