FOCAL POINT

⭐ Feline herpesvirus-1 (FHV1) and feline calicivirus (FCV) can be associated with various infections that can be challenging to diagnose and treat.

KEY FACTS

- The carrier state is crucially important in the maintenance and spread of both FHV1 and FCV, p. 167.
- FHV1 and FCV may cause similar clinical signs, although some manifestations appear to be unique to each virus, potentially aiding in diagnosis, p. 169.
- Despite the advent of new diagnostic tests, the diagnosis of FHV1 and FCV is confounded by the relative lack of virus in chronic cases and the inability of tests to differentiate between symptomatic and healthy carrier cats, p. 171.
- Using virus isolation or polymerase chain reaction can aid in assessing prognosis and implementing management strategies, p. 171.
- Indiscriminate use of antiviral compounds is not recommended because they may be toxic or poorly effective, p. 171.

Feline Upper Respiratory Tract Pathogens: Herpesvirus-1 and Calicivirus

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ABSTRACT: Feline herpesvirus-1 and feline calicivirus are common causes of feline upper respiratory tract disease and can cause clinically indistinguishable syndromes. However, they differ in their physical properties, epidemiology, and spectrum of clinical signs; and the differences can affect the diagnosis, prognosis, and management of these infections. The availability and sensitivity of diagnostic tests also differ for each virus. Diagnosis of both infections can be difficult and is best achieved by combining patient history, physical examination, and microbiologic assay results. Treatment is primarily symptomatic. Vaccination does not prevent infection but can help minimize the severity of clinical signs and duration of shedding.

Cats with both acute and chronic upper respiratory tract disease (URTD) are commonly seen in private and referral practices. The general syndrome varies in severity. When severe, it may be associated with considerable morbidity and mortality; it is therefore a concern of breeding and boarding cattery owners. Although the introduction of vaccines targeting feline herpesvirus-1 (FHV1) and feline calicivirus (FCV) in the mid-1970s may have reduced the overall disease severity, the vaccines do not prevent infection and shedding of virus and URTD may still occur. Despite the increasing availability of such molecular diagnostics as polymerase chain reaction (PCR), diagnosis and treatment of many cases of chronic feline URTDs remain frustrating.

This article provides an update on the epidemiology, clinical presentations, and ante-mortem diagnosis and management of FHV1 and FCV, common causes of feline URTD. *Chlamydothila felis* (previously known as feline *Chlamydia psittaci*) is another common cause of feline URTD. In addition, recent research suggests that *Bordetella bronchiseptica* may act as a primary upper respiratory tract pathogen in cats under certain circumstances, particularly in young kittens and in overcrowded, stressful environments; however, the relative importance of *B. bronchiseptica* infection remains unclear. The role of *Mycoplasma* infection in cats with URTD is also poorly defined, and some authors suggest that *Mycoplasma* species mainly act as secondary invaders.
CAUSES
Feline Herpesvirus-1

Feline herpesvirus-1 has a double-stranded DNA genome surrounded by an icosahedral capsid, which is enveloped by a delicate lipid membrane containing several different glycoproteins. The fragility of the envelope limits the survival of FHV1 in the environment to only 18 hours in moist conditions and makes it highly susceptible to destruction by common disinfectants.

Feline herpesvirus-1 isolates worldwide are remarkably similar and belong to a single serotype. However, the existence of attenuated vaccine strains suggests that the virulence of different isolates may vary. FHV1 has recently been divided into four groups based on restriction endonuclease patterns and the presence or absence of a 36-kDa immunogenic protein. The relationship between these groups and virulence has not been determined.

Feline Calicivirus

Feline calicivirus is a nonenveloped, single-stranded RNA virus with a roughly spherical capsid that is studied with cup-shaped depressions. The virus is more resistant in the environment than is FHV1, surviving up to 10 days. It is inactivated by iodine, hypochlorite, and glutaraldehyde but not by quaternary ammonium products, anionic detergents, and ethanol.

Although considerable antigenic heterogeneity occurs among FCV isolates, the degree of cross-reactivity is sufficient for them to be classified as a single serotype. Isolates worldwide apparently fall into a single, diverse group based on nucleotide sequence analysis.

