ABCD GUIDELINES ON:

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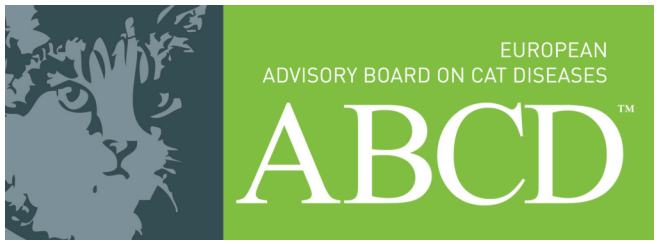

The European Advisory Board on Cat Diseases is an independent panel of 17 veterinarians from ten European countries, with an expertise in immunology, vaccinology and/or feline medicine. The ABCD was set up to compile guidelines for the prevention and management of major feline infectious disease in Europe based on current scientific knowledge and available vaccines.

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3. Feline calicivirus

3.1 Virus properties

Feline calicivirus (FCV) is a highly contagious pathogen with a widespread distribution in the feline population. It belongs to the *Caliciviridae* family that includes important pathogens of man (such as the Norwalk virus, one of the commonest causes of infectious gastroenteritis in people) and animals, including the European brown hare syndrome virus and rabbit haemorrhagic disease virus (Green at al., 2000).

Feline calicivirus has a small single-stranded RNA genome of positive (messenger) polarity; this allows FCV to evolve quickly. The genome is enclosed by multiple copies of the major capsid protein. The surface of this protein contains the most variable region of the virus which is also believed to be immunodominant (the region principally targeted by the host immune response; Geissler et al., 2002; Radford et al., 1999; Tohya et al., 1997). Despite this variability, there is sufficient overlap between isolates to allow classification of the viruses as belonging to a single serotype (Povey, 1974; Povey & Ingersoll, 1975). However, antigenic differences exist between most FCV isolates, which creates considerable difficulties when trying to maximise vaccine cross protection. Genetically, most FCVs belong to a single diverse genotype (Glenn et al., 1999, Geissler et al., 1997); a second genotype has recently been described in Japan (Ohe et al., 2006).

3.2 Epidemiology

It is generally believed that there are no important reservoirs or alternative hosts for FCV. Humans are not susceptible to FCV infection. Interestingly, apart from the existence of a specific canine calicivirus, FCV-like viruses have also been isolated from dogs (Hashimoto et al., 1999; Martella et al., 2002; Roerink et al., 1999). Their role in the epidemiology of FCV in the cat (and dog) is uncertain (Binns et al., 2000; Helps et al., 2005), but is not thought to be significant.

The virus is shed predominantly with oral and nasal secretions in acute disease. On recovery, many cats continue shedding, most of them for at least 30 days post-infection, a few for several years (Wardley, 1976). A small proportion of cats may be resistant to infection (Coyne et al., 2006a), which is probably dependent on host and virus strain factors.

Feline calicivirus infection is widespread in the general cat population. The prevalence is broadly proportional to the number of cats in the household, with the highest prevalence

usually seen where large groups are housed together. The prevalence in household cats kept in small groups is generally low (~10%; Wardley et al., 1974). In contrast, cats living in colonies or shelters have a high chance of being infected, with prevalence figures between ~25% and 40% (Wardley et al., 1974; Coutts et al., 1994; Bannasch and Foley 2005; Helps et al., 2005). The prevalence within individual colonies is variable, ranging from low (Radford et al., 2001; Coyne et al., 2006a) to high (50-90%) values (Radford et al., 2003; Coyne et al., 2006a).

Infection generally occurs through direct contact with secretions from acutely infected and carrier cats. However, the virus can also persist in the environment and remains infectious for up to one month on dry surfaces at room temperature, and longer in colder weather conditions (Doultree et al., 1999; Duizer et al., 2004; Clay et al., 2006). Indirect transmission can therefore occur, especially within the close confines of a cattery, where secretions may contaminate cages, feeding and cleaning tools or personnel. Direct contact between susceptible individuals and FCV shedding carriers is probably the most common way of transmission (Wardley, 1977).

