CONFERENCE 8
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CASE I – XN2922 (AFIP 2940469)

Signalment: Adult male, ferret (*Mustela putorius furo*).

History: An adult male ferret of unknown age was one of several housed in a council animal park open to the public. It developed progressively worsening dyspnea, with lethargy and mild abdominal distension, in early June 2004 and was euthanized.

Gross Pathology: The adult male ferret weighed 1120 g and had a length from the nose to the tail tip of 60 cm. The pleural cavity contained 50 ml of light brown, opaque fluid. The heart was enlarged and globular, with a cross-section of 25 mm by 20 mm at mid-ventricular level. At this level, the thickness of the left ventricular free wall was 4 to 5 mm, the thickness of the right ventricular free wall was 2 mm and the interventricular septum was 3 to 4 mm thick. The heart had patchy areas of pallor on the epicardium and there were irregular, locally extensive, white to pale yellow areas of myocardium in the inner third of the left ventricle. The right ventricle was affected to a lesser degree. The subendocardial myocardium and the bases of the papillary muscles were most severely affected. The lungs had patchy, red to purple atelectasis affecting 60% of the total lung volume. The abdomen was mildly distended and contained 5 ml of slightly yellow, clear, transparent fluid. The liver was enlarged, with rounded borders, an irregular, granular capsule and exudation of fibrin on the surface. The lobular pattern of the liver was accentuated, with red-purple mottling. The kidneys and spleen were congested.

Contributor's Morphologic Diagnosis: Heart: Myocardium, degeneration, necrosis, fibrosis, nonsuppurative inflammation, subendocardial and subepicardial, severe, extensive, consistent with dilatative cardiomyopathy, ferret (*Mustela putorius furo*).

Contributor's Comment: Histologically, the subendocardial myocardium of this ferret exhibits degeneration and necrosis of cardiomyocytes, with extensive replacement by fibrous connective tissue. The bases of papillary muscles and the inner third of the myocardium of the left ventricle, including the left ventricular free wall and the interventricular septum, are most severely affected. Subepicardial degeneration and fibrosis tend to have a perivascular distribution. Mild cardiomyocyte degeneration and fibrosis are also evident in the subendocardial myocardium of the right ventricle, especially in the interventricular septum. There are mild, diffuse and locally extensive infiltrates of macrophages, lymphocytes and plasma cells, as well as occasional neutrophils, in areas of degeneration and fibrosis. Other tissues had evidence of congestive heart failure. Congestion, atelectasis and edema were evident in the lungs. There was congestion, periacinar fibrosis, mild hemosiderosis and atrophy of subcapsular hepatocyte cords in the liver, reflecting chronic hepatic venous congestion. The kidney was congested and hemosiderosis was evident in renal tubular epithelial cells. The adrenal and thyroid glands were unremarkable. The gross and histological findings are consistent with dilatative (dilated or congestive) cardiomyopathy of ferrets.[1-3]

Cardiomyopathy is the most common cause of heart failure in the ferret.[2-5] The usual form of this condition in ferrets is dilatative (dilated or congestive) cardiomyopathy, although hypertrophic and restrictive cardiomyopathies have also been described in this species.[2,5] Ferrets are most frequently affected from 5 to 7 years of age, although some severe cases can develop clinical signs as early as 1 year of age.[4,5]

Clinical signs of dilatative cardiomyopathy in ferrets include weight loss, weakness, lethargy, dyspnea, tachypnea, tachycardia, coughing and abdominal distension.[4,5] Heart and lung sounds are muffled on auscultation of the thorax and moist rales may be evident. A holosystolic murmur is usually most intense on the left side of the thorax at the level of the seventh to eighth intercostal space. Ascites and pleural effusions may be present. Hepatomegaly and splenomegaly may be evident on palpation of the abdomen. Diagnosis of cardiomyopathy in ferrets is usually based on clinical signs, radiography and echocardiography.[2-4] Ancillary diagnostic tests may include electrocardiography[2,6] and cytological examination of fluids obtained by thoracocentesis or abdominocentesis. Hematology and biochemical testing including endocrinology are useful for evaluation of concurrent disease.

