สมาคมสัตวแพทย์ผู้ประกอบการบำบัดโรคสัตว์แห่งประเทศไทย
โครงการ VPAT ACADEMY

VPAT ACADEMY WEBINAR

วีดีโอสำหรับทบทวนความรู้พื้นฐานด้านสัตว์เลี้ยง และสัตว์เลี้ยงชนิดพิเศษ

VPAT PARTNERSHIP 2021

“เนื้อหา ข้อความ รูปภาพ ภาพเคลื่อนไหว และเสียงทั้งหมด
ในคลิปบรรยายที่จัดทำขึ้นนี้ เป็นลิขสิทธิ์อย่างถูกต้อง
ของสมาคมสัตวแพทย์ผู้ประกอบการบำบัดโรคสัตว์แห่งประเทศไทย (VPAT)
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Common viral disease in small animal

Chaya Tinnakorn D.V.M.

Outline

01
Canine parvovirus

02
Canine Distemper virus

03
Feline parvovirus

04
Feline leukemia virus

Outline

05
Feline immunodefic-iency virus

06
Feline calicivirus

07
Feline herpesvirus

08
Feline coronavirus

Outline

09
Rabies virus

01 Canine parvovirus

Life-threatening diarrheal disease

Canine parvovirus

Etiology

- Family Parvoviridae Genus protoparvovirus
- **Non-enveloped**, DNA-containing viruses
- Require rapidly dividing cells for replication i.e. **enterocyte**
- CPV-1 and **CPV-2**
- Highly contagious and often fatal
- One of the most resistant canine viruses and can persist for months to years away from sunlight and disinfectants
- **Cross infection** between dog and cat (2a, 2b, 2c strain)
- Most severe in **puppy** (6 weeks and 6 months of age) esp. co-infect with other pathogen

Canine parvovirus

Route of transmission

- **Fecal-oral route**
  - Direct
  - Indirect i.e. instrument, insect (mechanical vector)

Incubation and shedding

- Incubation period are **7-14 days**
- Can be 4-6 day in case of 2a, 2b, 2c strains infection
- **Shedding start in 3-4 days after infection**, generally before overt clinical signs appear
- CPV-2 is shed extensively in the feces for a maximum of 7 to 10 days postinoculation (can be several weeks in case of 2a, 2b, 2c strain)

Pathogenesis

01

Oronasal exposure to contaminated feces

02

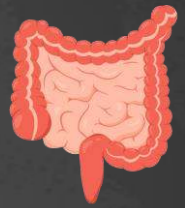
Replicate in local lymphoid tissue

03

Viremia (peak in 1-5 days after infection)

04

Disseminated to the intestinal crypts of the small intestine esp. **germinal cell**



05

destroys mitotically **active precursors** of circulating leukocytes and lymphoid cells

Canine parvovirus

Clinical finding

- **GI sign**
  - Vomit
  - Diarrhea (yellow-gray and are streaked or darkened by blood)
- Severe dehydrate
- Fever
- Leukopenia (mostly **lymphopenia**)
- Hypoglycemia, hypokalemia
- Cutaneous lesion i.e. ulcer and vesicle, erythematous patches
- Myocarditis (develop from infection in utero or in pups younger than 6 weeks)

Canine parvovirus

Diagnosis

- The sudden onset of foul-smelling, bloody diarrhea in a young dog (under 2 years of age) is often considered indicative of CPV infection.
- WBC count (lymphopenia)
- **Fecal ELISA antigen tests**
  - False negative can be occur from intermittent shedding and enteric antibody
- PCR

Canine parvovirus

Treatment

- **Fluid therapy**
  - Correct dehydrate
  - Correct electrolyte (hypokalemia)
  - Correct blood glucose
- ABOs use for control secondary infection
- Antiemetic drug
- Immunomodulator
- Supportive treatment
- Specific antibody to CPV

Sodium hypochlorite 0.1% W/V
for 10 minute (1:30-50)

Canine parvovirus

Who can kill parvovirus ?

- **Sodium hypochlorite 0.1% W/V (1:30-1:50)**
- 4% formaldehyde
- Peracetic acid
- Sodium hydroxide (0.1 M at pH 12.8 or higher)
- 1% glutaraldehyde
- 90 ° C

for 10 minute

“require rapidly dividing cells for replication”

“incubation period 7-14 days”

“long survival time in environment”

“correct hydration and electrolyte first”

Canine parovirus

02 Canine Distemper virus

Respiratory and neurological disease

Canine Distemper virus

Etiology

- Family Paramyxoviridae, Genus *Morbillivirus*
- Enveloped, single negative-stranded RNA
- Extremely **susceptible to heat and drying**
  - survives for at least an hour at 37 ° C and for 3 hours at 20 ° C
- **Wide host range**, mostly terrestrial carnivores i.e. fox, ferret, bear, civet, meerkat, raccoon, otter, large wild cat etc.
- Susceptible in 3-6 months of age but **can affecting all age**
- The infection rate is higher than the disease rate

Canine Distemper virus

Route of transmission

- Excrete via **respiratory secretion and urine**, commonly spread by aerosol or droplet exposure (direct contact and mechanical vector)
- Transplacental

Incubation and sheding

- Incubation period is 3-6 days
- **Shedding start in 5-7 days after infection and can be excreted up to 60 to 90 days after infection**
- Most recovered dogs clear the virus completely, some may harbor virus in their CNS.

Pathogenesis

01

Virus contacts epithelium of the upper respiratory tract

02

multiplies in tissue macrophages, spread to local LN and replicate

03

Widespread virus proliferation in lymphoid organs, damage to lymphoid cells result in lymphopenia and transient fever

04

Further spread of CDV probably occurs hematogenously as a cell-associated and plasma phase viremia and **depends on the dog's immune status**

Pathogenesis

Good immunity

infection may be eliminated, no clinical sign

Intermedieat immunity

Mild clinical sign and resolve, but virus can persist for extended periods in uveal tissues, neurons and in integument such as footpads, creating the potential for late-onset sequelae, particularly ocular and neurologic disease

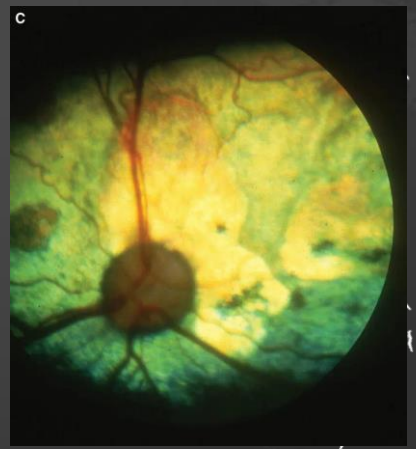
Poor immunity

classic acute distemper may occur, with subsequent death or recovery

Canine Distemper virus

Clinical finding

- Fever
- **Canine infectious respiratory disease complex (CIRDC)** or Kennel cough
  - Sneeze, cough, oculonasal discharge, pneumonia
- Vomit, Diarrhea
- Nasal and digital hyperkeratosis **(hard pad)**
- **Occular sign** (may occur after systemic sign resolve)
  - Conjunctivitis
  - KCS (transient or persistance)
  - anterior uveitis
  - blindness



