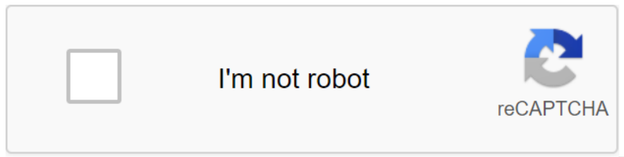

Continue

Calicivirus treatment pdf

Edited December, 2017 Cat Kalicivirus Infection Guidelines were first published in J Feline Med Surg 2009; 11: 538-546 and updated in J Feline Med Surg 2015; 17:570-582; this update of the head of vaccination was sanctioned by Alan Radford. Watch a video interview with Professor Alan Radford, Liverpool Click here to download the file Feline Calicivirus cat calicivirus virus (FCV) is a highly contagious pathogen with widespread prevalence in the cat population. It belongs to the family Caliciviridae, genus Vesivirus; caliciviruses include important human pathogens (such as the Noroulka virus, one of the most common causes of infectious gastroenteritis in humans) and animals, including the European brown hare syndrome virus and the rabbit haemorrhagic virus (Green at al., 2000). Calicivirus particles are hexagonal or star-shaped and show a hollow in the form of a cup in electron microscopic drugs; The name comes from the Greek calix, meaning cup or cup (Figure 1). Fig. 1. False colored electronic micrograph of calicivirus particles (virits) Virus has a small single-stringed RNA genome of positive (messenger) polarity, allowing it to develop rapidly. It is enclosed by several copies of the main capsid protein, the most variable, probably, immunodominant protein domain (mainly aimed at the host's immune response; Tohya et al., 1997; Radford et al., 1999; Geisler et al., 2002). Despite this variability, there is enough antigenic overlap between isolates to classify viruses as a single serotype (Povey, 1974; Povi and Ingersoll, 1975). However, there are antigenic differences between FCV isolates (table 1), which creates significant difficulties in trying to maximize cross-protection of vaccines. Genetically the majority of FCV belong to one diverse genotype (Glenn et al., 1999, Geissler et al., 1997); the second genotype was recently described in Japan (Ohe et al., 2006). Fig. 2. At high magnity on the surface of the virion are visible eponimic cutlets (caliki). © Marian K. Horzinek Table 1. The results of cross-tests for neutralization between potassicivirus strains (vertical) and corresponding hyperimmune goat antisers (horizontal). Symbols: - neutralization in both directions; 1/2 - one-way neutralization; Lack of neutralization; Kalunda et al., 1975. Epidemiology There are no known reservoirs or alternative hosts for FCV, and people are not susceptible to infection. In addition to having proper calicivirus dogs, FCV-like viruses have been isolated from dogs (Hashimoto et al,1999; Roerink et al., 1999; Martella et al., 2002; di Martino et al., 2009). Their role in epidemiology in both uncertain (Binns et al., 2000; Helps et al., 2005), but probably likely The virus is spilled mainly with oral and nasal secretions in acute disease. On recovery, many cats continue to shed, most of them at least 30 days after infection, several over several years (Wardley, 1976). A small proportion of cats may be resistant to infection (Coyne et al., 2006a) probably depends on the factors of the host strain and the virus. Cat calicivirus infection is widespread in the general cat population. The prevalence is generally proportional to the number of cats in the household and is highest when large groups are placed together. It is low in domestic cats, hosts in small groups (10%; Wardley et al., 1974), but in colonies or shelters, data between 25% and 40% were reported (Wardley et al., 1974; Coutts et al., 1994; Bannash and Foley, 2005; Helps et al., 2005). The prevalence in individual colonies is variable, ranging from low (Radford et al., 2001; Coyne et al., 2006a) to a maximum (50-90%) Values (Radford et al., 2003; Coyne et al., 2006a). Infection usually occurs in direct contact with secretions in acutely infected and carrying cats (Wardley, 1977). However, the virus survives in the environment and remains infectious for one month on dry surfaces at room temperature and even longer in colder conditions (Doultree et al., 1999; Duizer et al., 2004; Clay et al., 2006). Therefore, indirect transmission can occur, especially within the immediate boundaries of the nursery, where secretions can contaminate cells, feeding and cleaning products or staff. The virus can also remain infectious in feces fleas for up to 8 days, and kittens can be experimentally infected with FCV when exposed to infected fleas or their faeces (Mencke et al., 2009). Cat pathogenesis can be infected with FCV through the nasal, oral or conjunctival route. Orofarix is the main place of replication. Transitor vichemia occurs 3-4 days after infection, after which the virus is detected in many other tissues. The virus causes necrosis of epithelial cells: bubbles, usually on the edge of the tongue, are transferred to ulcers; in the affected areas, the dermis penetrates neutrophils. Healing takes place within two to three weeks (Gaskell et al., 2006). FCV may be less likely to resent other tissues, leading to pneumonia (focus alveolite, progresses in areas of acute exudative pneumonia, and then to proliferative, interstitial pneumonia) and chromote (rice. 3.) Acute synovitis was observed with thickening of the synovial membrane and elevated synovial fluid (Dawson et al., 1994). The pathogenesis of chromium syndrome is not clear; immune complexes are thought to play a role (Bennett et al., 1989). The virus can also be isolated from affected joints (Dawson et al., 1994). Fig. 3. Chromium syndrome calicivirus infection ©Uwe Truyen Pathogeneze systemic disease caused by FCV (VS-FCV) differs from the typical picture described above. These strains cause widespread vasculitis, involving multiple organs and death of up to two-thirds of infected cats (Pedersen et al., 2000; Hurley and 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 components with an immune mediator, as well as environmental and control factors (Hurley, 2006). Recently, these virulent strains have been shown to grow at a faster rate in cell culture (Ossiboff et al., 2007). After recovering from an acute illness, most cats do not clear the infection within 30 days; minority sheds the virus for much longer, perhaps for life. In these healthy FCV carriers, the virus can be localized in the epithelium of the tonsils. However, tonsillectomy does not eliminate the host state, suggesting that the virus is also found elsewhere. It is believed that the evolution of the variable capsid protein allows FCV to avoid the host's immune response and persist in carrier cats (Johnson, 1992; Kreutz et al., 1998; Radford et al., 1998; Coyne et al., 2007). Passive immunity immunity acquired by maternal-derived colosseal antibodies (MDA) is essential for protection during