PATHOGENESIS AND EPIDEMIOLOGY

Feline herpesvirus-1 and FCV infections may be acquired by contact with an acutely infected cat, organisms persisting in the environment, or a carrier cat (see the Carrier State section). Cats produce relatively ineffective aerosols that travel a maximum of approximately 4 feet. The chance of contact with an acutely infected cat is increased when many cats are housed together (e.g., breeding and boarding catteries, multiple-cat households). Catteries also provide greater opportunity for indirect transmission through contamination of human hands, feeding and grooming utensils, and other fomites.

Both viruses replicate mainly in the tonsils and respiratory tissue. Viremia may occur with FCV infection and spread to other viscera. Replication of FHV1 is usually restricted to the upper respiratory tract, but generalized disease with viremia can occur in debilitated animals. In addition to the nasal, conjunctival, and oral shedding common to both viruses, FCV is shed in the feces and occasionally in the urine.

Carrier State

The ability of FHV1 and FCV to persist in the tissue of subclinically infected cats is crucial to their pathogenic success. The mechanism of persistence in each case is different and influences the prognosis, diagnosis, and management decisions.

Over 80% (and probably 100%) of cats infected with FHV1 develop latent infections in which the virus enters a nonreplicative state that persists for the life of the animal. In cats, sites of latency include the trigeminal ganglia, optic nerves, optic chiasms, lacrimal gland, cornea, tonsils, and nasal turbinates. Reactivation of virus shedding occurs with or without concurrent clinical signs in approximately 50% of infected cats. It may occur spontaneously or after natural or artificial (corticosteroid-induced) stress. Travel (e.g., to a cat show, stud, or veterinarian) can be sufficient stress to induce reactivation. Parturition and lactation are particularly stressful, allowing spread of virus from a queen to the kittens. Shedding occurs 4 to 11 days after the stress and lasts 1 to 2 weeks. If clinical signs occur, they tend to be less severe than are those after the initial infection. The precise mechanism underlying reactivation is unknown, and the frequency varies among individuals. Humans with frequent recurrences of herpes simplex may have depressed cell-mediated immunity.

In contrast to FHV1, shedding of FCV by persistently infected cats is continuous and not caused by stress. Viral shedding occurs from the oropharynx and fluctuates with time; the level of excretion varies among individuals. Humans with frequent recurrences of herpes simplex may have depressed cell-mediated immunity.

Prevalence

Before the introduction of respiratory viral vaccines,
serologic studies revealed that 50% to 70% of cats had antibodies against FHV1. In contrast, a recent study using a sensitive ELISA found that 97% of client-owned cats had detectable FHV1 IgG and 95% had titers greater than 32. The magnitudes of these titers were higher than were expected from vaccination alone, suggesting that natural exposure to FHV1 occurs commonly and boosts titers induced by vaccination.\textsuperscript{14}

Numerous studies have investigated the prevalence of respiratory viruses in both diseased and asymptomatic cats. Comparisons between studies are difficult because prevalence is highly dependent on the sensitivity and specificity of the assay used for diagnosis, anatomic site sampled (conjunctival versus oropharyngeal), number of cats sampled, seasonal variation of disease, and clinical criteria used to define a case (e.g., acute, chronic). Most studies have used virus isolation. Reports of the prevalence of FHV1 and FCV in cats with URTD have generally ranged from 10% to 34% and 20% to 53%, respectively. In healthy cats, the prevalence of FCV has ranged from 8% of household cats to 24% of show cats.\textsuperscript{15} The prevalence of FHV1 in asymptomatic cats has generally been lower (0% to 1.75%) than that for FCV, reflecting the nature of the carrier state for each virus. However, in one recent study, the prevalence of FHV1 in conjunctival swabs from asymptomatic cats using fluorescent antibody testing or virus isolation was higher (10.9% and 28.3%, respectively) and not significantly different from that in diseased cats.\textsuperscript{16} Using PCR, the prevalence of FHV1 in asymptomatic animals has ranged from 0% to 31%, with the highest prevalence being reported with nested PCR (see the Diagnosis section). The existence of a high prevalence of FCV and possibly FHV1 infection in asymptomatic cats requires consideration when interpreting positive test results in diseased cats.