3.3 Pathogenesis

Cats can be infected with FCV via the nasal, oral or conjunctival route. The oropharynx is the primary site of replication. Transient viraemia occurs 3 to 4 days after infection, at which time the virus can be detected in many other tissues. The virus induces necrosis of epithelial cells: vesicles, typically on the margin of the tongue, develop into ulcers; in the affected regions, the dermis is infiltrated with neutrophils. Healing takes place over a period of two to three weeks (Gaskell et al., 2006).

FCV may less commonly affect other tissues, leading to pneumonia (focal alveolitis, progressing to areas of acute exudative pneumonia and then to proliferative, interstitial pneumonia) and lameness (acute synovitis with thickening of the synovial membrane and increased synovial fluid; Dawson et al., 1994). The pathogenesis of the limping syndrome is not clear; immune complexes are thought to play a role (Bennett et al., 1989). Virus may also be isolated from affected joints (Dawson et al., 1994).

The pathogenesis of virulent systemic disease caused by FCV (VS-FCV) differs considerably from the typical picture described above. These strains cause widespread vasculitis, multi-organ involvement and death in up to two thirds of the infected cats (Pedersen et al., 2000; Hurley & Sykes, 2003; Schorr-Evans et al., 2003; Coyne et al., 2006b). The pathogenesis of VS-FCV infection is unknown and may include viral evolution and/or immune-mediated components as well as environmental and management factors (Hurley, 2006). Recently,

these virulent strains have been shown to grow more rapidly in cell culture (Ossiboff et al., 2007).

Following recovery from acute disease, most cats do not clear the infection for around 30 days; a minority sheds virus for much longer, possibly for life. In these healthy FCV carriers, virus can be localised in the epithelium of the tonsils. However, tonsillectomy does not eliminate the carrier state, suggesting the virus is also located in other sites. It is believed that evolution of the variable capsid protein allows FCV to escape the host immune response and to persist in carrier cats (Johnson, 1992; Kreutz et al., 1998; Radford et al., 1998; Coyne et al., 2007).

3.4 Immunity

3.4.1 Passive immunity acquired via colostrum

Maternally derived antibodies (MDA) are important for protection during the first weeks of life and may interfere with vaccination. There are very few data on the extent and longevity for FCV MDA in cats. In general, their levels are higher and persist for longer than for feline herpesvirus (FHV-1). In an experimental study, the average half-life of MDA was determined to be 15 days and their persistence as 10-14 weeks (Johnson & Povey, 1983). However, in a field study, 20% of kittens at only six weeks of age had no detectable antibodies against a widely used vaccine strain (Dawson et al. 2001).

3.4.2 Active immune response

Virus neutralising antibodies (VNA) appear by approximately seven days post infection (Kahn et al., 1975). In general, antibody titres are higher than for FHV infection and their levels correlate well with protection against homologous challenge (Povey & Ingersoll, 1975). There is a considerable degree of antigenic variability amongst FCV strains, but it was concluded from studies of *in vitro* cross-reactivity that FCVs belong to a single serotype (Povey, 1974). Prior infection with one strain can significantly reduce the acute clinical signs upon exposure to a heterologous strain, and in some cases oral shedding may be reduced (Povey & Ingersoll, 1975; Knowles et al., 1991). In general, the level of heterologous protection will depend on the virus strains involved.

Cats may be protected also in the absence of detectable VNA (Knowles et al., 1991; Poulet et al., 2005), suggesting a role for other immune mechanisms: indeed, cellular responses have been demonstrated in vaccinated cats (Tham & Studdert, 1987). Also, FCV-specific IgG and

IgA antibodies have been demonstrated in the saliva during the course of infection (Knowles et al., 1991), although their significance in protection is unknown.

3.5 Clinical signs

FCV infection can cause acute oral and upper respiratory signs but also has been associated with chronic stomatitis which may be immune-mediated. Recently, a new syndrome, the "virulent systemic feline calicivirus (VS-FCV) disease" has been described.