the first weeks of life and may interfere with vaccination. There are only a few data on the scale and life expectancy of FCV MDA in cats. In general, their levels are higher and last longer than that of feline herpes (FHV-1). In the pilot study, the average half-term MDA was defined as 15 days, and their preservation as 10-14 weeks (Johnson and Povey, 1983). However, in a field study, 20% of kittens aged just six weeks had no detectable antibodies against the widely used vaccine strain (Dawson et al., 2001). The active immune response virus neutralizing antibodies (VNA) appear about seven days after infection (Kahn et al., 1975). In general, antibody are higher than those of FHV infection, and their levels are well correlated with protection against homologous problems (Povey and Ingersoll, 1975). There is a significant degree of antigenic variability among FCV strains, but it has been concluded from studies in cross-reactivity vitro that FCVs belong to the same serotype (Povey, 1974). Pre-infection with a single strain can significantly reduce acute clinical signs when exposed to a heterological strain, and in some cases oral shedding may be reduced (Povey and Ingersoll, 1975; Knowles, etc., 1991). Overall, the level of heterology protection will depend on the strains of the virus involved. Cats can also be protected in the absence of detectable VNA (Knowles et al., 1991; Poulet et al., 2005), offering a role for other immune mechanisms: indeed, cellular reactions have been demonstrated in vaccinated cats (Tham and Studdert, 1987). In addition, antibodies IgG and IgA were showcased in during the infection (Knowles et al., 1991), although their importance in protection is unknown. Clinical signs of FCV infection can cause acute oral and upper respiratory signs, but have also been associated with chronic stomatitis, which can be immune-mediated. Recently, a new syndrome, virulent systemic cat calicivirus (VS-FCV) disease, was described. Fig. 4. Mild epithelial defects after a surge in calicivirus aftah ©Sann-Yvonne Michalevich Acute oral and upper respiratory tract Clinical findings may differ, depending on the virulence of the FCV strain concerned, on the age of the affected cats and on the factors of the husband. While in some cases subclinal infection, in many others, there is a typical lincal ulcer syndrome (Figure 4, 5.) and a relatively mild acute respiratory disease. The more severe signs may resemble respiratory diseases caused by FHV-1. Fig. 5. Characteristic language card forms of lesions due to infection of FCV ©Marian C. Horzinek Acute oral and upper respiratory disease signs are mostly seen in kittens. The incubation period is 2 to 10 days (Hurley and Sykes, 2003). Oral ulcers, sneezing and serous discharge from the nose are the main signs (Gaskell et al., 2006). There is also a fever. Anorexia, sometimes accompanied by hypershipsyl due to oral erosion located mainly on the tongue, is usually much more noticeable than the signs of rhinitis (rice. They are usually resolved after a few days. In some severe cases, pneumonia, which occurs dyspnoe, cough, fever and depression, especially in small kittens, may occur. Fig. 6. Hyper-life due to oral ulcers after FCV infection. Chronic STOMATitis FCV can be isolated from almost all cats with chronic lymphoplasmac cell gingivitis/stomatitis complex, and many cats tested positive for PCR (Dowers et al., 2010, Belgard et al., 2010). It has been suggested to be an immune mediatic reaction to FCV (and possibly others) oral antigens and is characterized by severe proliferative/ulcerative faucitis. However, the disease has not been replicated experimentally (Knowles et al,1991), and the exact role of FCV remains unclear. Although FIV and Bartonella were also offered to be involved, the evidence is limited (Glaus et al., 1997; Dowers et al., 2010; Belgard et al., 2010); consensus they are not related to the disease in most cats. Chromote syndrome Acute transient lameness (rice. 3.) fever may be associated with FCV infection (Pedersen et al., 1983; Ter Wee et al., 1997) and vaccination. In natural infection, this occurs a few days or weeks after acute oral or respiratory symptoms (Pedersen et al., 1983; Bennett et al., 1989). Virulent systemic cat calicivirus (VS-FCV) infection Outbreaks of high virulent and often fatal FCV infection in domestic have been described in the United States and Europe Europe Et al., 2000; Coyne et al., 2006b; Reynolds et al. 2009). One outbreak has also been described in exotic ferids in captivity in the US (Harrison et al, 2007). The disease was called haemorrhagic fever (Pedersen et al., 2000) and high virulent cat calicivirus disease (Schorr-Evans et al., 2003). Strains of the virus-causing virus are most commonly referred to as virulent systemic cat calicivirus (VS-FCV); however, the term is somewhat misleading, as all FCV infections are systemic - but the disease caused by other FCV strains is usually local. The incubation period for natural cases of VS-FCV infection in cats exposed in hospitals is usually 1-5 days; in the home environment it can hundredths of 12 days (Hurley and Sykes, 2003). The disease appears to be more severe in adults than kittens. Vaccination did not protect cats from field infections (Hurley and Sykes, 2003), although some protection was experimentally shown (Pedersen et al., 2000; Brunet et al., 2005). It is not known whether this is due to the characteristics inherent in hypervirulent strains or simply that the strains of the vaccine receptive are unlikely to arrive for outbreaks, as vaccination is widely practiced (Pedersen et al., 2000; Hurley, 2006). Fig. 7. VS-FCV virulent systemic disease ©Tim Gruffydd-Jones Unlike common strains, VS-FCV causes systemic disease characterized by severe systemic inflammatory response syndrome, spread by intravascular coagulation (CIC), multi-chord insufficiency, and usually death. Mortality is up to 67% (Foley et al., 2006). Clinical signs of this form of the disease are variables. Initial findings are often typical of severe acute upper respiratory tract disease. Characteristic signs are skin swelling and ulcerative lesions of the skin and paws (Hurley and Sykes, 2003). Swelling is located mainly on the head and limbs (figure. 7.). Cortical lesions, ulcers and alopecia can be seen on the nose, lips and ears around the eyes (figure. 