**CLINICAL FEATURES**

Acute disease caused by FCV and FHV1 infection occurs after an incubation of 2 to 10 days. The nature and severity of signs may depend on the infecting strain, although examination of large numbers of FCV strains has failed to show a clear link between disease manifestation and antigenic and genetic heterogeneity of the FCV capsid protein.\textsuperscript{17,18} The most severe signs tend to occur in very young or elderly debilitated cats. Concurrent immunosuppressive illness or infection with other respiratory pathogens (e.g., *B. bronchiseptica*) and secondary bacterial invaders can also dramatically influence the severity of disease.

**Respiratory and Ocular Disease**

Signs of URTD common to both FHV1 and FCV infections include serous or mucopurulent nasal discharge and sneezing and, less commonly, coughing and/or dyspnea. Both FHV1 and FCV cause conjunctivitis with conjunctival hyperemia, blepharospasm, serous to mucopurulent ocular discharge, and chemosis. Signs may be acute or chronic. Depression, anorexia, hypersalivation, and pyrexia may also be present in cats with acute infections. In young cats, damage to the upper respiratory tract epithelium and osteolysis of the nasal turbinates may cause persistent or recurrent bacterial rhinitis and sinusitis.

Feline herpesvirus-1 may be associated with corneal ulceration and keratitis. Dendritic ulcers are branchlike and result from limited viral replication in the corneal epithelium. The cause of the branching pattern is unclear. Dendritic ulcers are generally considered pathognomonic for herpes simplex virus and FHV1 infection; however, ocular adenovirus infection was recently associated with dendritic ulceration in humans.\textsuperscript{19} Only some infected cats develop keratitis. Researchers suspect an immune-mediated pathogenesis that may be directed against both residual virus antigen in the corneal stroma and the cornea itself. There is evidence that FHV1 may be involved in some cases of uveitis, corneal sequestration (Figure 1), and eosinophilic keratitis.\textsuperscript{20,21}

**Stomatitis**

Ulcerative glossitis is more common and severe in cats with FCV infection but may be associated with FHV1 infection. I have noted severe pharyngitis associated with FHV1 infection without concurrent signs of URTD.

A small proportion of FCV carriers develop chronic lymphoplasmacytic or chronic ulceroproliferative stomatitis (Figure 2), which is often refractory to therapy. Coinfection with FIV may also play a role in the development of chronic stomatitis.\textsuperscript{22}

**Dermatologic Disease**

Skin and nasal ulceration is possible with both FHV1
and FCV infections. FHV1 has been recognized as a cause of ulcerative facial and nasal dermatitis characterized histologically by necrosis, eosinophil infiltration, and intranuclear inclusion bodies, which can mimic the eosinophilic granuloma complex and insect bite hypersensitivity.  

**Lameness and Pyrexia**

Transient lameness and pyrexia with or without concurrent oral or respiratory signs have been reported in association with acute FCV infection and after FCV vaccination. These signs appear to be associated with localization of virus and/or immune complexes in the joints.

**Reproductive and Perinatal Disease**

Feline herpesvirus-1 may cause severe disease in kittens. Neonatal mortalities of up to 60% have been reported. Ocular discharges may be so severe that kittens’ eyes may not open (Figure 3). Pregnant cats infected with FHV1 may abort, resorb fetuses, or give birth to congenitally affected kittens. Attempts to isolate FHV1 from the reproductive tract have yielded mixed results, and it is unclear whether abortion is secondary to the nonspecific debilitating effects of FHV1 infection or results from infection of the reproductive tract. Using PCR, I have detected FHV1 DNA in vaginal swabs from two cats with reproductive disease. FCV has rarely been associated with abortion in cats.