3.5.1 Acute oral and upper respiratory tract disease

Clinical findings may differ, depending on the virulence of the FCV strain concerned, on the age of the affected cats and on husbandry factors. While in some cases infection is subclinical, in many others, there is a typical syndrome of lingual ulceration and a relatively mild acute respiratory disease. More severe signs can resemble the respiratory disease caused by FHV-1.

Acute oral and upper respiratory disease signs are mainly seen in kittens. The incubation period is 2 to 10 days (Hurley and Sykes, 2003). Oral ulcerations, sneezing and serous nasal discharge are the main signs (Gaskell et al., 2006). Fever is also observed. Anorexia, sometimes accompanied by hypersalivation due to oral erosions - located mainly on the tongue - are usually much more prominent than the signs of rhinitis. They usually resolve after several days. In some severe cases, pneumonia, manifested by dyspnoea, coughing, fever and depression can occur, particularly in young kittens.

3.5.2 Chronic stomatitis

FCV can be isolated from nearly all cats with the chronic lymphoplasmacytic gingivitis/stomatitis complex. It has been suggested to be an immune-mediated reaction to FCV (and potentially other) oral antigens and is characterised by a severe proliferative/ulcerative faucitis. However, the disease has not been reproduced experimentally (Knowles et al., 1991), and the exact role of FCV remains unclear, as does the role of co-infections with FIV and *Bartonella* (Glaus et al., 1997).

3.5.3 Limping syndrome

An acute transient lameness with fever can be associated with FCV infection (Ter Wee et al., 1997; Pedersen et al., 1983) and vaccination. In natural infection, it occurs a few days or weeks after the acute oral or respiratory signs (Pedersen et al., 1983; Bennett et al., 1989).

3.5.4 Virulent systemic feline calicivirus (VS-FCV) infection

Outbreaks of highly virulent and often lethal FCV infection have recently been described in the United States and in Europe (Pedersen et al., 2000; Coyne et al., 2006b). The disease has been named "hemorrhagic-like fever" (Pedersen et al., 2000) and "highly virulent feline calicivirus disease" (Schorr-Evans et al., 2003). The causative virus strains are most commonly referred to as "virulent systemic feline calicivirus" (VS-FCV); however, this term is somewhat misleading as all FCV infections are systemic - but the disease caused by other FCV strains is usually local.

The incubation period in natural cases of VS-FCV infection in cats exposed in hospitals is usually 1-5 days; in the home environment it may extent up to 12 days (Hurley and Sykes, 2003). The disease appears to be more severe in adults than kittens. Vaccination did not protect cats against field infections (Hurley and Sykes, 2003), although experimentally, some protection has been shown (Pedersen et al., 2000; Brunet et al., 2005). It is unknown whether this is due to inherent characteristics of hypervirulent strains or simply that vaccine-"susceptible" strains are unlikely to cause outbreaks since vaccination is so widely practiced (Hurley, 2006; Pedersen et al., 2000).

In contrast to the common strains, VS-FCV causes systemic disease characterized by severe systemic inflammatory response syndrome, disseminated intravascular coagulation, multi-organ failure, and commonly death. Mortality is up to 67% (Foley et al., 2006).

The clinical signs of this form of disease are variable. The initial findings are frequently typical of a severe acute upper respiratory tract disease. Characteristic signs are cutaneous oedema and ulcerative lesions on the skin and paws (Hurley and Sykes, 2003). Oedema is located mainly on the head and limbs. Crusted lesions, ulcers and alopecia can be seen on the nose, lips, and ears, around the eyes and on the footpads. Some cats are jaundiced (e.g. due to hepatic necrosis, pancreatitis); some may show severe respiratory distress (e.g. due to pulmonary oedema). Thromboembolism and coagulopathy caused by DIC may be observed including petechiae, ecchymoses, epistaxis or bloody faeces (Hurley and Sykes, 2003; Coyne et al., 2006b).

3.6 Diagnosis

Because of the asymptomatic carrier phase, caution should be taken when interpreting any FCV positive result because of the poor correlation between the presence of virus and clinical signs (Sykes et al., 1998).