The cause of cardiomyopathy in the ferret is unknown. A genetic basis has been suspected in some lines of ferrets in North America.[5] Cardiomyopathy has been

associated with hyperadrenocorticism in ferrets.[7,8] In one study, 10% of ferrets with hyperadrenocorticism had concurrent cardiomyopathy.[8] A ferret with meningitis due to *Cryptococcus neoformans* had concurrent congestive cardiomyopathy.[9]

Treatment of cardiomyopathy in ferrets usually includes diuretics such as furosemide, vasodilators such as enalapril, positive inotropes such as digoxin and a low sodium diet.[2-4,10] The long term prognosis for ferrets with cardiomyopathy is poor. There is insufficient evidence to determine if supplementation with taurine or carnitine is beneficial in preventing or treating the disease.[2,11]

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AFIP Diagnosis: Heart, myocardium: Degeneration, necrosis, and loss, with replacement fibrosis, multifocal, marked, ferret (*Mustela putorius furo*), mustelid.

Conference Comment: Cardiac disease is common in older ferrets and is usually due to dilated cardiomyopathy, hypertrophic cardiomyopathy or valvular disease. The contributor provides a thorough overview of dilated cardiomyopathy. Hypertrophic cardiomyopathy has not been extensively studied in ferrets. Unlike cats, there is no known association with hyperthyroidism or hypertension. Grossly the interventricular septum and left-ventricular free walls are abnormally thickened with decreased internal dimensions, and often an enlarged left atrium. Histologically, fibrous connective tissue is present throughout the myocardium. Valvular heart disease is reported with increasing frequency, with gross lesions consisting of abnormally thickened valves and dilated atria. Histologically, there is myxomatous degeneration of the valve as with dogs with endocardiosis. Other, less common causes of heart disease in ferrets include myocarditis, which may be due to Toxoplasma-like organisms, parvovirus (Aleutian mink disease), septicemia, and *Dirofilaria immitis* (heartworm disease).[2]

Cardiomyopathies have been reported in a number of other species including the cat, dog, pig, cow, hamster, turkey, mouse, and man and are classified as primary or secondary. Primary cardiomyopathies, those without a known etiology, are further subdivided into dilated, hypertrophic, and restrictive. Secondary cardiomyopathies are associated with known etiologies, such as viral myocarditis.[1]

Dilated cardiomyopathy is an important cause of congestive heart failure in dogs and cats. Often cats will have low tissue concentrations of taurine and supplementation with taurine has reversed the clinical signs of cardiac failure. Affected dogs are often males of large breeds, such as Doberman Pinschers, Irish Wolfhounds, and Newfoundlands. Grossly the heart is rounded due to biventricular dilatation, often with a diffusely white, thickened endocardium. Histological

changes are non-specific, may be mild or absent, and include interstitial fibrosis and myocyte degeneration. Hypertrophic cardiomyopathy occurs frequently in middle-aged male cats. Animals often present with congestive heart failure and approximately 10-20% will have posterior paresis from a concurrent thromboembolism of the caudal abdominal aorta ("saddle thrombus"). Grossly the heart is enlarged with prominent hypertrophy of the left ventricle and interventricular septum, the left ventricular cavity is small, and the left atrium is dilated. Histologically there is prominent disarray of cardiac myocytes, with interweaving rather than parallel fibers, interstitial fibrosis, and myocyte degeneration. Restrictive cardiomyopathy occurs infrequently and includes such conditions as endocardial fibrosis in certain strains of rats and congenital endocardial fibroelastosis in Burmese cats.[12]

Causes of secondary cardiomyopathy include infectious, hereditable, nutritional, toxic, physical injuries, endocrine disorders, neoplasia, and systemic hypertension in cats. Some infectious agents include:[12]