8.) and on the footrests (Figure 9). Some cats jaundice (for example, because of necrosis of the liver, pancreatitis); some of them may manifest severe respiratory distress (e.g. due to pulmonary edema). Thromboembolism and coagulopathy caused by DIC can be observed including petechiae, ecchimos, epistaxis or bloody faeces (Hurley and Sykes, 2003; Coyne et al,2006b). Fig. 8. Solid lesions and sores due to vs-FCV infection ©Tim Gruffydd-Jones Fig. 9. Viirulent systemic calicivirus disease, excoriations of the paws ©Uwe Truyen Other clinical photos FCV has also been involved in other diseases such as polyps and cystitis; there is no evidence of these associations (Klose et al., 2010; Larson et al., 2011). Diagnosis due to the imptomatic media phase, as well as the fact that viruses in live vaccines can sometimes be spilled after vaccination et al., 2011), caution should be exercised when any positive FCV result due to poor correlation between the presence of the virus and clinical signs (Sykes et al., 1998). The VS-FCV diagnosis is based on clinical traits, high contagion and high mortality rates and isolation of the same strain from the blood of several diseased cats assessed by the sequencing of hypervariate areas of the capsid gene. Detection of nucleic acid Conventional, embedded and real-time reverse transcriptase PCR (RT-PCR) tests have been developed to detect FCV RNA in conjunctival and oral tampons, blood, skin scraping or lung tissue, depending on the clinical form and outcome of the disease. The diagnostic sensitivity of RT-PCR may depend on both the primers used and the detected strain due to the high variability of the viral genome; therefore, molecular analyses should be optimized using a large panel of strains to minimize false negative results. The PCR multiplex has also been designed to detect at the same time as FHV-1 and FCV (Sykes et al., 2001), but such analyses may be less sensitive. In addition to the possibility of diagnosing FCV infection, RT-PCR provides tools for unambiguous identification of the virus strain and has proven to be useful in molecular epidemiology and outbreak research. However, consistent genetic markers associated with virulence, in particular hypervirulant strains, are not yet available (Abd-Eldaim et al., 2005; Foley et al., 2006; Ossiboff et al., 2007). Isolation Virus Isolation Virus (VI) is a useful method for detecting FCV infection; it indicates the presence of virus replication and has the advantage of being less sensitive to the effect of strain change than RT-PCR. FCV replicates in the cellular lines of feline origin; its rapid growth in tissue culture may jeopardize the detection of simultaneous herpesvirus (Pedersen, 1987). The virus may be isolated from nasal, conjunctival or orco-pharyngal tampons (Gaskell and Dawson, 1998), but VI may fail due to the small number of virions in the sample, inactivation of the virus during transit or the presence of antibodies in extracellular fluids that prevent replication of the virus in the test tube. The chances of success vi can be maximum if tampons are collected from both conjunctiva and orofarix (Marsilio et al., 2005). Serology FCV antibodies can be detected by neutralizing the virus or ELISA (Lappin et al., 2002). Seropation is generally high in cat populations due to natural infection and vaccination. Consequently, the presence of specific antibodies is not helpful in diagnosing infection (Gaskell and Dawson, 1998; Grade I EBM). VNA levels can be used to predict whether a cat is protected or not, but should be interpreted properly as false negative results be obtained if VNA does not cross-respond with the laboratory strains used in the test. In addition, the credits may appear higher when homologous rather than heterological pairs of virus-antibody virus-antibody When the strain used is not defined, it makes it difficult to interpret the results (Scott and Geissinger 1997, 1999; Dawson et al., 2001; Gore et al., 2006). Treatment of acute diseases of the upper respiratory tract Cats seriously affected by FCV infection need intensive care and supportive therapy. Resolution of dehydration and recovery of electrolyte and acid-based disorders preferably by the introduction of intravenous fluid is required in cats with severe clinical signs. Food intake is extremely important. Many cats with FCV infection do not eat mainly due to pyrexia and/or mouth ulcers, sometimes also due to their loss of sense of smell due to nasal congestion. Non-steroidal anti-inflammatory drugs can be used to reduce temperature and oral pain. Food can be mixed to cause less pain while eating, should be very tasty, and can be warmed up to increase the smell. If the cat does not eat for more than three days, the placement of the tube for feeding and enteral nutrition is indicated. At the doctor's discretion, antibiotics should be given to cats with serious illnesses and suspicion of secondary bacterial infection. Broad-spectrum antibiotics should be selected. It is very important to use antibiotics with good penetration into the airways and/or mouth. If there is a discharge from the nose, it should be cleaned several times a day with saline solution, and the ointment should be applied locally. In the nasal mucosa can be useful drugs with mucolytic under the influence (e.g. bromgexin), and to combat dehydration of the airways can be used nebulization with saline solution. Antiviral therapy for acute upper respiratory tract disease Most antiviral drugs used in veterinary medicine only inhibit the replication of DNA viruses or retroviruses, and the treatment of FCV infections has not entered into clinical practice. Ribavirin is one of the few antiviral drugs capable of inhibiting FCV replication in vitro. However, it appears to be very toxic to cats and side effects have ruled out its systemic use (Povey, 1978; EBM Grade III). It has been shown that cat interferon -z (licensed to treat parvovirus and cat-related infections of leukemia in some European countries) suppresses the replication of FCV in vitro (Fulton and Burge, 1985; Mochizuki et al., 1994; Taira et al., 2005; EBM IV). However, there are no controlled field studies. There is some suggestion that strains may vary in their sensitivity to interferon (Ohe et al., 2008). Treatment vs-FCV infection In VS-FCV outbreaks, seriously affected cats have been treated with intensive care therapy (e.g. fluid therapy, antibiotics) plus steroids and interferon, clinical improvement has been reported anecdotally. However, controlled clinical trials have not been published; therefore the specific treatment of this disease is not currently known (Hurley, 2006; Ebm Ebm Treatment of chronic stomatitis Full description of the treatment of chronic stomatitis goes beyond these guidelines. However, several methods have been used to treat chronic ulcerative-ferative stomatitis, although controlled studies are lacking. The recommended options depend on the severity of the disease and stage and include antibiotics plus severe tooth brushing, corticosteroids and/or other