The diagnosis of VS-FCV relies on clinical signs, high contagiousness and high mortality rate and isolation of the same strain from blood of several diseased cats, assessed by sequencing of hypervariable regions of the capsid gene.

3.6.1 Methods of viral detection

3.6.1.1 Detection of nucleic acid

Conventional, nested and real-time reverse-transcriptase PCR (RT-PCR) assays have been developed to detect FCV RNA in conjunctival and oral swabs, blood, cutaneous scrapings or lung tissue, depending on the clinical form and the outcome of the disease. Diagnostic sensitivity of RT-PCR may depend on both the primers used and the detected strain, because of the high variability of the viral genome (Helps et al., 2002; Marsilio et al., 2005; Scansen et al., 2004; Sykes et al. 1998; Wilhelm & Truyen, 2006). Therefore, molecular assays should be optimised using a large panel of strains to minimize false negative results. Multiplex PCR have also been developed in order to detect at the same time both FHV-1 and FCV (Sykes et al., 2001), but such assays may be less sensitive.

As well as having the potential to diagnose FCV infection, RT-PCR provides the means of identifying uniquely the virus strain and has proven useful in molecular epidemiology and outbreak investigations. However, consistent genetic markers associated with virulence, specifically hypervirulent strains are as yet unavailable (Foley et al., 2006; Abd-Eldaim et al., 2005; Ossiboff et al., 2007).

3.6.1.2 Virus isolation

Virus isolation (VI) is a useful method for detecting FCV infection; it indicates the presence of replicating virus and has the advantage of being less sensitive to the effect of strain variation than RT-PCR. FCV replicates in cell lines of feline origin; its rapid growth in tissue culture may compromise identification of concurrent herpesvirus (Pedersen, 1987).

Virus can be isolated from nasal, conjunctival or oro-pharyngeal swabs (Gaskell & Dawson, 1998), but VI may fail due to small numbers of virions in the sample, virus inactivation during transit, or to the presence of antibodies in extracellular fluids that prevent virus replication *in vitro.* The chance of successful VI can be maximised if swabs from both conjunctiva and oropharynx are collected (Marsilio et al., 2005).

3.6.2 Serology

FCV antibodies can be detected by virus neutralization or ELISA (Lappin et al., 2002). The seroprevalence is generally high in cat populations due to natural infection and vaccination. Consequently, the presence of specific antibodies is not useful to diagnose infection (Gaskell & Dawson, 1998).

Levels of VNA can be used to predict whether a cat is protected or not, but must be interpreted properly, as false negative results may be obtained if VNA do not cross-react with the laboratory strains used in the test. In addition, titres may appear higher when homologous rather than heterologous virus-antibody pairs are used. When the strain used is not defined, it makes interpretation of the results difficult (Scott & Geissinger 1997, 1999; Dawson et al., 2001; Gore et al., 2006).

3.7 Disease management

3.7.1 Treatment of acute upper respiratory tract disease

Cats severely affected by FCV infection need intensive nursing care and supportive therapy. The resolution of dehydration and restoration of electrolyte and acid-base disturbances preferably by intravenous fluid administration is required in cats with severe clinical signs. Food intake is extremely important. Many cats with FCV infection do not eat mainly because of pyrexia and/or ulcers in the oral cavity, sometimes also because of their loss of smell due to nasal congestion. Non-steroidal anti-inflammatory drugs can be used to decrease fever and oral pain. Food may be blended to cause less pain when eating, should be highly palatable, and may be warmed up to increase the smell. If the cat is not eating for more than three days, placement of a feeding tube and enteral nutrition is indicated. At the clinician's discretion, antibiotics should be given to cats with severe disease and suspected secondary bacterial infection. Broad-spectrum antibiotics should be chosen. It is crucial to use antibiotics with good penetration in the respiratory tract and/or oral cavity.

If there is nasal discharge, this should be cleaned away several times a day with physiological saline solution, and ointment should be applied locally. If there is a mucous nasal discharge, drugs with mucolytic effects (e.g. bromhexine) may be helpful, and nebulisation with saline can be used to combat dehydration of the airways.