**Other Clinical Signs**

Feline calicivirus has also been associated with enteritis and feline lower urinary tract disease, but its role in these syndromes is not clear. A highly virulent strain of FCV was recently isolated from an outbreak of a systemic hemorrhagic-like fever. The condition was characterized by high mortality, fever, anorexia, ulcerative facial dermatitis, and diffuse cutaneous edema. Several cats developed coagulopathy along with hypoproteinemla and mild hyperbilirubinemia. Necropsy findings included pneumonia, intestinal crypt necrosis, hepato-cellular necrosis, and severe pancreatitis. A systemic vasculitis was suspected.

**DIAGNOSIS**

It is almost impossible to make a diagnosis of FHV1 or FCV infection based on clinical signs of URTD alone, although such signs as keratitis, lameness, or severe oral ulceration may be more suggestive of one virus or the other (Table I). Mixed infections may also occur, further complicating the diagnosis. Vaccination history cannot be used to aid diagnosis because the vaccines do not prevent infection or clinical signs. A history of recurrent bouts of disease, especially when combined with stressful events, can be suggestive of FHV1 infection. When conjunctivitis is present, chlamydiosis should be excluded through appropriate testing or treatment with doxycycline for 3 weeks. Collection of nasal or oropharyngeal swabs for isolation of _B. bronchiseptica_ should be considered. The presence of epistaxis, unilateral nasal discharge, facial deformity, fundic abnormalities, or marked submandibular lymphadenopathy should prompt a search for other causes of URTD (e.g., malignant neoplasia, nasopharyngeal polyps, coagulopathy, foreign bodies, dental disease, fungal infection).

The most reliable microbiologic assay is virus isolation from nasal, conjunctival, and/or oropharyngeal swabs after inoculation of cell monolayers grown in the laboratory. Oropharyngeal swabs are more likely to yield a diagnosis than are conjunctival swabs, and virus must be viable for detection. The fragile envelope of FHV1 may be destroyed while transporting and storing swabs, decreasing its infectivity for cell culture; FCV is more resistant to destruction than is FHV1. Therefore, swabs should be transported on ice in a viral transport medium containing antibiotics to prevent bacterial overgrowth. Rose Bengal stain should not be applied before sample collection because of its light-dependent antiviral properties. Virus isolation is time-consuming...
and can be expensive for large numbers of samples. Because FCV tends to grow more rapidly in culture, its presence in a sample can prevent identification of concurrent FHV1 infection, especially when confirmatory immunofluorescent antibody staining of cell monolayers is not conducted.

Application of fluorescent antibody by direct or indirect techniques to cytology slides has commonly been used to diagnose FHV1 and FCV infections but is less sensitive than is virus isolation and may be associated with false-positive results because of nonspecific fluorescing debris. Prior staining of eyes with fluorescein may also produce false-positive findings.30

Although development of the ELISA has improved the sensitivity of serologic tests for FHV1 and FCV,14 serology is not recommended for routine diagnosis of respiratory viral infections. Acute and convalescent phase samples must be taken 1 to 2 weeks apart for a retrospective diagnosis. Titers induced by vaccination can confound diagnosis. Because titers for FHV1 tend to remain constant in cats with chronic low-grade infections, the paired sera approach loses its value. For cats with FCV, titers can vary, depending on the degree of homology between the infecting virus and the antigen used in the assay.

Amplification of viral DNA fragments using PCR is rapid, exquisitely sensitive, and increasingly available for the detection of FHV1. Development of diagnostic PCR assays for FCV has been slow because of the difficulty in designing assays that amplify nucleic acid from a variety of strains and susceptibility of RNA to degradation. The detection limit of reported assays for FHV1 has ranged from 30 to millions of copies of the FHV1 genome, with sensitivities 25% to 80% higher than those of virus isolation.7,31,32 PCR may be particularly sensitive for detection of FHV1 in vaccinated cats or during reactivation, when the presence of antibody renders the virus uncultivatable. Concern has been