<u>Viral</u>:
> Canine parvovirus (canine parvovirus type 2)
> Encephalomyocarditis (cardiovirus)
> Foot-and-mouth disease (picornavirus)
> Pseudorabies (porcine herpesvirus)
> Canine distemper (canine morbillivirus)
> Cytomegalovirus (porcine betaherpesvirus)
> Newcastle disease (avian paramyxovirus)
> Eastern and western equine encephalomyelitis (alphavirus)
> West Nile Virus (flavivirus)

<u>Bacterial</u>:
> *Clostridium chauvoei* (Blackleg), *C. piliforme* (Tyzzer's disease)
> *Listeria monocytogenes* (Listeriosis)
> *Fusobacterium necrophorum* (Necrobacillosis)
> *Mycobacterium* spp. (Tuberculosis)
> *Corynebacterium pseudotuberculosis* (Caseous lymphadenitis)
> *Actinobacillus equuli*
> *Staphylococcus* sp.
> *Streptococcus pneumonia*

<u>Parasitic</u>:
> *Toxoplasma gondii*
> *Sarcocystis* sp.
> *Encephalitozoon cuniculi*
> *Trypanosoma cruzi*
> *Cysticercus cellulosae*
> *Trichinella* sp.

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CASE II – 39080 (AFIP 2936322)

Signalment: 1.5 year old, male-neutered, Domestic Shorthair cat.

History: Cat exhibited lethargy and anorexia, and presented with a temperature of 105 degrees Fahrenheit and respiratory harshness. Feline Leukemia Virus and Feline Immunodeficiency Virus tests in clinic were negative. All in-house lab work

was normal. Cat declined despite antibiotic treatment and IV fluids. Within the last 12 hours preceding death, it became very agitated and aggressive. Clinical diagnosis was unknown.

Gross Pathology: The cat presented in fair flesh and poor postmortem preservation. There was moderate dilatation of the colon, with a dark red serosa. The descending colon's content was dark red.

Laboratory Results: No clinical pathology data was available. Bacteriologic examination: Lung, intestine: Nonpathogenic bacteria were isolated. Liver: No growth was observed.

Contributor's Morphologic Diagnosis: Protozoal schizonts, intravascular, multisystemic, feline.

Contributor's Comment: Multifocal intravascular protozoal schizonts were in the brain, heart, lung, intestine, spleen and kidney. Abundant acidophilic material admixed with RBC was in the colon. Tapeworms were present in the duodenum.

Cytauxzoonosis is a rare disease. In the U.S. it is most common in free-roaming felines in the south and is usually, but not always, fatal. Diagnosis is made by finding the piroplasms of *Cytauxzoon felis* in blood smears (although parasitized erythrocytes are seen in low numbers, or are absent in 50% of cases) or by identification of schizonts in tissues. Cytauxzoonosis should be considered in animals with anemia, icterus and fever. Cytauxzoonosis is probably spread by arthropod vectors (ticks). Clinical signs include anorexia, conjunctival/scleral injection, constant or increased vocalization, dehydration, dullness, dyspnea, fever, generalized weakness, hepatosplenomegaly, hypothermia, icterus, increased respiratory rate, lymphadenopathy, mucoid nasal discharge, pale mucous membranes, palpably enlarged kidneys, petechiae or ecchymoses, prolapsed third eyelid, red or brown urine, reluctance to move and tachycardia.

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AFIP Diagnosis: Brain, cerebrum; lung: Intramonocytic protozoal schizonts, many, etiology consistent with *Cytauxzoon felis*, domestic shorthair, feline.

Conference Comment: *Cytauxzoon felis* is an apicomplexan intracellular organism in the family *Theileriidae* that usually causes fatal disease in domestic and exotic cats in the south central and southeastern United States. The natural reservoir is the North American bobcat (*Lynx rufus*), which typically has only a subclinical infection. Domestic cats are considered dead-end hosts because the disease is rapidly fatal. In experimental infections, transmission has been through ingestion or

through inoculation of infected blood or tissue or via ticks which have previously fed on infected bobcats.[8]