Canine Distemper virus

Clinical finding

- Neurologic signs **occur 1–6 weeks after onset of systemic illness** or **can occur in mature or partially immune dogs that have been previously vaccinated and have no history of systemic disease**
- Typically progressive and **rarely resolve**
- Neurologic signs vary according to the area of the CNS involved
  - seizures (especially in young dogs)
  - Hyperesthesia (meningeal inflammation)
  - cerebellar and vestibular signs
  - paraparesis or tetraparesis
  - **Myoclonus**
  - Focal “chewing gum” seizures (common in puppy with acute CDV)

Canine Distemper virus

Clinical finding

- Transplacental
  - Abortion/stillbirth
  - Develop neurologic sign in 4-6 weeks of age
  - permanent immunodeficiencies because of damage to primordial lymphoid elements

Canine Distemper virus

Diagnosis

- based on clinical suspicion
- ELISA test kit
  - Collect sample from affected organ i.e. oculonasal discharge
- IFA
  - cytologic smears from conjunctival, tonsillar, genital, and respiratory epithelium, CSF, blood (buffy coat), urine sediment, and bone marrow
- CSF analysis
  - Increases in protein (more than 25 mg/ dL) and cell count (more than 10 cells/ μ L with a predominance of lymphocytes)
  - **Intracytoplasmic inclusions body**
- RT-PCR

Canine Distemper virus

Treatment

- Supportive care
- Antibiotic in case with secondary infection
- Dexamethasone use in case with CNS-edema or acute blindness
- Vitamin A support
- Specific antibody to CDV

“Severity of disease depend on immunity”

“One of the cause of CIRDC”

“Neurological and ophthalmological sing can occur after systemic sign or suddenly occur without systemic sign in case of persistance infection”

Canine Distemper virus

03 Feline parvovirus

Panleukopenia and diarrheal disease

Feline parvovirus

Etiology

- Family Parvoviridae Genus protoparvovirus
- **Feline panleukopenia virus (FPV)**
- **Non-enveloped**, DNA-containing viruses
- require rapidly dividing cells for replication i.e. **enterocyte, bone marrow**
- Genetically, structurally, and antigenically, it is closely related to canine parvovirus (CPV) **but dog is tolerate to FPV**
- Viverridae, Procyonidae, and Mustelidae are susceptible
- Mostly subclinical in healthy adult cat
- Severe clinical illness usually occur in kitten between **3-5 month of age**

Feline parvovirus

Route of transmission

- **Fecal-oral route**
  - Direct
  - Indirect i.e. instrument, insect (mechanical vector)

Incubation and shedding

- Incubation period are **2-14 days, mostly 5-7 days**
- Can shed 1-2 days before clinical sign and maximum of 6 weeks after recovery

Feline parvovirus

Clinical finding

- Peracute form
  - Die within 12 hour with few or no premonitory signs (**Fadding kitten syndrome**)
  - dehydrated, hypothermic, and comatose.
  - Septic shock

Feline parvovirus

Clinical finding

- Acute form
  - Fever (104 ° F - 107 ° F)
  - **Vomit**, unrelated to eating
  - Severe dehydration and hypokalemia
    - head hanging over water bowl
  - With or without diarrhea
  - **Mesenteric lymphadenomegaly** but normal peripheral LN
  - Susceptible to 2° infection and may develop DIC

Feline parvovirus

Clinical finding

- In ureto infection
  - Resorption, mummified fetus (queen may not develop other illness)
  - Late gestation
    - **Cerebellar hypoplasia** (can be affected by infections occurring as late as 9 days of age)
      - ataxia, incoordination, tremors
    - Hydrocephalus
    - Hydranencephaly
    - Optic nerve and retinal abnormalities
    - Habor virus for 8-9 weeks with no immunity

Feline parvovirus

Diagnosis

- Base on clinical sign
- WBC count
  - **leukopenia** (can decrease to 50 and 3000 cells/μL)
  - The severity of leukopenia usually parallels that of clinical illness
- **Fecal ELISA antigen tests**
  - False negative can be occur from short shedding period
  - Can use CPV-antigen test
- PCR
  - More sensitivity for subclinical shedding

Feline parvovirus

Treatment

- Fluid therapy
  - Correct dehydrate
  - Correct electrolyte (hypokalemia)
  - Correct blood glucose
- Beware secondary infection
  - Virus
  - Bacteria : ABO is in consider
- Antiemetic drug
- Immunomodulator
- Supportive treatment

“Similar to CPV in characteristic and pathogenesis”

“Panleukopenia”

“Vomit is dominte sign, diarrhea is less frequency”

“Cerebellar hypoplasia”

Feline parvovirus

04 Feline leukemia virus

Lymphoma induce disease

Feline leukemia virus

Etiology

- Family Retroviridae Genus Gammaretrovirus
- Enveloped single stranded RNA virus
- Target organ is **bone marrow**
- Replicate **provirus and integrated** into the host's cell genome
  - **Lifelong infection**
- Divined in 4 main subgroup
  - FeLV-A : transmit from cat to cat
  - FeLV-B : associate with neoplastic disease
  - FeLV-C : associate with non-regenerative anemia
  - FeLV-T : associate with severe immunosuppression

mutation and recombination of the FeLV-A genome with cellular genes or genes from endogenous retroviruses in the cat's genome

Feline leukemia virus

Route of transmission

- Viral load is highest in **saliva**
  - Close contact, oronasal route
  - Bite wound
  - Indirect via sharing water and food dish (less common due to unstability in environment)
- Blood transfusion
- Transplacental, milk

Incubation

- 1-12 month

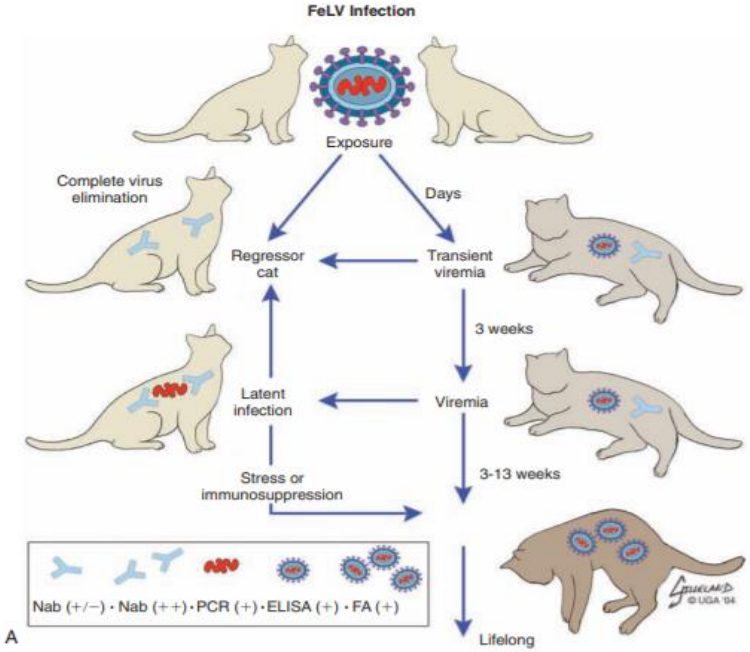


FIG. 11-4 ***A,*** Time course of FeLV infection. ***B,*** Components of FeLV from part A. *ELISA,* Enzyme-linked immunosorbent assay; *FA,* fluorescent antibody; *FeLV,* feline leukemia virus; *PCR,* polymerase chain reaction. (Art by Brad Gilleland © 2004, University of Georgia Research Foundation Inc.)