3.7.2 Antiviral therapy of acute upper respiratory disease

Most antivirals used in veterinary medicine only inhibit replication of DNA viruses or retroviruses, and treatment of FCV infections has not entered clinical practice. Ribavirin is one of the few antiviral agents able to inhibit FCV replication *in vitro*. However, it appears to be very toxic to cats and side effects have precluded its systemic use (Povey, 1978).

Feline interferon-ω (licensed for the treatment of canine parvovirus and feline leukaemia virus infections in some European countries) has been shown to inhibit FCV replication *in vitro* (Fulton & Burge, 1985; Mochizuki et al., 1994, Taira et al., 2005). Controlled field studies, however, are not available.

3.7.3 Treatment of VS-FCV infection

In outbreaks of VS-FCV, severely affected cats have been treated with intensive care supportive treatment (e.g. fluid therapy, antibiotics) plus steroids and interferon, and clinical improvement was reported anecdotally. However, controlled clinical studies have not been published so specific treatment for the disease is not currently known (Hurley, 2006).

3.7.4 Treatment of chronic stomatitis

Several modalities have been used to treat chronic ulceroproliferative stomatitis although controlled studies are lacking. Recommended options depend on the disease severity and stage and include antibiotics plus rigorous dental cleaning, corticosteroids and/or other immunosuppressant or immunomodulatory drugs (gold salts, clorambucil, thalidomide and cyclosporine; White et al., 1992; Addie et al., 2003; Vercelli et al., 2006) and full teeth extractions (Hennet, 1994). Recently, anecdotal and clinical case reports have suggested the use of both feline interferon-ω and human interferons to treat cats with chronic stomatitis associated with FCV shedding, by intra-lesional or combined systemic plus intra-lesional application (Southerden & Gorrel, 2007). Again, controlled studies on using that treatment are currently not available.

3.8 General recommendations on vaccine type and vaccination protocol

FCV infection is ubiquitous and may induce severe disease. ABCD therefore recommends that all healthy cats should be vaccinated against FCV. Although vaccination provides good protection against acute oral and upper respiratory tract disease in most cases, it does not prevent cats from becoming infected and from shedding FCV afterwards (Radford et al.,

2006). In addition, there is currently no vaccine available in Europe that protects against all FCV field strains.

Currently, FCV is combined with FHV-1 in divalent vaccines (only in some countries) or, more commonly, with additional other antigens. Both modified live and inactivated parenteral vaccines are available. Modified live intranasal vaccines are no longer available in Europe, but still current in the USA.

FCV vaccines provide protection mainly by inducing humoral immunity (VN antibodies). As the virus can mutate quickly, field strains could evolve resistance to any vaccine-induced immune response, particularly if a vaccine is used for a prolonged period of time in the population (Lauritzen et al., 1997). Though there is little published evidence available, ongoing field studies support this hypothesis. They are conducted to obtain more information about the strains circulating in Europe, and vaccine companies are seeking to identify newer strains that provide wider cross protection (Poulet et al., 2005). The most commonly used vaccine strains of FCV are: F9, which is the oldest, isolated in the 1950s, FCV 255, and two new strains G1 and 431 (Poulet et al., 2000; Poulet et al., 2005). Some vaccine companies do not state the strain of virus used in their vaccine.

In the absence of compelling published data, it is difficult to make a general recommendation about which vaccine strain to use. However, if disease is occurring in fully vaccinated cats that are housed in groups, then changing to a different vaccine antigen may offer advantages.

The impact of vaccination on the shedding of field viruses is controversial, with one study showing a moderate reduction (Poulet et al., 2005) whilst others show that vaccination might actually extend the period of virus shedding post infection. (Dawson et al., 1991; Pedersen & Hawkins, 1995). Live parenteral FCV vaccine strains can be shed, although it seems rare. (Pedersen & Hawkins, 1995; Radford et al., 1997, 2000, 2001; Coyne et al., 2007).