Cytauxzoon felis has both a leukocytic phase and an erythrocytic phase. The leukocytic (tissue) phase begins when *C. felis* infects mononuclear cells or macrophages and undergoes asexual reproduction, producing schizonts. As the schizonts accumulate and mature, leukocytes enlarge up to 75 μm, often resulting in blood flow obstruction in the liver, lung, lymph nodes, spleen, and bone marrow, leading to severe circulatory impairment. These schizonts then bud, forming merozoites, which lead to further host cell engorgement and cell rupture. Once the merozoites rupture from the host cells, they infect erythrocytes, leading to the erythrocytic phase, which often results in hemolytic anemia. During the erythrocytic phase, the piroplasms are approximately 1-2 μm in diameter, and are ring shaped (signet-ring pattern), and can be identified on peripheral blood smears usually 1-3 days prior to death. Normally only 1-2% of erythrocytes will be affected; however, in moribund cats up to 25% of erythrocytes may be affected.[8]

Gross necropsy findings may include pallor, icterus, petechial and ecchymotic hemorrhages over the surface of the lungs and heart, excessive clear yellow fluid in the pericardial sac, enlarged dark spleen, prominent distended intra-abdominal veins, and swollen, edematous, hyperemic, and sometimes petechiated lymph nodes. The characteristic histologic lesion of cytauxzoonosis, as is present in this case, is the occurrence of numerous intravascular large monocytes with intracytoplasmic schizonts containing multiple small, basophilic, granular bodies (cytomeres) that represent merozoites in various stages of development. The infected monocytes may be found in association with the endothelial lining of venous channels and sinusoids in most major organs, but are usually more abundant in the lung, spleen, lymph nodes, and bone marrow.[5]

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CASE III – CB04-13 (AFIP 2947494)

Signalment: An approximately 37 year-old, sexually intact female rhesus macaque, (*Macaca mulatta*), non-human primate.

History: This macaque was singly housed indoors and tested for 19 years in a neurobiology study involving interactions between catecholamine receptors in the prefrontal cortex and cognitive function. Seven months prior to presentation, a diagnosis of severe spinal osteoarthritis was made. After a 5 month period of biweekly intramuscular injections of a polysulfated glycosaminoglycan (Adequan®) treatment was changed to one month trials of oral nonsteroidal anti-inflammatories, carprofen and deracoxib. Seroconversion to Simian retrovirus was detected 10 weeks prior to presentation. Clinical signs at presentation included a sudden onset of profound lethargy, weakness, hypoglycemia and hypothermia. Treatment with intravenous dextrose, as well as fluid and thermal support resulted in a dramatic rapid improvement. Due to a poor prognosis for extended use, the investigator elected euthanasia and perfusion 4 days later.

Gross Pathology: A 2 cm round, slightly red mass is present within the body of the pancreas adjacent to the duodenum. Within the dorsal aspect of the uterine body a 4 mm round, white, firm raised myometrial mass extended to the perimetrium (serosa). A 0.5 cm paraovarian cyst was present. The liver was rounded with three large foci of hepatic nodules. Scattered, 0.1-0.2 mm, translucent pleural foci were present throughout all lung lobes.

Laboratory Results:
Serum biochemistry: results from a blood sample taken at presentation revealed marked hypoglycemia (9mg/dl; reference range 84-131mg/dl) and relative hyperinsulinemia (51.1 μIU/ml).
Electronmicroscopy: neoplastic cells contain numerous polymorphic granules containing a dense, rectangular, crystalline core separated from the limiting

membrane by a distinct, wide halo. Granules are consistent with those found in beta cells.

Immunohistochemistry: neoplastic cells stained negatively for glucagon and somatostatin; were diffusely moderately positive for chromogranin A and rarely weakly positive for insulin.

Contributor's Morphologic Diagnosis: Pancreas: Islet cell (beta) tumor; insulinoma.