Abortive infection (regressor cat)

- Complete viral elimination

Regressive infection

- Latent infection, have proviral DNA in blood but have no viral RNA
- Can recurrent when have activator

Progressive infection

- Persistence viremia (>16 wks)
- Usually relate to clinical sign

Focal or Atypical infection

- Replication restrict in specific tissue
- Rarely seen

Feline leukemia virus

Clinical finding

- Tumors
  - **Lymphoma**
    - Mediastinal : dyspnea due to pleural effusion
    - Alimentary : Vomit, diarrhea
    - Extranodal : kidney, CNS, nasal
  - Leukemia
  - Fibrosarcroma
  - Other hematopoietic tumor

Feline leukemia virus

Clinical finding

- Anemia
- Immunosuppression
  - Co-infection with other virus
  - Bacterial secondary infection
  - Thymic atophy in kitten
- Immune-mediated disease
  - IMHA
  - Glomerulonephritis
  - Uvitis

Feline leukemia virus

Diagnosis

- Hematology
  - Leukopenia
  - Thrombocytopenia
  - Anemia
  - WBC morphology : increase blast cell
- Bone marrow biopsy, Tissue biopsy
- **Detect FeLV Ag (P27 protein)** in blood : ELISA, Direct FA test
  - Recheck with ELISA within 1-3 month if expose risk
- PCR
  - Detect viral RNA
  - Detect proviral DNA

TABLE 11-3

Characteristics of Stages of Feline Leukemia Virus Infection

Outcome of FeLV Infection	FeLV p27 Antigen in Blood	Virus Blood Culture	Viral RNA in Blood	Viral DNA in Blood	Viral Tissue Culture	Viral Shedding	FeLV-Associated Disease
Progressive	Positive	Positive	Positive	Positive	Positive	Positive	Likely
Regressive	Negative	Negative	Negative	Positive	Negative	Negative	Unlikely
Abortive	Negative	Negative	Negative	Negative	Negative	Negative	Unlikely
Focal	Negative	Negative	Not tested	Not tested	Positive	Variable	Unlikely

Progressive = persistent viremia; Regressive = transient viremia followed by latent infection; Abortive = complete elimination.
From Ref. 260.

Feline leukemia virus

Treatment

- Control secondary infection
- Lymphoma and Leukemia
  - **Chemotherapy : COP protocal**
  - Radiotherapy
  - Surgical remove
- Outher sarcroma
  - Surgical remove, deep and wide excision
- Immunomodulator
  - INF-α, INF-ω, etc,
- Antiviral drug
  - Zidovudine (AZT)

“outcome of infection depend on immune response”

“regression infection have only proviral DNA in blood, don’t have clinical sign but can develop if have activator”

“progression infection have both viral RNA and viral Ag in blood”

Feline leukemia virus

05

Feline immunodeficiency virus

Immunodeficiency disease

Feline immunodeficiency virus

Etiology

- Family Retroviridae Genus Lentivirus
- Enveloped RNA virus
- Replicate **provirus and integrated** into the host's cell genome
  - **Lifelong infection**
- 5 subtype : A, **B, C, D**, E, F
- Main target cell is **CD4+ T-cell**
  - Also infect CD8+ T-cell, B-cell, Macrophage, dendritic cell and astrocyte
- Very unstable in environment

Feline immunodeficiency virus

Route of transmission

- Viral in **saliva and blood**
- Main route of transmission is **bite wound and parenteral route**
- Transplacental, milk, and transvenerial are uncommon

Incubation

- 3-6 month for acute phase
- Unpredictable for terminal stage, commonly several years

Pathogenesis

01

Virus infect lymphoid tissue, integrate and replicate. Then viremia in 2 wks and peak in 8-12 wks

02

Acute phase occur in 3-6 month with transient illness associate with decline in CD4+ and CD8+ T cells in peripheral blood, Treg activate and altered dendritic cell function

03

Result in cytokine dysregulation, immunologic anergy and increased apoptosis of T-cell those **impair T-cell function**

04

most cats survive this phase because of a rebound in CD8+ T cell numbers and a strong humoral immune response and turn to **asymptomatic phase**

Pathogenesis

05

Cat may survive for year in asymptomatic phase with no clinical sign but **virus production continues at low levels and slow progressive decline in CD4+ T cell numbers**

06

Rate of progression depend on immunity and other factor, cat will turn to **terminal stage** that cause cytokine dysregulation, immunologic anergy and increased apoptosis of T-cell again

07

opportunistic infections, neoplastic disease, myelosuppression, and neurologic disease will occur

Feline immunodeficiency virus

Acute phase

- Viral replicate and altered function of CD4+ T-cell, CD8+ T-cell, B-cell, macrophage and dendritic cell
- Transient illness, will recovery from rebound of CD4+/CD8+ Tcell and HI

Asymptomatic phase

- No clinical sign
- Virus slowly replication and slow progressive decline in CD4+ T cell numbers

Terminal phase

- Paradoxically, cytokine dysregulation, immunologic anergy and increased apoptosis of T-cell again lead to illness and death
- May or may not develop to this phase

Feline immunodeficiency virus

Clinical finding

- **Acute phase**
  - Fever / anorexia
  - Diarrhea
  - stomatitis
  - Lethargy and weight loss
  - Lymphadenomegaly
- **Asymptomatic phase**
  - No clinical sign

Feline immunodeficiency virus

Clinical finding

- **Terminal phase**
  - Wasting syndrome
  - Secondary infection esp. dermatologic disease
  - **Moderate to severe periodontal disease**
  - Myelosuppression / myelodysplasia
    - Anemia
    - Bleeding disorder
  - inflammatory disorders
    - immune-mediated glomerulonephritis
    - inflammatory cardiac and other myopathies

Feline immunodeficiency virus

Clinical finding

- Terminal phase
  - Neurologic disease
    - behavioral changes, Cognitive dysfunction
    - Seizure, paresis, delay reflex
  - ophthalmic disease
    - Uvitis
    - Glaucoma
  - neoplastic disease
    - B-cell lymphoma
    - Leukemia
    - SCC