<table>
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<tr>
<td><strong>Features of Feline Herpesvirus-1 and Feline Calicivirus Infections that Influence Diagnosis, Prognosis, and Management</strong></td>
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<table>
<thead>
<tr>
<th>Feature</th>
<th>FHV1</th>
<th>FCV</th>
<th>Implications</th>
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<tbody>
<tr>
<td>Clinical signs</td>
<td>Keratitis, corneal ulceration</td>
<td>Lymphoplasmacytic, stomatitis, severe oral ulceration, lameness</td>
<td>If present, these signs aid in making a diagnosis (mixed infections may occur)</td>
</tr>
<tr>
<td>Environmental survival</td>
<td>&lt;18 hours</td>
<td>&lt;10 days</td>
<td>Resting areas for 24 hours may destroy FHV1 but not FCV; poor specimen handling of culture is more likely to affect diagnosis of FHV1 than that of FCV</td>
</tr>
<tr>
<td>Disinfectant susceptibility</td>
<td>Most common disinfectants</td>
<td>Iodine, hypochlorite, and glutaraldehyde but not QUATs, anionic detergents, and ethanol</td>
<td>Use of such disinfectants as QUATs does not inactivate FCV in the environment</td>
</tr>
<tr>
<td>Chronic shedding pattern</td>
<td>Virus is shed intermittently; may be induced by stress</td>
<td>Virus is shed continuously; level of shedding is not affected by stress</td>
<td>FCV can be detected more readily in carrier cats than can be FHV1; history of intermittent signs, especially after stress, suggests FHV1 infection; FCV may be more of a threat to susceptible cats; minimizing stress only reduces shedding of FHV1</td>
</tr>
<tr>
<td>Duration of carrier state</td>
<td>Lifelong</td>
<td>Variable; most cats eliminate FCV within months to years</td>
<td>May influence prognosis for cats with chronic or recurrent disease</td>
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*FCV = Feline calicivirus; FHV1 = feline herpesvirus-1; QUAT = quaternary ammonium compound.*
raised that highly sensitive assays, particularly those involving nested PCR, may detect asymptomatic shedding or shedding of vaccine virus rather than that associated with disease. Some suggest the use of nested PCR be minimized for routine diagnosis.

False-positive results associated with contamination can occur during sample collection or in the laboratory and thus can be particularly problematic with such assays.

Regardless of the method used to detect the organism, results must always be interpreted in light of the history and clinical signs because viral shedding is not always accompanied by clinical signs. Chronic infections involving immune-mediated complications may be associated with undetectable levels of virus, regardless of the test used. The prevalence of FHV1 infection is much lower in cats with chronic disease than in cats with acute disease.

Nevertheless, attempts to obtain a diagnosis are encouraged. Knowledge of causative organism(s) has prognostic value and can aid management strategies, especially in chronic cases and when multiple cats are involved (Table I).

TREATMENT

Treatment is primarily symptomatic. Broad-spectrum antimicrobials, fluid and nutritional support, nursing care, and airway humidification should be considered. The macrolide azithromycin has been a popular choice for antimicrobial treatment, partly because of its efficacy in human chlamydial infections. Other antibiotics (e.g., oral amoxicillin 22 mg/kg every 12 hours; oral doxycycline 5 mg/kg every 12 hours) are effective against secondary bacterial infection and preferred to avoid development of resistance to the new macrolides. C. felis and B. bronchiseptica are also sensitive to doxycycline; however, in one study, doxycycline failed to eliminate shedding of B. bronchiseptica from experimentally infected cats after clinical recovery.

Controlled, blinded, and randomized studies assessing the efficacy of antivirals in cats with chronic respiratory viral infections are needed, and experience is anecdotal. Topical antiviral preparations (e.g., trifluridine, idoxuridine) are available for FHV1-induced keratitis and, when applied frequently, may help minimize viral replication. These synthetic nucleosides incorporate into viral nucleic acid and inhibit the enzymes of DNA synthesis. Toxic side effects, including hepatopathy (associated with trifluridine), leukopenia, and gastrointestinal disease (associated with idoxuridine), occur when these drugs are given systemically. Because corneal irritation can occur, these drugs should be used with caution. Idoxuridine, which can be obtained from a compounding pharmacy as a 0.1% solution or 0.5% ointment, is less efficacious than is trifluridine in vitro but is also less irritating and less expensive. Some ophthalmologists have suggested topical application of dilute povidone–iodine solution as a safe and inexpensive alternative. t-Lysine and oral and topical recombinant human α-interferon have also been used for refractory FHV1 infec-