Contributor's Comment: Within the pancreas there is a 2 cm round, discrete, expansile mass compressing the adjacent normal pancreatic tissue. Arranged in variably sized (20 - 500 μm) disorganized nests and clusters, neoplastic cells are suspended in a highly vascular, fatty, loose connective tissue stroma. These cells are 10-20 μm in diameter, round to oval with indistinct cell boundaries. They contain predominantly one and occasionally two, 5 μm diameter, basally aligned, basophilic nuclei with one or more small, indistinct nucleoli. The cytoplasm is amphophilic, abundant and vacuolated. Less than one mitotic figure is seen per 40 power field.

The most frequently described spontaneous neoplasms in nonhuman primates involve those of the digestive system.[1] Although reports of pancreatic tumors are limited, the most commonly described neoplasms involve islet cell adenomas; few of which present with gross lesions.[2] Clinical signs indicative of pancreatic disease have not been seen in any instance of nonhuman primate pancreatic tumors. Neoplasms of the pancreatic islets have been observed in several species including cattle[3], ferrets[4], cats[5], and mice[6]. In dogs, beta cell tumors have been observed in many different breeds and are more frequently carcinomas exhibiting clinicopathological evidence of hyperinsulinism rather than adenomas.[3] In humans, insulinomas, are exceedingly rare (1-4 reported cases per million yearly), but constitute 70-80% of the clinically symptomatic endocrine pancreatic tumors. Greater than 80% of these are solitary masses and, unlike other endocrine neoplasms of the pancreas where malignancy predominates, insulinomas are benign in more than 85% of cases.

The usual histological criteria for malignancy (nuclear pleomorphism, mitotic activity, infiltration of surrounding tissues) are considered unreliable markers in endocrine pancreatic tumors. The accepted parameter for classification of malignancy is local or capsular extension to adjacent organs or a demonstration of vascular invasion and metastases.

Insulinoma cells contain less insulin than do normal beta cells, yet neoplastic tissue has a higher concentration of insulin and proinsulin than the surrounding normal pancreatic cells. The presence of higher serum proinsulin levels in human patients with insulinoma has been attributed to a decreased storage capacity leading to

inappropriate insulin release. Such release at times of low to normal blood glucose levels leads to hypoglycemia and associated neuroglycopenic symptoms (e.g. mental dullness, weakness, abnormal behavior and seizures).

Immunohistochemistry for Chromogranin A, a well established marker for tissues of neuroendocrine origin, is useful in classifying pancreatic neoplasms. Islet cell tumors can stain positive for several immunohistochemical stains and are named according to the hormone responsible for producing clinical signs (insulin for insulinoma).

This rhesus macaque exhibited clinical signs of acute hypoglycemia and a response to appropriate therapy. Concomitant hypoglycemia and elevated blood levels of insulin confirmed hyperinsulinism. In addition, a distinct solitary nodule was present with histological, immunohistochemical, and ultrastructural characteristics consistent with an insulinoma.

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AFIP Diagnosis: Pancreas: Islet cell tumor, Rhesus macaque, primate.

Conference Comment: The contributor provides a thorough overview of insulinomas in various animal species and humans. Pancreatic islets are composed of six distinct cell types, each with specific secretory products that are best distinguished by immunohistochemical techniques. The alpha cells, which produce glucagon, compose approximately 15% of cells in the islets and are usually located at the periphery, but may not be found in all islets. The beta cells, which produce insulin, compose approximately 70% of cells in the islets, and are distributed throughout all islets. The delta cells are present in all islets and have two subtypes; one produces somatostatin, the other produces vasoactive intestinal peptide. Both the gamma cells, which produce pancreatic polypeptide, and the enterochromaffin cells, which produce serotonin, are sparsely and variably distributed.

Most islet tumors comprise a variety of peptide-producing cells and not all islet tumors are associated with clinical manifestations of hormone excess. There is poor correlation between the immunohistochemical profile of islet tumors and clinical disease,[7] with the exception of the adenomas/carcinomas of the insulin secreting beta cells, which are frequently endocrinologically active and are associated with functional disturbances related to hypoglycemia.[8] Another noteworthy exception is the gastrinoma. Although islet cell tumors are rarely responsible for the production of polypeptides with gastrin activity, they have been described in dogs and are associated with the Zollinger-Ellison syndrome. Gastrin is normally produced in the gastric and duodenal mucosa, where it stimulates

glandular secretion. However, pancreatic gastrinomas producing gastrin result in hypergastrinemia, which leads to gastric hyperacidity, mucosal hyperplasia of the antral region, and gastric and duodenal ulceration (Zollinger-Ellison syndrome). [7]

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CASE IV – S 3019/03 (AFIP 2936461)

Signalment: 3 week-old, male, Merino sheep (*ovis aries*), ovine.