Feline immunodeficiency virus

Diagnosis

- Hematology
  - Leukopenia with neutropenia and lymphopenia
  - Anemia
  - Thrombocytopenia
- Blood chemistry
  - Hyperproteinemia with hyperglobulinemia
- **ELISA test for Antibody detection**
  - Should be re-test in 2-3 months if cat expose risk of infection

Feline immunodeficiency virus

Treatment

- Supportive care
- Control secondary infection
- Immunomodulator
  - INF-α, INF-ω
- Antiviral drug
  - Zidovudine (AZT)

“lifelong infection with asymptomatic phase”

“May or may not develop terminal phase”

“Destroy immune system esp. CD4+ T-cell”

“Detect Ab in blood by ELISA ”

Feline immunodeficiency virus

06

Feline calicivirus

Oral ulcer associated disease

Feline calicivirus

Etiology

- Genus *Vesivirus*, Family calicivirus
- **Non-enveloped**, single-stranded RNA virus
- All member of Felidae are susceptible
- Can survive in environment for several weeks at room temperature
- A large number of different strains of FCV exist, which vary slightly in antigenicity and pathogenicity

Carrier stage

- Some cat (around 10%) are **persistence infection and continuous shadding**
- The virus persists in carriers in tonsillar and other oropharyngeal tissues

Feline calicivirus

Route of transmission

- The viruses are mainly shed in **ocular, nasal, and oral secretions**
- Transmit by **direct contact** and indirect contact
- sneezed macrodroplets may transmit infection over a distance of 1 to 2 m

Incubation

- FCV : 2-14 days

Pathogenesis

01

Virus enter via oral, nasal and conjunctival

02

Replication mainly occurs in the oral and respiratory tissues

03

Some strains have a predilection for the lung, and others have been found in the macrophages within the synovial membrane of joints

04

Develop lesion in **oral, lung and joint**

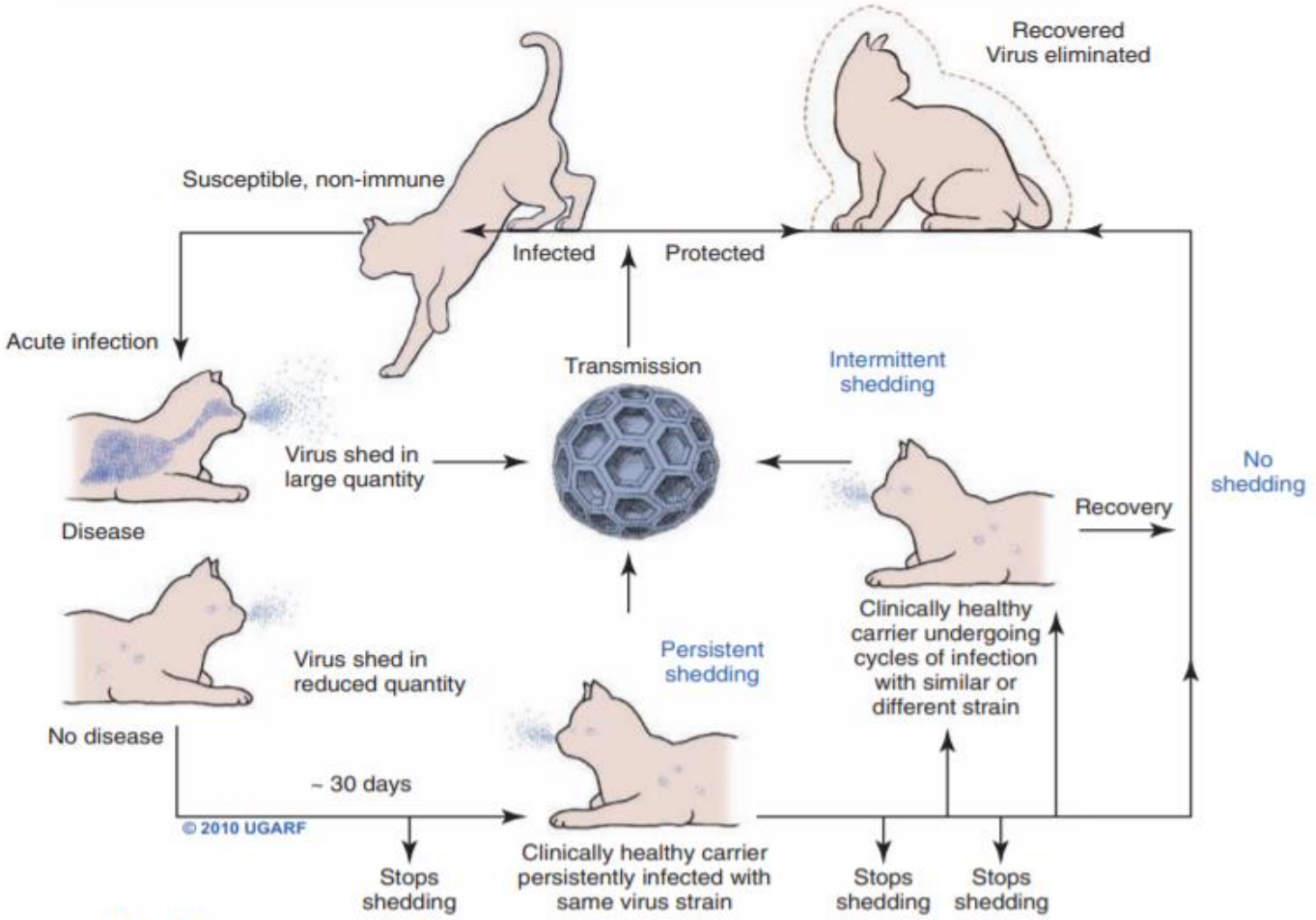


FIG. 14-2 FCV carrier state: epidemiology. (Art by Brad Gilleland © 2010, University of Georgia Research Foundation Inc.)

Feline calicivirus

Clinical finding

- wide range of clinical signs, depend on strain of virus
- begin as vesicles, then rupture and develop to **Oral ulcers**
    - the most prominent pathologic feature of FCV infection
    - Mostly on tongue, but can occur elsewhere in the mouth, lips, and nose
- Hypersalivation (wet fur around the mouth but not drooling)
- **Occulonasal sign**

    Less when compare with FHV
- Joint swelling and lameness

    Usually recovery within 1-2 days
- Pneumonia can occur in severe case

Feline calicivirus

Diagnosis

- based on clinical signs
- PCR test
  - oropharyngeal swabs or conjunctival swab
  - Not wildly use due to high false negative from strain variability

Feline calicivirus

Treatment

- No specific treatment
- Symptomatic treatment and supportive care

“oral ulcer lesion”

“10% develop persistence shedding”

“variable of symptom depend on strain, don’t overlook it !”