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### TABLE II

<table>
<thead>
<tr>
<th>Drug</th>
<th>Route of Administration</th>
<th>Dose Schedule</th>
<th>Comments</th>
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<tr>
<td>Idoxuridine (0.1%)</td>
<td>Topical ophthalmic</td>
<td>1 drop every 2 hours for first 2 days, then every 4 to 6 hours</td>
<td>May be less efficacious than is trifluridine but less irritating and less expensive</td>
</tr>
<tr>
<td>Trifluridine (1%)</td>
<td>Topical ophthalmic</td>
<td>1 drop every 2 hours for first 2 days, then every 4 to 6 hours</td>
<td>Very expensive; most efficacious in vitro</td>
</tr>
<tr>
<td>Povidone–iodine solution (0.3% in normal saline)</td>
<td>Topical ophthalmic</td>
<td>1 drop every 4 to 6 hours</td>
<td>Inexpensive, safe; efficacy unknown</td>
</tr>
<tr>
<td>t-Lysine</td>
<td>Oral</td>
<td>250 mg every 12 hours (adults) or once daily (kittens)</td>
<td>No reported adverse effects; efficacy unknown</td>
</tr>
<tr>
<td>α-Interferon</td>
<td>Oral; topical ophthalmic/intranasal</td>
<td>30 U once daily; 1 drop of 30–50 U/ml solution in artificial tears once daily</td>
<td>No reported adverse effects; efficacy unknown</td>
</tr>
</tbody>
</table>
tions. Neither have been associated with adverse effects, but their efficacy is unknown. Recombinant α-interferon is degraded by proteolytic enzymes before it can be absorbed, but there may be some absorption by the upper gastrointestinal mucosa. Stimulation of oral lymphatic tissue has also been suggested as a mechanism of action. Recombinant α-interferon has been used; but FHV1 is relatively resistant to this drug, and severe toxicity may occur in cats when therapeutic blood levels are achieved. One study that evaluated valacyclovir, which undergoes rapid first-pass hepatic metabolism to acyclovir and L-valine, was prematurely terminated because of the development of severe renal, hepatic, and bone marrow toxicity in treated cats. Use of topical corticosteroids in cats with ocular complications of FHV1 infection is controversial because of the risk for reactivation, worsening of infection, and ulcerative keratitis.

CONTROL

In catteries, preventing the spread of FHV1 and FCV is very important. Elimination of infection is virtually impossible because of the carrier state. Even when latent FHV1 carriers were identified and culled after inducing shedding with corticosteroids, eradication was not achieved. Transmission is reduced by regular disinfection (1:32 bleach:detergent solution), optimal environmental temperature, low relative humidity, and adequate ventilation. Proper quarantine and testing procedures can minimize introduction of the viruses into breeding colonies.

Vaccination does not protect against infection from incoming carrier cats or prevent a cat from becoming a carrier. Modified-live virus vaccine may establish a persistent infection. However, vaccination can reduce disease severity and may shorten the duration of shedding, reducing environmental contamination. Modified-live systemic vaccines probably provide the longest duration of immunity. Signs of URTD may result in cats that inhale an aerosol created during parenteral vaccination or lick the injection site. Killed vaccines do not have the risk of postvaccinal signs of URTD and are the vaccine of choice for pregnant and immunosuppressed cats; their protective effect may last several years. Two doses of a parenteral vaccine given 3 to 4 weeks apart are generally required for adequate protection. Intranasal vaccines are useful against outbreaks in catteries because they provide rapid immunity despite maternal antibodies. Protection may be achieved within 2 to 4 days. Mild to moderate sneezing and ocular discharge often occur within 4 to 7 days after vaccination. Intranasal vaccines were previously believed to protect
against latency, but this has recently been disproved. There is some concern that vaccine pressure may have led to selection of FCV strains that have poor cross-reactivity with the vaccine strain F9. Incorporating additional FCV strains into vaccines has been proposed. Because of the potential risks associated with vaccination, including vaccine-induced fibrosarcomas and induction of immune-mediated disease, as well as evidence suggesting a duration of immunity of more than 3 years after vaccination of cats with inactivated vaccines, the American Association of Feline Practitioners and the Academy of Feline Medicine have published general recommendations that cats undergo a booster 1 year after the primary course, then every 3 years. Nevertheless, the frequency of booster administration remains controversial; and immunization recommendations should be based on the specific lifestyle and history of each cat. Thus some authors have recommended annual boosters for outdoor cats, whereas triennial boosters may be sufficient for those living indoors.