immunosuppressants or immunomodulatory drugs (golden salts, clorambucil, thalidomide and cyclosporine; White et al., 1992; Addie et al., 2003; Vercelli et al., 2006; IV class EBM) and complete tooth extraction (Hennet, 1994; EBM Grade III). Anecdotal and clinical case reports have suggested the use of both cat interferon-i and human interferons to treat cats with chronic stomatitis associated with FCV shedding through intravertebral or combined systemic plus intra-tralesional applications (Southerden and Gorrel, 2007). Again, controlled studies on the use of this treatment are not currently available. Topical oral interferon has been shown to lead to statistical improvement, in clinical evaluations, but this improvement is no different from cats receiving steroids alone (Hennet et al., 2011; EBM IV). General recommendations on the type of vaccine and the FCV vaccination protocol infection are ubiquitous and can cause severe illness. THE ABCD recommends that all healthy cats should be vaccinated against FCV. Although vaccination provides good protection against acute oral and upper respiratory tract diseases in most cases, it does not prevent cats from contracting and shedding FCV afterwards (Radford et al., 2006). In addition, there is currently no vaccine that equally protects against all fcV field strains. Currently, FCV is combined with FHV-1 in divalent vaccines (only in some countries) or, more often, with other antigens. Both modified live and inactivated parenteral vaccines are available. Modified live intranasal vaccines are no longer available in Europe, but are still relevant in the United States. FCV vaccines provide protection mainly by inducing humoral immunity (VN antibodies). Because the virus can mutate rapidly, strains on the ground can develop resistance to any immune response caused by the vaccine, especially if the vaccine is used for a long period of time in the population (Lauritzen et al., 1997). While there are some studies that support this hypothesis (Addie et al., 2008), the evidence of FCV avoiding vaccine-induced immunity at the population level is not conclusive (Porter et al., 2008). Such studies are being carried out to find out more about strains circulating in Europe, and vaccine companies are seeking to identify new strains that wider cross-protection (Poulet et al., 2005). The most commonly used vaccine strains are F9, which is the oldest, isolated in the 1950s, FCV 255, and two new strains of G1 and (Poole et al., 2000; Poulet et al., 2005). Recently, one manufacturer introduced a hypervirulant strain into its vaccine in the United States (Huang et al., 2010), and a Japanese research team developed a triple strain vaccine (Masubuchi et al., 2010); however, at the time of writing (2015) they are not available in Europe. Some vaccine companies will not knead the strain of the virus used in their vaccine. In the absence of conclusive published data, it is difficult to make a general recommendation on which strain of vaccine or strain to use. However, if the disease occurs in fully vaccinated cats that are housed in groups, then switching to another antigen vaccine may offer benefits. The impact of vaccination on the shedding of field viruses is debatable, with some studies showing a moderate reduction (Poulet et al., 2005; Jas et al., 2009), while others show that vaccination may actually prolong the virus spill period after infection (Dawson et al., 1991; Pedersen and Hawkins, 1995). It is possible to shed live parenteral and intranasal strains of the FCV vaccine, although this seems rare (Pedersen and Hawkins, 1995; Radford et al., 1997, 2000, 2001; Coyne et al., 2007; Ruch-Gallie et al., 2011). Vaccines in real time retain some pathogenic potential and can cause diseases when administered correctly, for example, when they are accidentally sprayed or spilled into the skin and enter the body (Dawson et al., 1993; Pedersen and Hawkins, 1995; Radford et al., 1997 and 2000). However, this seems to be a rare event. Cats that have recovered from caliciviral diseases are probably not protected for life from further episodes of disease, especially caused by different strains. Thus, vaccination of recovered, healthy cats is usually recommended, even in situations where FCV is endemic. The importance of serological tests in predicting protection is limited because antibodies to the calicivirus strain used in laboratory tests may not necessarily protect against strains that the cat will subsequently be exposed to in the field. Primary Kitten Vaccination Course: ABCD recommends that all kittens should be vaccinated against FCV. Because MDA can interfere with vaccination response, the primary vaccination course usually starts around the age of about nine weeks, although some vaccines are licensed for use at an earlier age. Kittens should get a second vaccination in two to four weeks, but not earlier than at the age of twelve weeks. This protocol is designed to provide optimal protection. However, due to the longer preservation of MDA, some kittens may not respond to this protocol (Dawson et al., 2001; Grade I EBM). Therefore, in high-risk situations, especially where FCV has been shown to cause disease in vaccinated a third vaccination should be considered within 16 weeks. After the course of primary vaccination kittens all cats receive an additional additional additional dose between the ages of 10 and 16 months: this will provide an adequate vaccine of induced immunity for cats that may not have adequately responded to the main course. We recommend using the same brand for the entire primary vaccination course. Older cats with uncertain FCV vaccination status should also receive two injections at intervals of two to four weeks, and pulse after a year using vaccines containing the same strains of the virus. This applies even if the vaccine contains a modified live virus. The revaccination of the issue of recommended intervals between accelerators is still controversial. However, based on positive results from a study published by several independent groups, the ABCD recommends that boosters should be given at three-year intervals to protect individual cats from field infections of FCV (EBM class II) for cats in low-risk situations, mostly only indoors of cats with little or no contact with others. However, owners should be aware that over time, from the moment the last vaccination increases, the degree of clinical protection decreases. However, cats in overcrowded high-risk situations (such as boarding houses) should be revaccinated after a year. For other cats, an informed decision should be made based on risk and benefit analysis. ABCD recommends one injection if the interval from the last vaccination is no