Feline calicivirus

07

Feline herpesvirus

Occulonasal associated disease

Feline herpesvirus

Etiology

- Family Herpesviridae Genus vericellovirus
- **Enveloped** double-stranded DNA virus
- fragile outside the host and is highly susceptible to the effects of common disinfectants
- All member of Felidae are susceptible

Carrier stage

- all recovered cats become **latently infected carriers**
    - latent in carriers in the **trigeminal ganglia**
    - Intermittent shedding esp. after stress i.e. parturition and lactation
    - Start shedding 1 wk after stress and continue for 1-2 wks

Feline herpesvirus

Route of transmission

- The viruses are mainly shed in **ocular, nasal, and oral secretions**
- Transmit by **direct contact** and indirect contact (less)
- sneezed macrodroplets may transmit infection over a distance of 1 to 2 m

Incubation

- FHV : 2-6 days

Pathogenesis

01

Virus enter via oral, nasal and conjunctival

02

Replicate in mucosae of the nasal septum, turbinates, nasopharynx, and tonsils

03

multifocal epithelial necrosis with neutrophilic infiltration and exudation with fibrin occur in infected area

04

secondary infection may enhance the severity of disease

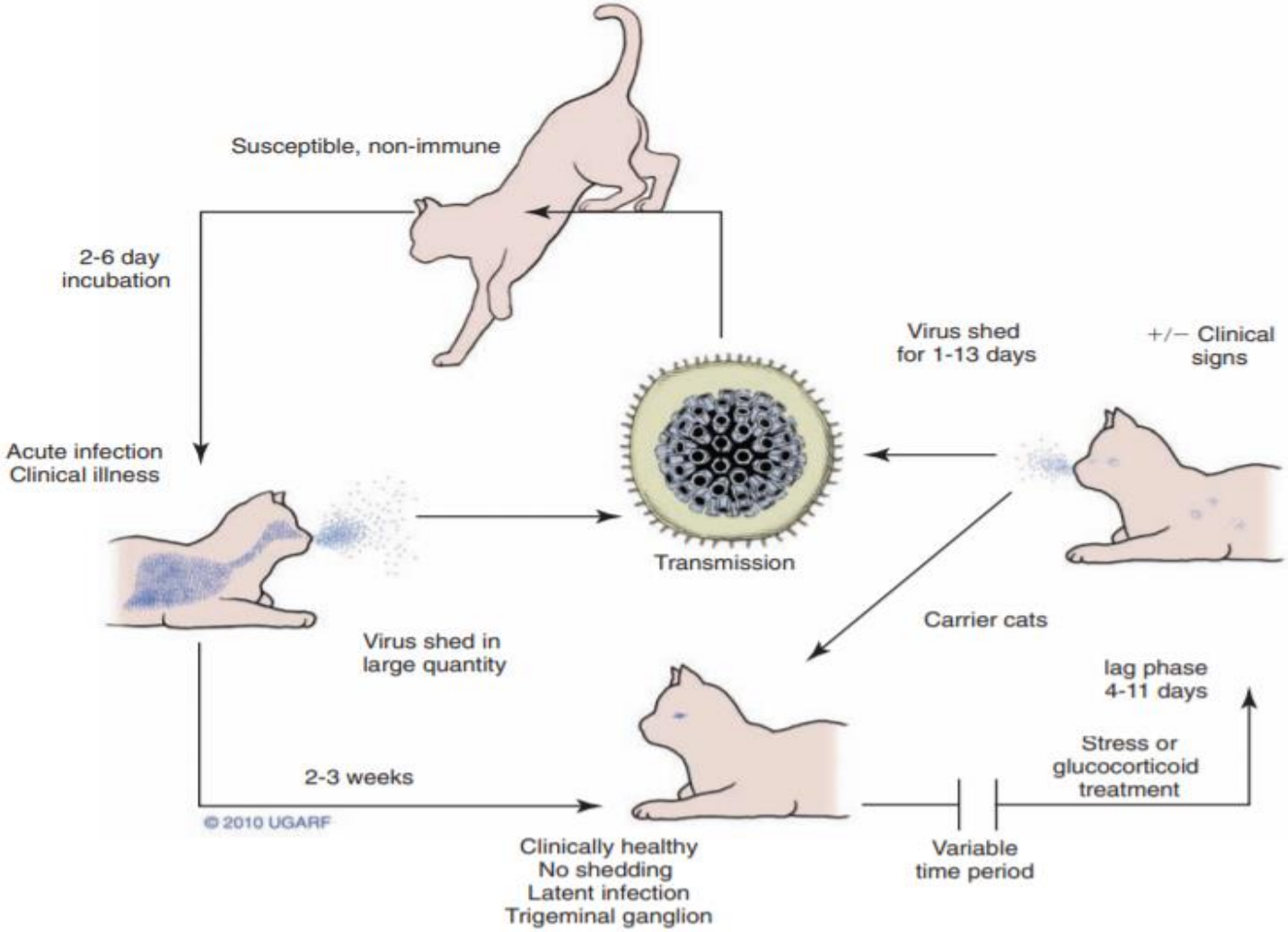


FIG. 14-1 FHV-1 carrier state: epidemiology. (Art by Brad Gilleland © 2010, University of Georgia Research Foundation Inc.)

Feline herpesvirus

Clinical finding

- Depress, Fever, Sneezing
- serous **occulonasal discharges**, then develop to mucopurulent
- **Occular lesion**
  - Conjunctivitis
  - Chemosis
  - Ulcerative keratitis
- turbinate osteomyelitis
  - May permanent damage, prone to chromic bacterial rhinitis
  - Higher incidence in short-nosed purebred cats
- Can develop pneumonia in severe case

Feline herpesvirus

Diagnosis

- based on clinical signs
- PCR test
    - oropharyngeal swabs or conjunctival swab

Feline herpesvirus

Treatment

- Antiviral drug
  - topical use : acyclovir, cidofovir, vidarabine, idoxuridine
  - oral use : famciclovir
- Interferon-omega
- Lysine
- ABOs use in severe case
  - **Tetracycline / Doxycycline**
  - Sulfa-trimetroprim, Floroquinolone, Azithromycin
- Symptomatic treatment and supportive care

TABLE 14-1

Essential Features of Clinical Respiratory Disease[a] Related to the Pathogen Involved

Feature	FHV-1	FCV[b]	Bb	ChF
Lethargy	+++	+	+	+
Sneezing	+++	+	++	+
Conjunctivitis	++	+	–	+++[c]
Hypersalivation	++	–[d]	–	–
Ocular discharge	+++	+	(+)	+++
Nasal discharge	+++	+	++	+
Oral ulceration	(+)	+++	–	–
Keratitis	+	–	–	–
Coughing	(+)	–	++	–
Pneumonia	(+)	(+)	+	+/–
Lameness	–	+	–	–

+++, Marked; ++, moderate; +, mild; (+), less common; +/–, subclinical; –, absent; *Bb, Bordetella bronchiseptica; ChF, Chlamydophila felis*; *FCV*, feline calicivirus; *FHV-1*, feline herpesvirus.

[a]For FCV-associated virulent systemic disease signs, see text.