History: This newborn lamb was nourished with colostrum only several hours after birth. The lamb suffered from omphalitis and shortening of the flexor tendons of the forelimbs. The animal was referred to the Clinic of Pigs and Small Ruminants, School of Veterinary Medicine, at one day after birth. It developed severe respiratory disease and was euthanatized at 2.5 weeks of age because of poor prognosis.

Gross Pathology: The lungs failed to collapse and exhibited a generalized reddish color and a firm consistency. The cranial lobes and ventral portions of the caudal

lobes showed lobular atelectatic areas. A focal fibrinous exudate was present on the pulmonary pleura. On the cut surface, moderate amounts of a grey viscous fluid were present extruding from the large airways. The bronchial and mediastinal lymph nodes were moderately enlarged. The forelimbs could not be extended completely. One omphalic artery showed subacute, purulent inflammation.

Laboratory Results: Bacteriologic culture of the lung yielded a low degree of non-hemolytic streptococci, *Bacteroides fragilis*, *Pseudomonas aeruginosa*, *enterococci* and *Escherichia coli*.

Contributor's Morphologic Diagnosis: Lung: 1. Bronchitis and bronchiolitis, proliferative and necrotizing, diffuse, subacute, marked, with large basophilic intranuclear inclusion bodies and cytomegalic cells, Merino sheep, ovine, etiology consistent with ovine adenovirus infection.
2. Bronchopneumonia, suppurative, multifocal, subacute, moderate.
3. Pneumonia, interstitial, lymphohistiocytic, multifocal, subacute, moderate.

Contributor's Comment: A large number of bronchiolar and alveolar epithelial cells in this lung section contained large basophilic intranuclear inclusion bodies measuring up to 50 μm in diameter often resulting in cytomegaly with marked enlargement of the cell. In some areas, bronchioles and alveolar spaces are filled with moderate amounts of neutrophils, cellular debris, and numerous sloughed epithelial cells, with few containing inclusion bodies. Hyperplasia of type II-pneumocytes and bi- or multinucleated syncytial cells were found in the alveolar spaces. A severe infiltration of lymphocytes and macrophages was observed in the interstitium. In addition, interstitial hemorrhage and epithelial necrosis appeared in areas, where inflammatory infiltration and sloughing of epithelium was most severe.

Electron microscopical examination revealed intranuclear virus particles arranged in a paracrystalline array consistent with adenovirus infection.

Adenovirions are naked icosahedral particles measuring 60-90 nm in diameter. The genome is a single linear molecule of double-stranded DNA of a molecular weight between 20 and 25 x 10^6.[1] A typical paracrystalline array of virus particles in the nucleus can be found by transmission electron microscopy.[2]

Ovine adenoviruses have been classified into 7 serotypes, six of them are included in the genus *Mastadenovirus* and one in the new genus *Atadenovirus*, both are members of the family Adenoviridae.[3]

After natural infection of adenoviruses via the oronasal route, primary virus replication occurs in the respiratory and intestinal epithelium. Lesions in other

organs, like nasal mucosa, lymph nodes, spleen, kidney and liver are due to a viremia appearing as early as 4 days post infection (p.i.). The virus is shed in the feces, urine and nasal discharge, starting on day 2-3 p.i.. Direct contact between animals is the most important factor for virus spread. Permanent shedding of the virus by recovered animals may occur and contributes to endemics in large farms.[4]