more than three years. If the interval exceeds three years, two vaccinations will provide optimal protection. Accelerators using FCV vaccines from various manufacturers are acceptable. ABCD appreciates that single-component FCV vaccines are currently unavailable. Annual boosters that protect against other antigens can in practice result in more frequent boosters than three years. Disease control in specific situations OF FCV shelters is often a problem in cat shelters. The Office to Limit or Even Prevent Transmission of the Virus is just as important as vaccination control. Housing design and management should be aimed at preventing cross-infection of cats. Cats should be placed individually if they are known to come from the same household. Dogs and cats should be placed separately, and flea control should be implemented to minimize the risk of transmission of FCV and other diseases. If an acute respiratory disease occurs in a shelter, identifying the agent involved (with FCV differentiation from FHV-1, Chlamydia felis, Bordetella bronchiseptics, and Mycoplasma spp.) may be helpful in deciding on appropriate preventive measures. In the case of an FCV outbreak, it should be taken into account that FCV can persist in the environment for approximately one month and is resistant to many common disinfectants. Substances include sodium hypochlorite (5% bleach diluted in 1:32), pecox-monosulfate potassium, chlorine dioxide and commercial products that have been approved for their vivucidal activity. New healthy cats should be vaccinated as soon as Modified live viral vaccines are preferred in shelters because of the earlier onset of protection. Breeding FCV kennels can be a serious problem for cat breeders. The infection most often appears as an upper respiratory tract disease in young kittens, usually around 4-8 weeks as the MDA weakens. The disease in such small kittens can be severe and often includes all kittens in litter; some kittens may die. Vaccination of the queen will not prevent the spill of the virus, but may be helpful in ensuring that kittens benefit from higher levels of MDA through colossutivo and milk, providing protection for the first month or so of life. Revaccinations should occur before mating. Vaccination during pregnancy is not recommended. Modified live virus vaccines are not licensed to use in pregnant cats, and if they are considered at all, an inactivated vaccine should be used. The queens need a kitten in isolation, and in order to avoid the risk of exposure to potential carrier cats, litter should not mix with other cats until it has been fully vaccinated. Early vaccination should be considered for litters of queens who have infected litters previously or for whom there is a concern for infection. The earliest age for which FCV vaccines are licensed is six weeks, but vaccination can be considered even earlier in kittens deemed at risk. When MDA levels may be too low for protection, vaccination should be repeated every two weeks until the primary vaccination course is completed in twelve weeks. When all other control strategies have failed, early laundering in isolation from about four weeks of age is an alternative approach to protect kittens from infection from their mothers. Vaccination of immune-weakened cats vaccines cannot generate optimal protection in animals with weakened immune function, such as malnutrition, genetic and acquired, viral immunodeficiency, systemic diseases, simultaneous preparation of immunosuppressive drugs and environmental stress. Efforts should be made to protect cats with weakened immunity from exposure to infectious agents and to correct these conditions prior to vaccination; If this cannot be guaranteed, vaccination should be carried out however 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 failure to control the replication of the vaccine virus can lead to clinical signs. FIV positive cat vaccination of FIV-infected cats is controversial. Infected FIV cats are able to mount reactions to injected antigens, except for the terminal phase of infection, but also primary immune responses may be delayed or reduced (Dawson et al., 1991; Reubel et al., 1994; Foley et al., 2003; EBM Grade III). FCV vaccination was less in cats shortly after the experimental FIV infection, compared to uninfected cats, and vaccination can increase long-term spilling 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 synthetic peptide was associated with a decrease in CD4/CD8 ratio (Lehmann et al., 1992; Reubel et al., 1994). Thus, a potential trade-off to protect against FCV-related diseases 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 who are clinically healthy or in a stable health condition should be vaccinated, and only killed vaccines are used. FeLV-positive feLV-infected cats should be kept indoors and isolated to avoid exposure to FCV, but also reduce the likelihood of transmitting retrovirus to other cats. Asymptomatic FeLV-infected cats should be vaccinated against FCV. While there is no evidence that feLV-infected cats are at increased risk of vaccine-induced disease from residual virulence modified live viral vaccines, slain vaccines are preferable. FeLV-infected cats cannot mount adequate immune responses to rabies vaccines and possibly none on other vaccines. Therefore, the protection of feLV infected cats may not be comparable to the protection of uninfected cats, and consideration should be given to more frequent vaccination. Chronic diseases Exceptions from the general vaccination rule only healthy animals apply to cats with chronic diseases, where vaccination may sometimes be necessary. Manufacturers assess the safety and efficacy of vaccines in healthy animals and, accordingly, vaccines are labeled for use only in healthy animals. However, cats with stable chronic diseases such as kidney disease, diabetes or hyperthyroidism should receive vaccines at the same frequency as healthy cats. In contrast, cats with acute disease, weakening, or high fever should not be vaccinated. In cats with chronic stomatitis and fcV infection, the administration of a modified live FCV vaccine is best avoided (EBM Class IV). Cats receiving corticosteroids or other immunosuppressive drugs In cats with corticosteroid treatment, vaccination should be carefully considered. Depending on the dosage and duration, corticosteroids can cause functional suppression of cellular mediation of the immune response in particular. In dogs, corticosteroids do not prevent effective immunization if given for short periods of time in low to moderate doses (Nara et al., 1979), but the effect of corticosteroids on Vaccines in cats are not known. Therefore, the use of corticosteroids and/or other immunosuppressants