Feline calicivirus infection
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**Virus**

Feline calicivirus (FCV) is a highly contagious pathogen with a widespread distribution in the feline population. It belongs to the Caliciviridae family, genus Vesivirus; caliciviruses include 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).

Calicivirus particles are hexagonal or star-shaped and show cup-shaped depressions in electron microscopic preparations; the name is derived from Greek calyx meaning cup or goblet (Fig. 1).
The virus has a small single-stranded RNA genome of positive (messenger) polarity, which allows it to evolve quickly. It is enclosed by multiple copies of the major capsid protein, the most variable, probably immunodominant protein domain (principally targeted by the host’s immune response; Geissler et al., 2002; Radford et al., 1999; Tohya et al., 1997). Despite this variability, there is sufficient antigenic overlap between isolates to allow classification of the viruses as a single serotype (Povey, 1974; Povey & Ingersoll, 1975). However, antigenic differences exist between FCV isolates (Table 1), 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).

### Epidemiology

There are no known reservoirs or alternative hosts for FCV, and humans are not susceptible to infection. Apart from the existence of a proper canine calicivirus, FCV-like viruses have been isolated from dogs (Hashimoto et al., 1999; Martella et al., 2002; Roerink et al., 1999, di Martino 2009). Their role in the epidemiology in both carnivores is uncertain (Binnis et al., 2000; Helps et al., 2005), but probably not important. 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), 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 a household, and is highest when large groups are housed together. It is low in household cats kept in small groups (~10%; Wardley et al., 1974), but in colonies or shelters, figures between 25% and 40% have been reported (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 (Wardley, 1977). However, the virus survives in the environment and remains infectious for up to one month on dry surfaces at room temperature, and even longer under colder 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. The virus can also remain infectious in flea faeces for up to 8 days, and kittens may be experimentally infected with FCV by contact with infected fleas or their faeces (Mencke et al 2009).

### 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 is 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 (Fig. 3). Acute synovitis with thickening of the synovial membrane and increased synovial fluid have been noted (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...
Immunity

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 only 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).

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.

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.
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 (Fig. 4, 5.) and a relatively mild acute respiratory disease. More severe signs can resemble the respiratory disease caused by FHV-1.

Chronic stomatitis

FCV can be isolated from nearly all cats with the chronic lymphoplasmacytic gingivitis/stomatitis complex, and many cats test positive by PCR (Dowers et al., 2010, Belgard et al., 2010). 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. Although FIV and Bartonella have also been proposed to be involved, the evidence is limited (Glaus et al., 1997, Dowers et al., 2010, Belgard et al., 2010); the consensus is they are not associated with disease in most cats.

Limping syndrome
An acute transient lameness (Fig. 3) 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).

**Virulent systemic feline calicivirus (VS-FCV) infection**

Outbreaks of highly virulent and often lethal FCV infection in domestic cats have been described in the United States and in Europe (Pedersen et al., 2000; Coyne et al., 2006b, Reynolds et al 2009). A single outbreak has also been described in exotic captive felids in the USA (Harrison et al 2007). 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 extend 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 (DIC), 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 (Fig. 7.). Crusted lesions, ulcers and alopecia can be seen on the nose, lips, and ears, around the eyes (Fig. 8.) and on the footpads (Fig. 9). 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).
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; EBM grade III).

Feline interferon-

Antiviral therapy of acute upper respiratory disease

of the airways.

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.

If there is nasal discharge, this should be cleaned away several times a day with physiological saline solution, and antibiotics with good penetration in the respiratory tract and/or oral cavity. 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 minimize false negative results. Multiplex PCR have also been developed in order to detect 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).

Virus isolation

Serology

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).

Levels of VNA can be used to detect 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. Feline interferon-antiviral therapy of acute upper respiratory disease

Disease management

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.

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; EBM grade III). Feline interferon-ω (licensed for the...
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; EBM grade III).

Treatment of chronic stomatitis

A full description of the treatment of chronic stomatitis is beyond the scope of these guidelines. However, 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, chlorambucil, thalidomide and cyclosporine; White et al., 1992; Addie et al., 2003; Vercelli et al., 2006; EBM grade IV) and full teeth extractions (Hennet, 1994; EBM grade III).

Anecdotal and clinical case reports have suggested the use of both feline interferon-α and human interferons to treat cats with chronic stomatitis associated with FCV shedding, by intra-lesional or combined systemic plus intralesional application (Southerdon and Gorrel, 2007). Again, controlled studies on using that treatment are currently not available. Topical oral interferon has been shown to lead to statistical improvement, in clinical scores, but this improvement was not different from cats receiving steroid alone (Hennet et al., 2011; EBM grade IV).

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 that protects equally well against all FCV field strains.

Currently, FCV is combined with FHV-1 in divalent vaccines (only in some countries) or, more commonly, with 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). Although there are some studies that lend support to this hypothesis (Addie et al 2008), the evidence for FCV escaping vaccine-induced immunity at a population level is not convincing (Porter et al 2008). Such studies 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 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).

Recently, one manufacturer has introduced a hypervirulent strain into its vaccine in the USA (Huang et al., 2010), and a Japanese research group has developed a triple strain vaccine (Masubuchi et al., 2010); however, at the time of writing (2015) these are not available in Europe. 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 or strains 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 some studies showing a moderate reduction (Poulet et al., 2005, Jas et al., 2009) whilst others show that vaccination might actually extend the period of virus shedding after infection (Dawson et al., 1991; Pedersen & Hawkins, 1995). Live parenteral and intranasal FCV vaccine strains can be shed, although it seems particularly isolated in the 1950s, FCV 255, and two new strains G1 and 431 (Poulet et al., 2000; Poulet et al., 2005).

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; Pedersen & Hawkins, 1995; Radford et al., 1997, 2000; Covente et al., 2007; Ruch Galle et al., 2011).

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.

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; EBM grade I). 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.

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 (EBM grade II). 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 would 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.
Disease control in specific situations

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. Dogs and cats should be housed separately, and flea control should be implemented to minimize the risk of transmission of FCV and other diseases. If acute respiratory disease occurs in a shelter, identification of the agent involved (with differentiation of FCV from FHV-1, Chlamydia 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.

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 thecolostrum 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 an inactivated vaccine must be used. Queens should be isolated, 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 have 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.

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.

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; EBM grade III). 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.

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-live 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.

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 a modified live FCV vaccine is best avoided (EBM grade IV).

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.

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