The serotypes differ in virulence and tissue tropisms. In naturally occurring infections, mild respiratory and enteric disease or a subclinical course can be observed.[4,5] Infection of other organs rarely leads to clinic or gross pathologic changes. Histopathologically, hyperplasia of lymphoid follicles in lymph nodes, lung and intestine, tubular degeneration in the kidney, and activation of the reticuloendothelial system in liver and spleen has been described. [4]

The respiratory effects are more severe in experimental intranasal or intratracheal infections. Lesions are characterized by cytomegaly, large intranuclear inclusions and necrosis of epithelial cells in the upper and lower respiratory tract. This is accompanied by early infiltration of neutrophils followed by accumulation of mononuclear cells.[6] Secondary infection with *Mannheimia haemolytica* may complicate signs and lesions.

In natural infections, severe lesions are most likely due to immunodeficiency, and are described in young animals which are deprived of colostral antibodies or which are exposed to environmental stress or other concurrent diseases.[5]

AFIP Diagnosis: Lung: Pneumonia, bronchointerstitial, proliferative, subacute, diffuse, moderate, with multifocal airway epithelial cell and pneumocyte cytomegaly, syncytia, and large basophilic intranuclear inclusion bodies, Merino sheep, ovine.

Conference Comment: Conference attendees discussed differences among slides, with some slides containing plant material in the bronchioles and alveoli. All slides have moderate numbers of both syncytia and multinucleated giant cells, which are not typical of adenovirus pneumonia. The multinucleated giant cells, primarily of the Langhans' type, may be due to a secondary bacterial infection, which is not uncommon with adenoviral pneumonia. However, no organisms were identified with tissue Gram stains.

Adenoviruses have been isolated from most animal species and humans. They have differing virulences and tissue tropisms, but frequently cause respiratory and enteric disease. Severe naturally occurring disease is usually seen only in

immunodeficient animals. The most prominent lesion of the pneumotropic strains is a necrotizing and proliferative bronchiolitis.[5]

The family *Adenoviridae* now contains four recognized genera. The genus *Mastadenovirus* contains most of the mammalian adenoviruses. The genus *Aviadenovirus* contains the group I avian adenoviruses. The genus *Siadenovirus* contains the group II avian adenoviruses and frog adenovirus. The genus *Atadenovirus* contains the group III avian adenovirus and several mammalian viruses.[7] Some recognized adenoviruses include the following:[3,7]

Virus	Disease
Mastadenovirus	
Canine adenovirus-1	Infectious canine hepatitis
Canine adenovirus-2	Infectious canine tracheobronchitis
Equine adenoviruses-1,2	Mild respiratory disease (except CID foals)
Bovine adenoviruses-1,2,3,9,10	Enzootic pneumonia (one of many agents)
Ovine adenoviruses-1-6	Mild respiratory and enteric disease
Goat adenoviruses-1,2	Mild respiratory and enteric disease
Porcine adenoviruses-1-5	Enteritis and encephalitis
Guinea pig adenovirus-1	Adenoviral pneumonitis
Mouse adenovirus-1,2	Enteritis and encephalitis
Simian adenoviruses-1-25	Mild respiratory and enteric disease
Human adenoviruses-1-51	
Aviadenovirus (subgroup I avian adenoviruses)	
Fowl adenoviruses-1-11	
Fowl adenovirus-1	Inclusion body hepatitis (chickens)
Fowl adenovirus-4	Hydropericardium syndrome (chickens)
Goose adenoviruses-1-3	
Siadenovirus	
Frog adenovirus-1	
<u>Subgroup II avian adenoviruses</u>	
Turkey adenovirus-3	Hemorrhagic enteritis (turkeys)
Pheasant adenovirus-1	Marble spleen disease (pheasants)
Atadenovirus	
Ovine adenovirus-7	Mild respiratory and enteric disease
Bovine adenoviruses-4-8	Enzootic pneumonia (one of many agents)
Black tail deer adenovirus-1	Pulmonary edema, hemorrhage, vasculitis
Possum adenovirus-1	
<u>Subgroup III avian adenovirus</u>	
Duck adenovirus-1	Egg drop syndrome (chickens)

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