during vaccination should be avoided. References Abd-Eldaim M, Potgieter L, Kennedy M (2005): Genetic analysis of feline caliciviruses associated with Disease. J Vet Diagn Invest 17, 420-429. Addie D, Poulet H, Golder MC, McDonald M, Brunet S, Thibault JC, Hosie MJ (2008): The ability of antibodies to two new strains of caliciviral vaccine to neutralize feline calicivirus isolates from the UK. Vet Rec 163 (12), 355-357. Eddie DD, Radford A, Yam PS, Taylor DJ (2003): The cessation of feline calicivirus shedding coincides with the resolution of chronic gingivomatitis in a cat. J Small Anim Practical 44 (4), 172-176. Bannasch MJ, Foley JE (2005): Epidemiological assessment of several respiratory pathogens in cats in animal shelters. J Feline Med Surg 7, 109-119. Belgard S, Truyen U, Thibault JC, Sauter-Louis C, Hartmann K (2010): Prevalence of feline calicivirus, feline immunodeficiency virus, cat leukemia virus, feline herpesvirus and bartonella henselae in cats with chronic gingaivostom. Burl Munch Tierarztl Wochenschr 123 (9-10), 369-376. Bennett D, Gaskell RM, Mills A, Knowles J, Carter S, McArdle F (1989): Detection of feline calicivirus antigens in the joints of infected cats. Vet Rec 124 (13), 329-332. Binns SH, Dawson S, Speakman AJ, Cuevas LE, Hart CA, Gaskell CJ, Morgan KL, Gaskell RM (2000): Study of feline upper respiratory tract diseases citing prevalence and risk factors for contracting feline calcivirus and feline herpesvirus. J Feline Med Surg 2, 123-133. Brunet S, Jas D, David F, Bublot M, Poulet H (2005): Cat calicivirus: vaccinations against virulent strains. In comparative and emerging viral infections of dogs and cats. Conference of the European Society of Veterinary Virology 2005, Liverpool. Clay S, Maherchandani S, Malik YS, Goyal SM (2006): Survival on unusual pemiths of feline calicivirus, surrogate norovirus. Am J Infect Control 34, 41-43. Coutts AJ, Dawson S, Willoughby K, Gaskell RM (1994): Isolation of feline respiratory viruses from clinically healthy cats in UK cat shows. Vet Rec 135, 555-556. Coyne KP, Dawson S, Radford AD, Cripps PJ, Porter CJ, McCracken CM, Gaskell RM (2006a): Long-term analysis of the prevalence of feline calicivirus and viral shedding patterns in naturally infected colonies of domestic cats. Vet Microbiol 118 (1-2), 12-25. Coyne KP, Gaskell RM, Dawson S, Porter CJ, Radford AD (2007): Evolutionary mechanisms for the conservation and diversification of calicivirus in endemic populations of natural hosts. J Virol 81 (4), 1961-1971. Coyne KP, Jones BRD, Cypar A, Chantrey J, Porter CJ, Barber PJ, Dawson S, Gaskell RM, Radford AD (2006b): Deadly outbreak of cat-related disease in cats. Vet Rec 158, 544-550. Dawson S, Bennett D, Carter SD, Bennett M, Meanger J, Turner PC, Carter MJ, Milton I, Gaskell RM (1994): Acute cat arthritis associated with feline calicivirus infection. Res Vet Sci 56, 133-143. Dawson S, McArdle F, Bennett M, Carter M, IP, Тернер P, Meanger J, Гаскелл RM (1993): Ввод Ввод cat calicivirus isolates from various clinical groups tests to neutralize viruses. Vet Rek 133 (1), 13-17. Dawson S, Smith NR, Bennett M, Gaskell RM, McCracken CM, Brown A, Gaskell CJ (1991): The effect of the primary stage of feline immunodeficiency viral infection on subsequent feline calicivirus vaccination and challenge in cats. AIDS 5, 747-750. Dawson S, Willoughby K, Gaskell RM, Woog G, Chalmers WCK (2001): Field study to assess the impact of cat herpesvirus vaccination, feline calcivirus and feline panleuation virus in 6-week-old kittens. J Feline Med Surg 3, 17-22. Di Martino B, Di Rocco C, Sesi C, Marsilio F (2009): Characteristics of the feline calicivirus strain are isolated from the dog's fecal sample. Vet Microbiol 139 (1-2), 52-57. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA (1999): Cat Calicivirus Inactivation, Norwalk Virus Surrogate. J Hosp Infect 41, 51-57. Dowers KL, Hawley JR, Brewer MM, Morris AK, Radecki SV, Lappin MR (2010): Association of Bartonella Species, Cat Calicivirus and Cat Herpesvirus 1 Infection Gingivostomitis in Cats. J Feline Med Surg 12(4), 314-321. Duizer E, Bijkerk P, Rockx B, De Groot A, Twisk F, Koopmans M (2004): Calicivirus inactivation. Appl Environ Microbiol 70, 4538-4543. Foley J, Hurley K, Pesavento PA, Poland A, Pedersen NC (2006): Virulent systemic feline calicivirus infection: local cytokines modulation and viral mutant contributions. J Feline Med Surg 8, 55-61. Foley JE, Leutenegger CM, Dumler JS, Pedersen NC, Madigan JE (2003): Evidence of modulated immune response to Anaplasma phagocytophila sensu lato in cats with FIV-induced immunosuppression. Comp Immunol Micribiol Infect 26, 103-113. Fulton RW, Burge LJ (1985): Susceptibility of cat herpesvirus 1 and feline calicivirus to feline interferon and recombinant interferons of human leukocytes. Antimicrobial Agents Chemother 28 (5), 698-699. Gaskell R, Dawson S (1998): Cat respiratory disease. In: Infectious Diseases Of Dogs and Cats, Green CE (Ed), WB Saunders Company, Philadelphia (USA), 97-106. Gaskell RM, Dawson S, Radford AD (2006): Cat Respiratory Disease. In: Infectious Diseases Dog and Cat, Green CE (Ed), Saunders Elsevier, 145-154. Geissler K, Schneider K, Platzer G, Truyen B, Caaden OR, Truyen U (1997): Genetic and antigenic heterogeneity among feline calicivirus isolates from various manifestations of the disease. Virus Res 48 (2), 193-206. Geissler K, Schneider K., Truyen W (2002): Mapping neutralizing and neutralizing epitopes on the capsid protein of the cat's calicivirus. J Vet Med B Infect Dis Vet Public Health 49(1), 55-60. Glaus T, Hofmann-Lehmann R, Greene C, Glaus B, Wolfensberger C, Lutz H (1997): The seropation of bartonella henselae infection and the correlation with the status of the disease in cats in Switzerland. J Wedge 35 (11), 2883-2885. Гленн M, Рэдфорд AD, Тернер PC, PC, M, Lowery D, DeSilver DA, Meanger J, Baulch-Brown C, Bennett M, Gaskell RM (1999): Nucleotide sequence of British and Australian cat calicivirus isolates (FCV) and phylogenetic analysis of FCVs. Vet Microbiol 67(3), 175-193. Gore TC, Lakshmanan N, Williams JR, Jirjis FF, Chester ST, Duncan KL, Coyne MJ, Lam MA, Sterner FJ (2006): Three-year immunity period in cats after vaccination against feline rhinotracheitis virus, feline caliciumvirus, and feline panleukion virus. Vet zer 7 (3), 213-222. Green KY, Ando T, Balayan MS, Berke T, Clarke IN, Estes MK, Matson DO, Nakata S, Neill JD, Studdert MJ, Thiel HJ (2000): Taxonomics. J Infect Dis 181 Suppl 2, S 322-330. HarrisonTM, Sikarskie J, Kruger J, Wise A, Mullaney TP, Kiupel M, Maes RK (2007): Systemic epidemic of calicivirus in captivity of exotic felids. J Wildl Med 38 (2), 292-299. Hashimoto M, Roerink F, Tohya Y, Mochizuki M (1999): Genetic analysis of the RNA gene polymerase caliciviruses from dogs and cats. J Vet Med Sci 61, 603-608. Helps CR, Light P, Damhuis A, Bjornehammar U, Bolt D, Brodida C, Chabanne L, Egberink H, Ferrand G, Fontbonn A, Pennisi MG, Gruffydd-Jones T, Gunn-Moore D, Hartmann K, Lutz H, Malandain E, Bridge K, Stengel C, Harbour DA, Graat EA (2005): Factors associated with upper respiratory diseases caused by feline herpes, feline caliciavirus, Chlamyd Vet Rec 156, 669-673. Hennet P (1994): Results of periodontal and extraction in cats with gingivotomitis. In Proceedings of the World Veterinary Dental Congress, 49. Hennet PR, Camy GA, McGahie DM, Albouy MV (2011): Comparative efficiency of recombinant feline omega interferon in fire-resistant cases of calicivirus-positive cats with caudal stomatitis: randomized, multicenter, controlled, double-blind study in 39 cats. J Feline Med Surg 13(8), 577-587. Juan C, Hess J, Gill M, Hustead D (2010): A double strain of feline calicivirus vaccine stimulates broader cross-neutralizing antibodies than a single-purpose vaccine and reduces clinical signs in vaccinated cats when challenged with a homologous feline calicivirus strain associated with virulent systemic disease. J Feline Med Surg 12(2), 129-137. Hurley KF (2006): Virulent calicivirus infection in cats. In the Proceedings of the American College of Veterinary Internal Medicine Congress 2006. Hurley KF, Sykes ES (2003): Cat Calcivirus Update: New Trends. Veterinarian Wedge North Am Small Anim Prak 33 (4), 759-772. Jas D, Aeberle C, Lacombe V, Guiot AL, Poulet H (2009): The onset of immunity in kittens after vaccination is not an adjuvant vaccine against feline panleucopy, cat calcivirus and cat herpes. Vet J 182 (1), 86-93. Johnson RP (1992): Antigenic change of cat calicivirus during инфекции. Can J Vet Res 56, 326-330. Джонсон Джонсон Povey RC (1983): Transmission and reduction of maternal antibody to feline calicivirus. Can Vet J 24, 6. Kahn DE, Hoover EA, Bittle JL (1975): Induction of immunity to feline calcivirus diseases. Infect immunol 11, 1003. Calunda M, Lee KM, Holmes DF, Gillespie JH (1975): Serological classification of feline caliciviruses by neutralizing plaque reduction and immunodiffusion. Am J Vet Res 36 (4 Pt.1), 353-356. Klose TC, MacPhail CM, Schultheiss PC, Rosychuk RA, Hawley JR, Lappin MR (2010): Prevalence of individual infectious agents in inflammatory auditory and nasopharynx polyps from customer-owned cats. J Feline Med Surg 12(10), 769-774. Knowles JO, McArdle F, Dawson S, Carter SD, Gaskell CJ, Gaskell RM (1991): Research on the role of feline calicivirus in chronic stomatitis in cats. Vet Microbiol 27, 205-219. Kreutz LC, Johnson RP, Seal BS (1998): Phenotypic and genotypic variation of feline calicivirus during a permanent cat infection. Vet Microbiol 59, 229-236. Lappin MR, Andrews J, Simpson D, Jensen WA (2002): Using serological tests to predict resistance to feline herpesvirus 1, feline calicivirus and cat parvovirus infection in cats. J Am Vet Med Assoc 220 (1), 38-42. Larson J, Kruger JM, Wise AG, Kaneene JB, Miller R, Fitzgerald SD, Kiupel M, Maes RK (2011): Nest case control of cat potassiumvirium research, oral transportation, and serum-neutralized antibodies in cats with idiopathic cystitis. J Vet Intern Med 25(2), 199-205. Lauritzen A, Jarrett O, Sabara M (1997): Serological analysis of feline calicivirus isolates from the United States and the United Kingdom. Vet Microbiol 56, 55-63. Lehmann R, Von Beust B, Niederer E, Condrau MA, Fierz W, Aubert A, Ackley CD, Cooper MD, Tompkins MB, Lutz H (1992): Immunization-induced reduction in CDA coefficient:CD8 in cats experimentally infected with cat immunodeficiency virus. Veterinary Immunopatol 35, 199-214. Marsilio F, Martino BD, Decaro N, Buonavoglia C (2005): A novel of nested PCR to diagnose calcivirus infections in cats. Vet Microbiol 105, 1-7. Martella V, Pratelli A, Gentile M, Buonavoglia D, Decaro N, Fiorente P, Buonavoglia C (2002): Analysis of the gene capsid protein of cat calicivirus isolated from the dog. Vet Microbiol 85, 315-322. Masubuchi K, Wakatsuki A, Iwamoto K, Takahashi T, Kokubu T, Shimizu M (2010): The immunological and genetic characteristics of cat caliciviruses used in the development of a new trivalent inactivated vaccine in Japan. J Vet Med Sci 72(9), 1189-1194. Mencke N, Vobis M, Mehlhorn H, D Haese J, Rehagen M, Mangold-Gehring S, Truyen U (2009): Transmission of feline calicivirus through cat flea (Ctenocephalides felis). Parasitol Res 105 (1), 185-189. Mochizuki M, Nakatani H, Yoshida M (1994): Inhibiting the effects of recombinant feline to replicate cat enteropathogenic viruses in vitro. Vet Microbiol 39 (1-2), 145-152. Nara Nara Krakowka S, Powers TE (1979): The effect of prednisone on the development of the immune response to the dog distemper virus in beagle puppies. Am J Vet Res 40(12), 1742-1747. Ohe K, Sakai S, Sunaga F, Murakami M, Kiuchi A, Fukuyama M, Furuhata K, Hara M, Soma T, Ishikawa Y, Taneno A (2006): Cat Calcivirus Detection (FCV) from vaccinated cats and phylogenetic analysis of its capsid genes. Vet Res Commun 30(3), 293-305. Ohe K, Takahashi T, Hara D, Hara M (2008): Sensitivity FCV recombinant feline interferon (rFeIFN). Vet Res Commun 32(2), 167-174. Ossiboff RJ, Sheh A, Shotton J, Pesavento PA, Parker JS (2007): Cat caliciviruses (FCVs), isolated from cats with virulent system diseases, have in vitro phenotypes other than other FCV isolates. J Gen Virol 88, 506-527. Pedersen NC (1987): Cat calicivirus. In: Viral carnivores infections. Appel MJ (Ed), Elsevier Science Publishers BV, New York, 339-346. Pedersen NC, Elliott JB, Glasgow A, Poland A, Keel K (2000): Isolated epizootic haemorrhagic fever in cats caused by novel and highly virulent strain of feline calicivirus. Vet Microbiol 73, 281-300. Pedersen NC, Hawkins KF (1995): Mechanisms to conserve acute and chronic feline calicivirus infections in vaccination settings. Vet Microbiol 47 (1-2), 141-156. Pedersen NC, Laliberte L, Ackman S (1983): Transitional feverish lame kitten syndrome caused by two different strains of feline calicivirus. Cat Practice 13, 26-35. Porter CJ, Radford AD, Gaskell RM, Ryvar R, Coyne KP, Pinchbeck GL, Dawson S (2008): Comparison of the ability of feline calicivirus (FCV) vaccines to neutralize the panel of the current UK FCV isolates. J Feline Med Surg 10(1), 32-40. Poulet H, Brunet S, Leroy V, Chappuis G (2005): Immunization with a combination of two additional strains of feline calicivirus causes extensive cross-protection against heterological problems. Vet Microbiol 106 (1-2), 17-31. Pule H, Brunet S, Soulier M, Leroy V, Goutebroze S, Chappuis G (2000): Comparison between acute oral/respiratory and chronic