[b]Strain variation.

[c]Often persistent.

[d]Slight wetness may be seen around the mouth if ulcers present.

Adapted from Ref. 50. Used with permission.

“Occulonasal lesion”

“latent infection, shedding after stress”

“Anti-herpesvirus drug”

Feline calicivirus

08

Feline coronavirus

Mutate and cause fatal disease

Feline coronavirus

Etiology

- Family Coronaviridae, Genus alphacoronavirus
- enveloped, positive-sense single-stranded RNA
- Normally FCoV cause enteric disease but 1-3% of infected cat will cause **Feline infectious peritonitis (FIP)**
    - virulent viruses arise as a result of de novo **mutation**
    - **Immune complex disease**
- FCoV can survive for 7 weeks in a dry environment

Route of transmission

- Fecal-oral route
- **Transmission occur before mutation**
    - After mutation, it can't replicate in GI tract

Feline coronavirus

Incubation period and shedding

- Start shedding 2 days after infection and **shedding for 2-3 months**
- **Some cat are persistence infection** (~13%)
  - May develop chronic disease of large intestine but rarely develop FIP
- >75% of FIP cat don't shed virus in feces

Risk factor

- Age
  - Kitten to 2 years of age
  - Cat over 10 years of age
- Breed
  - Pure breed

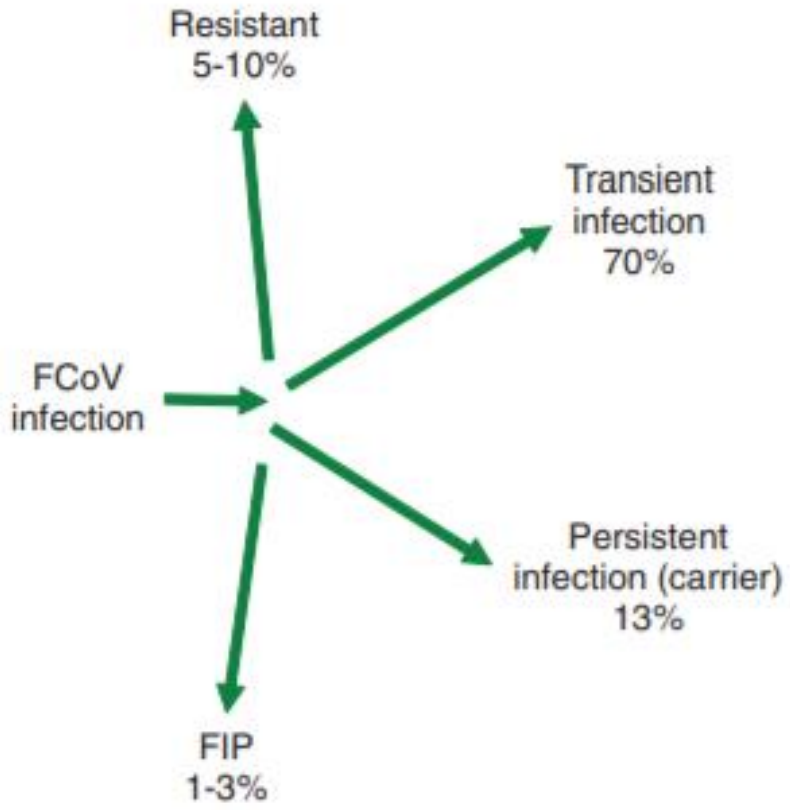


FIG. 10-1 There are four possible outcomes to FCoV infection, and only in a minority of infections is FIP the outcome. The percentage of cats that will have each outcome is shown. (Modified from Addie DD, Jarrett O. 2001. *Vet Rec* 148:649–653.)

Pathogenesis

01

Virus enter cat by fecal-oral route, then mutate and infect macrophage

02

Type and strength of immune response determine the outcome of FCoV infection

CMI

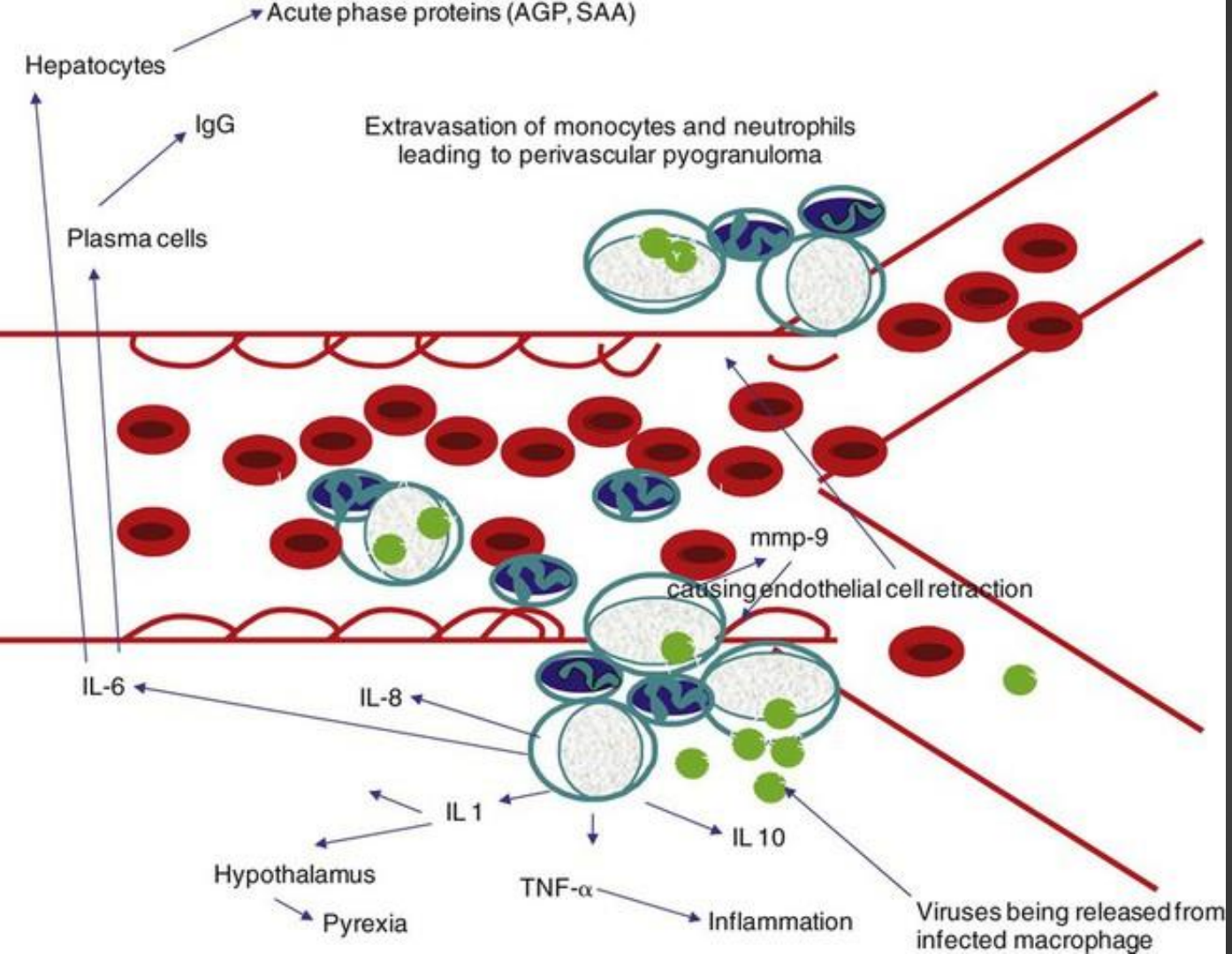
Strong CMI response will prevent FIP

Int

Partial CMI response result in non-effusive FIP (Dry form)