REFERENCES


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**ARTICLE #4 CE TEST**
The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose only the one best answer* to each of the following questions; then mark your answers on the test form inserted in *Compendium*.

1. Which of the following statements best reflects survival of FCV and FHV1 in the environment?
   a. FHV1 is more resistant than is FCV and survives up to 1 week in the environment.
   b. FCV is more resistant than is FHV1 and survives up to 2 years in the environment.
   c. FHV1 is more resistant than is FCV but is susceptible to quaternary ammonium compounds.
   d. FCV is more resistant than is FHV1, and quaternary ammonium compounds are poorly effective.
   e. FHV1 is more resistant than is FCV, and hypochlorite is required to inactivate it.

2. Approximately 2 months after infection, what proportion of cats are carriers of FHV1 and FCV?
   a. approximately 50% and 100%, respectively
   b. All cats infected with both FHV1 and FCV become carriers.
   c. approximately 80% to 100% and 5%, respectively
   d. approximately 5% and 50%, respectively
   e. approximately 80% to 100% and 50%, respectively

3. Which of the following has not been shown to be a site of latency for FHV1?
   a. trigeminal ganglia
   b. cornea
   c. submandibular lymph node
   d. tonsil
   e. nasal turbinate

4. Shedding of FCV in carrier cats is
   a. intermittent and associated with stress or corticosteroid use.
   b. continuous for the rest of the cat’s life.
   c. intermittent and not associated with stress or corticosteroid use.
   d. continuous for a variable period.
   e. intermittent in some cats and continuous in others.

5. Which clinical manifestation(s) is more likely to occur in FCV-infected cats than in FHV1-infected cats?
   a. keratitis and corneal ulceration
   b. abortion
   c. lameness
   d. high neonatal mortality
   e. ulcerative facial and nasal dermatitis, with eosinophil infiltration

6. Currently, antemortem diagnosis of FCV infection is best obtained using which of the following assays?
   a. PCR
   b. serum neutralization
   c. application of fluorescent antibody to smear preparations of affected tissue
   d. cell culture
   e. reverse transcription PCR

7. Which of the following is the first-choice treatment of acute FHV1 infection?
   a. trifluridine because it is the most effective antiviral drug for treating FHV1
   b. idoxuridine because it has fewer side effects than does trifluridine
c.acyclovir because it is effective when given systemically and has minimal side effects
d. human recombinant α-interferon because it is safe and has been shown to be effective against FHV1 infection when given topically and systemically
e. no specific treatment only supportive therapy

8. Which of the following is not a feature of intranasal FHV1 and FCV vaccines?
   a. protection despite maternal antibody
   b. failure to protect against latent FHV1 infection
   c. establishment of latent FHV1 infection by the vaccine virus
   d. slow onset of immunity
   e. postvaccinal sneezing and ocular discharge

9. Chronic ocular infections associated with FHV1 may be difficult to diagnose because
   a. the virus is located in deep tissue that is inaccessible using swabbing techniques.
   b. immune-mediated disease predominates.
   c. the virus is present in an altered form that does not replicate well in cell culture.
   d. antibody complexing with virus prevents its replication in cell culture.
   e. multiple strains of FHV1 form as a result of immune pressure and are not reliably detectable using PCR.

10. Which of the following is not an advantage of PCR for diagnosing FHV1 infection?
    a. False-positive results do not occur.
    b. Sensitivity is greater than that of culture.
    c. FHV1 need not be viable for detection; therefore special transport media are not required.
    d. Rapid turnaround time is possible.
    e. Latent virus can be rapidly detected in the tissue of infected cats.