Live vaccines retain some pathogenic potential and may induce disease if administered incorrectly, e.g. when accidentally aerosolised or spilled on the skin and ingested (Dawson et al., 1993; Pedersen & Hawkins, 1995; Radford et al., 1997; Radford et al., 2000). However, this appears to be a rare event.

Cats that have recovered from caliciviral disease are probably not protected for life against further episodes of disease, particularly those caused by different strains. Therefore, vaccination of recovered, healthy cats is generally recommended, even in situations where FCV is endemic.

The value of serological tests in predicting protection is limited, because antibodies to the calicivirus strain used in a laboratory test may not necessarily protect against the strains that the cat will subsequently be exposed to in the field.

3.8.1 Primary vaccination course

ABCD recommends that all kittens should be vaccinated against FCV. Because MDA can interfere with the response to vaccination, the primary course of vaccination is usually started at around nine weeks of age, although some vaccines are licensed for use at an earlier age. Kittens should receive a second vaccination two to four weeks later, but not earlier than at twelve weeks of age. This protocol has been developed to ensure optimal protection. However, due to a longer persistence of MDA some kittens may fail to respond to this protocol (Dawson et al. 2001). Therefore, in high-risk situations, particularly where FCV has been shown to cause disease in vaccinated kittens, a third vaccination at 16 weeks should be considered. We recommend using the same brand for the entire primary vaccination course.

Older cats of uncertain FCV vaccination status should also receive two injections with an interval of two to four weeks, using vaccines containing the same virus strains. This applies even if the vaccine contains modified live virus.

3.8.2 Booster vaccinations

The issue of recommended intervals between boosters is still controversial. However, based on positive study results published by several independent groups, ABCD recommends that boosters should be given at triennial intervals to protect individual cats against FCV field infections. These cats are in low-risk situations, mainly indoor-only cats with little or no contact to others. However, owners should be made aware that as time since the last vaccination increases, the degree of protection will decrease. Cats in crowded high-risk situations (e.g. boarding catteries) should be revaccinated at yearly intervals. For other cats, an informed decision should be made on the basis of a risk-benefit analysis.

The ABCD recommends a single injection if the interval since the last vaccination is less than three years. If the interval exceeds three years, two vaccinations would ensure optimal protection. Boosters using FCV vaccines from different manufacturers are acceptable.

The ABCD appreciates that single-component FCV vaccines are currently unavailable. Annual boosters that protect against other antigens may in practice entail more frequent boosters than triennially.

3.9 Disease control in specific situations

3.9.1 Shelters

FCV is often a problem in cat shelters. Management to limit or even prevent virus transmission is as important as vaccination in control. Shelter design and management should be aimed at avoiding cross infection of cats. Cats should be housed individually unless they are known to originate from the same household.

If acute respiratory disease occurs in a shelter, identification of the agent involved (with differentiation of FCV from FHV-1, *Chlamydophila felis*, *Bordetella bronchiseptica*, and *Mycoplasma* spp.) may be useful in deciding on the appropriate preventative measures. In case of an FCV outbreak, it should be considered that FCV can persist in the environment for about one month and is resistant to many common disinfectants. Effective substances include sodium hypochlorite (5% bleach diluted at 1:32), potassium peroxy-monosulfate, chlorine dioxide and commercial products that have been approved for their virucidal activity.

New healthy cats should be vaccinated as soon as possible. Modified live virus vaccines are preferred in shelters because of the earlier onset of protection.

3.9.2 Breeding catteries

FCV can be a major problem for cat breeders. Infection most often appears as upper respiratory disease in young kittens, typically at around 4-8 weeks as MDA wanes. Disease in such young kittens can be severe and frequently involves all the kittens in the litter; some kittens may die. Vaccination of the queen will not prevent virus shedding, but may be beneficial in ensuring that the kittens benefit from higher levels of MDA through the colostrum and milk, providing protection for the first month or so of life.

Booster vaccinations should take place prior to mating. Vaccination during pregnancy is not recommended. Modified live virus vaccines are not licensed for use in pregnant cats and if considered at all, an inactivated vaccine must be used.