HI

Weak CMI and strong HI response results in effusive FIP (Wet form)



"systemic pyogranulomatous vasculitis"

Feline coronavirus

Clinical finding

- **Diarrhea (coronavirus enteritis)**
  - **Mild and usually self-limiting**
  - May involve in some chronic diarrhea case
- Lethargy and anorexia
- Clinical sign depend on type of disease present (dry or wet form) and the location where lesions occur

Feline coronavirus

Clinical finding

- Dry form
  - **pyogranulomatous to granulomatous inflammation** within a variety of organs, but especially the mesenteric lymph nodes, kidneys, liver, lungs, brain, and eye
    - Clinical sign depend on location of granuloma
  - Solitary or multifocal granulomas of the intestinal wall
    - Mostly in the **colon or ileocecocolic junction**
    - Constipation or chronic diarrhea

Feline coronavirus

Clinical finding

- Wet form
  - **Cavity effusion**
    - Pleural effusion : tachypnea and respiratory distress
    - Abdominal effusion : abdominal distension and fluctuate
    - Pericardial effusion : cardiac tamponade
  - Dry form can develop to wet form

Feline coronavirus

Diagnosis

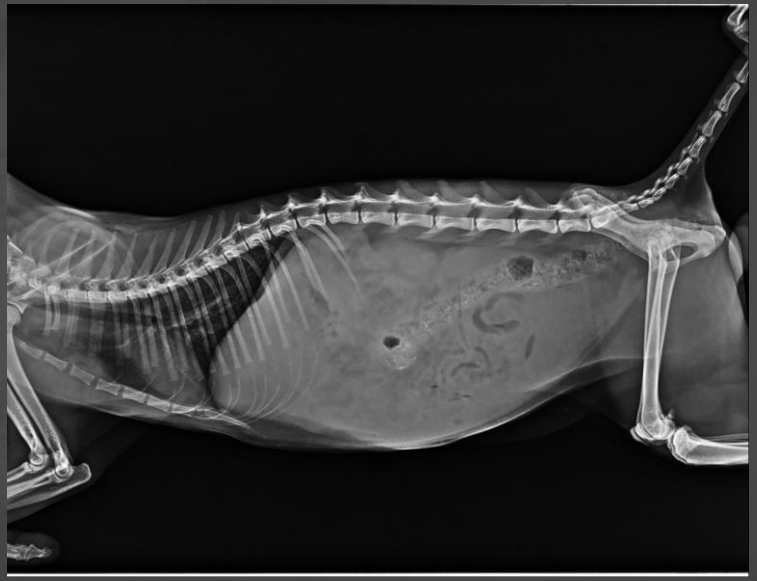
- Hematology
  - Leukocytosis with neutrophilia and monocytosis , but **lymphopenia** is commonly seen
  - Anemia
    - Anemia of chronic disease : non-regenerative
    - Microangiopathy : schistocyte
- Blood chemistry
  - Hyperglobulinemia
  - Hypoalbuminemia

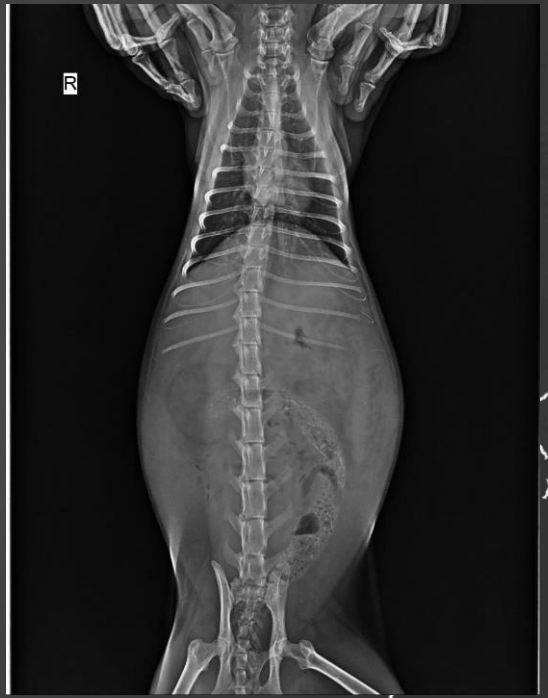
A:G ratio < 0.6 → likely to be FIP
A:G ratio > 0.8 → unlikely to be FIP

Feline coronavirus

Diagnosis

- Radiology
  - X-ray
  - Ultrasound





Feline coronavirus

Diagnosis

- Fluid analysis
  - Clear, straw colored, and viscous
  - **Modified transudate**
    - High-protein (greater than 3.5 g/dL)
    - Low number of nucleated cells (<5,000 cell/μL), dominate with Neutrophile and Macrophage
  - Albumin/Globulin ratio
    - >0.8 → exclude FIP
    - 0.45 – 0.8 → remain possible
    - <0.4 → likely FIP
  - Immunofluorescent staining

FIG. 10-8 Positive Rivalta test: one drop of 98% acetic acid is added to 5 mL of distilled water and mixed thoroughly, and a drop of effusion is carefully layered on top. If it disappears and the solution remains clear, the test is negative. If the drop retains its shape, stays attached to the surface, or floats slowly down the tube, the test is positive. (Courtesy Diane Addie, Feline Institute, Pyrenees, France.)