stomatitis/gingivitis isolates feline calicivirus: pathogenicity, antigenic profile and cross-neutralization studies. Arch Virol 145 (2), 243-261. Povey RC (1974): Serological relationship between feline caliciviruses. Infect Immun 10, 1307-1314. Povey RC (1978): Effect of perorically introduced ribavirin on experimental feline calicivirus infection in cats. Am J Vet Res 39(8), 1337-1341. Povey C, Ingersoll J (1975): Cross-protection among feline calories. Infect Immun 11, 877-885. Radford AD, Bennett M, McArdle F, Dawson S, Turner PC, Glenn MA, Gaskell RM (1997): Using a sequence of cat calicivirus analysis (FCV) hypervariate region in the epidemiological investigation of FCV related diseases and vaccine failures. Vaccine 15, 1451-1458. Radford AD, Dawson S, HP, Porter CJ, Galskell RM (2006): (2006): for the next generation of feline calicivirus vaccines. Vet Microbiol 117 (1), 14-18. Radford AD, Dawson S, Ryvar R, Coyne K, Johnson DR, Cox MB, Acke EF, Eddie DD, Gaskell RM (2003): High genetic diversity of the immunodominant area of the feline calicivirus capsid gene in endemic cat colonies. Viral genes 27, 145-155. Radford AD, Dawson S, Wharmby C, Ryvar R, Gaskell RM (2000): Comparison of serological and sequencing-based techniques for entering feline calicivirus isolates from vaccine failures. Vet Rec 146, 117-123. Radford AD, Sommerville L, Ryvar R, Cox MB, Johnson DR, Dawson S, Gaskell RM (2001): Endemic cat disease infection with feline caliciavirus is closely associated with isolate used in live hardened vaccines. Vaccine 19, 4358-4362. Radford AD, Turner PC, Bennett M, McArdle F, Dawson S, Glenn MA, Williams RA, Gaskell RM (1998): The quasi-speciality evolution of the hypervariate area of the cat's calicivirus capsid gene in cellular culture and in permanently infected cats. J Gen Virol 79, 1-10. Radford AD, Willoughby K, Dawson S, McCracken C, Gaskell RM (1999): The capsid gene of the feline calicivirus contains linear B-cell epitopes in variable and preserved regions. J Virol 73, 8496-8502. Reubel GH, Dean GA, George JW, Barlough JE, Pedersen NC (1994): The effects of accidental infections and immune activation on disease progression in expermimentally feline immunodeficiency virus-infected cats. J Ak Immun Defic Syndrome 7, 1003-1015. Reynolds BS, Poulet H, Pingret JL, Jas D, Brunet S, Lemeter C, Etievant M, Boucraut-Baralon C (2009): nosocomial cat cellivirus outbreak associated with virulent systemic disease in France. J Feline Med Surg 11 (8), 633-644. Roerink F, Hashimoto M, Tohya Y, Mochizuki M (1999): Genetic analysis of calicivirus dogs: evidence of a new hoard of animal caliciviruses. Vet Microbiol 69, 69-72. Ruch-Gallie RA, Veir JK, Hawley JR, Lappin MR (2011): Results of molecular diagnostic tests aimed at feline herpesvirus-1 and feline calicivirus in adult cats, are

administered with modified live vaccines. J Feline Med Surg 13 (8), 541-545. Schorr-Evans EM, Poland A, Johnson MY, Pedersen NC (2003): Epizootic highly virulent feline calicivirus disease at a hospital in New England. J Feline Med Surg 5(4), 217-226. Scott FW, Geissinger CM (1997): Immunity duration in cats vaccinated with inactivated feline panleukopia, herpesvirus and calcivirus vaccine. Cat Practice 25, 12-19. Scott FW, Geissinger CM (1999): Long-term immunity in cats vaccinated with inactivated trivalent vaccine. Am J Vet Res 60, 652-658. Southerden P, Gorrel C (2007): Treatment of a case of fire-resistant feline chronic gingivomatitis with feline recombinant omega interferon. J Small Anim Practical 48 (2), 104-106. JE, Allen JL, Studdert Vice President, Browning GF (2001): Detection of feline calicivirus, feline herpesvirus 1 and and psittaci slimy tampons multiplex RT-PCR/ PCR. Vet Microbiol 81 (2), 95-108. Sykes JE, Studdert VP, Browning GF (1998): Detection and differentiation of cat calicivirus strains in conjunctic tampons RT-PCR hypervariate area of the capsid protein gene. Arch Virol 147 (7), 1321-1334. Taira O, Suzuki M, Takeuchi Y, Aramaki Y, Sakurai I, Watanabe T, Motokawa K, Arai S, Sato H, Maehara N (2005): Expression of cat interferon-alpha subtypes in Eshchia coli, as well as their antiviral activity and animal species. J Vet Med Sci 67, 543-545. TerWee T, Lauritzen A, Sabara M, Dreier KJ, Kokjohn K (1997): Comparison of primary signs caused by experimental exposure to pneumotrophic or lame strain of cat calicivirus. Vet Microbiol 56, 33-45. Tham KM, Studdert MJ (1987): Antibodies and cellular immune responses to feline calicivirus after inactivated vaccine and call. J VetMed B 34, 640-654. Tohya Y, Yokoyama N, Maeda K, Kawaguchi Y, Mikami T (1997): Mapping antigenic sites involved in neutralizing the capsid protein of the feline calicivirus. J Gen Virol 78, 303-305. Vercelli A, Raviri G, Cornegliani N (2006): Use of oral cyclosporine for the treatment of feline dermatosis: retrospective analysis of 23 cases. Vet Dermatol 17, 201-206. Wardley RC (1976): Feline calicivirus carrier status. Exploring the relationship between the host and the virus. Arch Virol 52, 243-249. Wardley RC (1977): Clinical disease and discharge patterns associated with three different strains of feline caliciviruses. Res Vet Sci 23, 7-14. Wardley RC, Gaskell RM, Povey RC (1974): Cat respiratory viruses are common in clinically healthy cats. J Small Anime Practice 15, 579-586. White SD, Rosychuk RA, Janik TA, Denerolle P, Schultheiss P (1992): Plasma stomatitis-pharyngitis in cats: 40 cases (1973-1991). J Am Vet Med Assoc 200(9), 1377-1380. Further reading Helps CR, Light P, Tasker S, Harbor D (2002): Melting curve analysis of feline calicivirus isolates detected in real-time reverse transcription of PCR. J Virol Methods 106 (2), 241-244. McCann KB, Lee A, Wan J, Roginski H, Coventry MJ (2003): The effect of cattle lactoferrin and lactoferricin B on the ability of feline calicivirus (norovirus surrogate) and poliovirus to infect cellular cultures. J Appl Microbiol 95 (5), 1026-1033. Pesavento PA, MacLachlan NJ, Dillard-Telm L, Grant CK, Hurley KF (2004): Pathological, immunohystochemical and electron microscopic results in natural virulent systemic cat calicivirus infection in cats. Veterinarian Pathol 41, 257-263. Scansen BA, Wise AG, Kruger JM, Venta PJ, Maes RK (2004): Evaluation of the p30 gene based on real-time polymerase reverse transcriptase to detect feline caliciviruses. J Vet Intern Med 18 (1), 135-138. Pines SV, Belliot G, KO, Onwudiwe O, Green KY (2005): Cat calicivirus VP2 is essential for production of infectious vignons. J Virol 79, 4012-4024. Wilhelm S, Truyen U (2006): In real time, the reverse transcription of polymerase chain reaction analysis to detect a wide range of cat calcivirus isolates. J Virol Methods 133 (1), 105-108. 105-108.