Queens should kitten in isolation, and in order to avoid the risk of exposure to potential carrier cats, the litter should not mix with other cats until it has been fully vaccinated. Early vaccination should be considered for litters from queens that had infected litters previously or for which there is concern of infection. The earliest age for which FCV vaccines are licensed is six weeks, but vaccination may be considered even earlier in kittens deemed to be at risk.

When levels of MDA may be too low to protect, vaccination should be repeated every two weeks until the primary vaccination course is concluded at twelve weeks.

When all other control strategies have failed, early weaning into isolation from around four weeks of age is an alternative approach to protect kittens against infection from their mothers.

3.9.3 Vaccination of immunocompromised cats

Vaccines cannot generate optimum protection in animals with compromised immune function, such as deficient nutrition, genetic and acquired, viral immunodeficiencies, systemic disease, concurrent administration of immunosuppressive drugs and environmental stress. Efforts should be made to protect immunocompromised cats from exposure to infectious agents and to correct these conditions prior to vaccination; if this cannot be assured, vaccination should be performed nevertheless and repeated after the animal has fully recovered. Based on safety considerations, ABCD recommends inactivated vaccines in these circumstances. Modified live FCV vaccines should not be used in immunocompromised individuals, as the failure to control replication of the vaccine virus could lead to clinical signs.

3.9.3.1 FIV positive cats

Vaccination of FIV-infected cats is controversial. FIV-infected cats are capable of mounting immune responses to administered antigens except during the terminal phase of infection, but also primary immune responses may be delayed or diminished (Dawson et al., 1991; Reubel et al., 1994; Foley et al., 2003). FCV vaccination was less effective in cats shortly after experimental infection with FIV, as compared to uninfected cats, and vaccination might enhance long-term shedding of FCV (Dawson et al., 1991).

Immune stimulation of FIV-infected lymphocytes *in vitro* promotes FIV replication. *In vivo*, vaccination of chronically infected cats with a synthetic peptide was associated with a decrease in the CD4+/CD8+ ratio (Lehmann et al.1992; Reubel et al., 1994). Therefore, a potential trade-off to protection from FCV-related disease is the progression of FIV infection as a result of increased virus production. Thus, only FIV cats with a high risk of exposure to infectious agents that are clinically healthy or in a stable medical condition should be vaccinated, and only killed vaccines used.

3.9.3.2 FeLV-positive cats

FeLV-infected cats should be kept indoors and isolated, to avoid exposure to FCV, but also to diminish the likelihood of retrovirus transmission to other cats. Asymptomatic FeLV-infected cats should be vaccinated against FCV. Although there is no evidence that FeLV-infected cats are at increased risk of vaccine-induced disease from residual virulence of modified-life virus vaccines, killed vaccines are preferable. FeLV-infected cats may not mount adequate immune responses to rabies vaccines and perhaps neither to other vaccines. Protection of FeLV-infected cats may therefore not be comparable to that achieved in uninfected cats, and more frequent vaccination should be considered.

3.9.3.3 Chronic disease

Exceptions from the general rule to vaccinate only healthy animals apply for cats with chronic illness, where vaccination may sometimes be necessary. Manufacturers evaluate vaccine safety and efficacy in healthy animals and accordingly, vaccines are labelled for use in healthy animals only. Nonetheless, cats with stable chronic conditions such as 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.

In cats with chronic stomatitis and FCV infection, administration of modified live FCV vaccine is best avoided (Pedersen et al., 1995).

3.9.3.4 Cats receiving corticosteroids or other immunosuppressive drugs

In cats under corticosteroid treatment, vaccination should be considered carefully. Depending on dosage and duration, corticosteroids may cause functional suppression of cell-mediated immune responses in particular. In dogs, corticosteroids do not hamper effective immunization if given for short periods of time at low to moderate doses (Nara et al., 1979), but the effect of corticosteroids on vaccine efficacy in cats is not known. Hence the use of corticosteroids and/or other immunosuppressants at the time of vaccination should be avoided.

3.10 References

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