Rivalta test

- 98% Acetic acid add to distrilled water 5 ml

Feline coronavirus

Diagnosis

- RT-PCR
  - Not a definitive diagnosis due to high false-negative
  - Detect virus in effusion
- **Gold standard is immunohistochemical staining** for coronavirus antigen within lesions
  - Antemortem diagnosis is often only suspected on the basis of history, signalment, clinical sign and laboratory findings

Feline coronavirus

Treatment

- Immunosuppressive drug
- Interferon
- Anti-viral drug
    - GS-441524 (nucleoside ribose analogue)
- Supportive care

“Not all FeCoV infected cat develop FIP”

“Severity of disease depend on CMI response”

“Macrophage play importance role in pathogenesis”

“No specific test for diagnosis FIP”

Feline coronavirus

09 Rabies

Zoonotic and fatal disease

Rabies

Etiology

- family Rhabdoviridae, genus *Lyssavirus*
- enveloped, **bullet-shaped** RNA viruses
- Can infect **all mammal species**
- extremely labile when exposed to ultraviolet light and heat
    - Can survive in carcass for several day at 20 C

Route of transmission

- Mainly by bite wound or scratch, **virus is in saliva**

Rabies

Incubation period

- **generally 3–12 weeks**, but can range from several days to months, rarely exceeding 6 months
- Depend on
  - the **degree** of innervation of the bite site
  - the **distance** from the point of inoculation to the spinal cord or brain
  - the **amount** of virus introduced

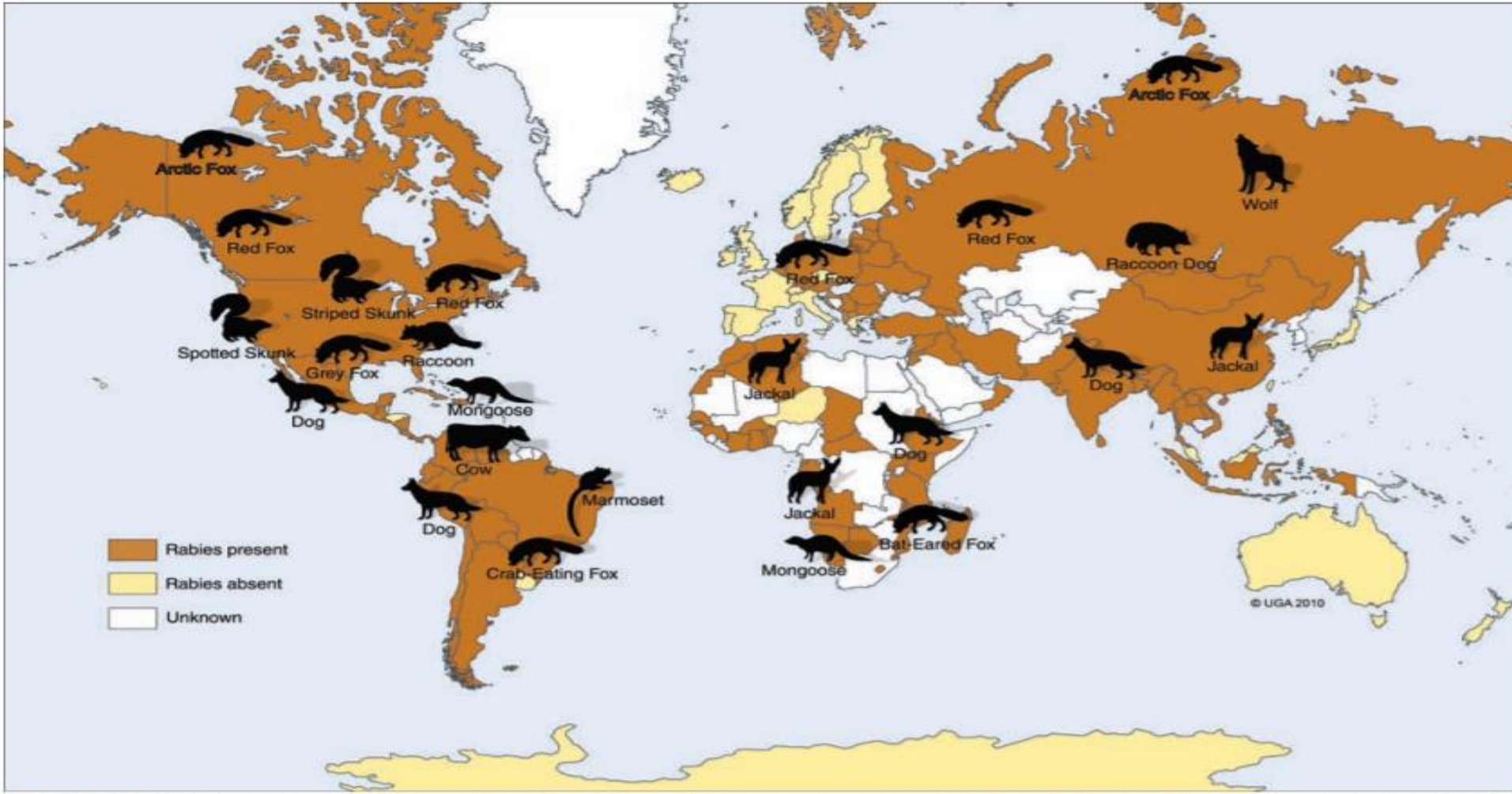


FIG. 20-2 Principal animal vectors of rabies for major regions of the world. Some countries are reportedly free of rabies (see Hosts and Range in text). (Art by Brad Gilleland © 2010 University of Georgia Research Foundation Inc.)

Pathogenesis

01

Virus in saliva enter peripheral nerves directly or replicate locally in non-nervous tissue through bite wound

02

Virus spreads passively by intra-axonal flow in peripheral nerves to CNS

03

Virus spread in the CNS corresponds to the progression of clinical signs

04

Virus spreads via cranial nerves to the acinar cells of the salivary glands and excrete via saliva **(usually 1-5 days before onset of neurological sign)**

Rabies

Clinical finding

- **Prodromal phase (2-3 days)**
  - anxiety, fever, pupillary dilation and behavioral change
  - lick or chew at the bite wound
- **Furious phase (1-7 days)**
  - increased responses to auditory and visual stimuli
  - excite, photophobic, and hyperesthetic
  - bark or snap at imaginary object, bite the cage
  - May develop tonic-clonic seizure

Rabies

Clinical finding

- **Paralytic phase (2-4 days)**
  - LMN paralysis (Flaccid paralysis), usually progresses from the site of injury
  - salivate or froth
  - dropped jaw
  - Coma
  - dies from respiratory failure

Rabies

Practical guideline

- For dog and cat those bite people
  - Stray dog : Euthanasia
  - Have owner : restrict area for **10 days** to observe
- For dag and cat those expose to infected animal
  - No vaccination history
    - Euthanasia
    - Restrict area for **180 days** and give vaccination for 4 time, 4 days apart between each shot (0, 4, 8, 12)
  - Vaccinated
    - Restrict area for **45 days** and boots vaccination for 2 time (0, 3)

Rabies

Diagnosis

- **No premortem diagnostic tests are sensitive enough** to be consistently reliable for rabies diagnosis in animals
- **direct fluorescent antibody (DFA) test** in suitable **brain tissue**
  - Small size : whole body
  - Large size : only head (dissect at atlanto-occipital joint)
  - Preserve in **4 C°, don't freeze** and send to lab **in 24 hr.**
  - Preserve in **-20 C°** if sending process take time **more than 24 hr.**

“Incubation period depend on inoculation site and viral load, mostly 3-12 weeks”

“Predromal phase, furious phase and paralytic phase”

“10 days for suspected, 45 days for vaccinated and 180 days for non-vaccinated”

Rabies virus