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A Review of the Feline Respiratory Viruses

Richard D. Jensen*

Introduction

The disease most stressed in this review will be feline pneumonitis. It was decided to concentrate on this disease because it represents a fairly well defined disease entity. For this reason, it can be more adequately described and discussed than the more recently discovered feline respiratory viral diseases. Also, many of the more recently isolated feline respiratory viruses are related to the virus of feline pneumonitis. Feline pneumonitis will be the first disease described and the other feline respiratory viruses will then be described and compared to it.

Feline Pneumonitis

Etiology: The etiological agent of feline pneumonitis is a virus, *Miyagawanella felis*, of the lymphogranuloma-psittacosis group of viruses. It belongs to the family Chlamydozoaceae which is the third family of the order Rickettsiales. The order Rickettsiales is composed of microorganisms which are minute, pleomorphic, and coccoid. They are obligate intracellular parasites. Members of the genus *Miyagawanella* pass through a developmental cycle from the individual cell to the formation of the elementary body. The presence of both types of nucleic acid and the sensitivity to antibiotics suggests the presence of enzyme systems. Binary fission is probably the method of reproduction. The organisms can be found in the cytoplasm of infected cells. Growth can be obtained in the chick embryo. The organism has remained viable, when dried, for six months but heating for ten minutes at 60°C. inactivated the organism. Mice, hamsters, guinea pigs, and rabbits, as well as cats, are susceptible to the virus when inoculated intranasally. The virus produces an endotoxin which is demonstrable in yolk sac suspensions from moribund embryos. The virus of feline pneumonitis is not susceptible to sulfonamides. Other members of the psittacosis-lymphogranuloma group vary in their sensitivity. The organism is more closely related to the meningiopneumonitis virus than to the agent of mouse pneumonitis. This was determined by a comparison of the tropism of the feline pneumonitis virus with the other viruses of the group. The lack of sensitivity to the sulfonamides exhibited by the feline pneumonitis virus was also used. It can be distinguished from the virus of meningiopneumonitis by (a) its toxin and corresponding antitoxin and (b) by the establishment of intracerebral infections in mice with the meningiopneumonitis virus while infection with feline pneumonitis occurs only when a relatively large amount of inoculum is used.

Transmission: Cats inoculated intracerebrally and intraperitoneally with the virus showed no external signs of the disease but the disease is readily spread by intranasal inoculation and readily passed from inoculated to uninoculated cats when they are placed in contact. The virus was present in ocular and nasal discharges during the course of the disease. It was possible to recover the virus from the nose for one to two months after recovery.

Symptoms: The first symptom seen was a slight increase in body temperature. The increased temperature was of short dura-
The first marked manifestations of illness developed six to ten days after exposure. They consisted of photophobia, lacrimation, and a mucopurulent nasal discharge. Prolonged attacks of sneezing frequently occurred, especially after the cats were handled. A cough usually developed and this usually indicated the presence of pneumonia. Symptoms persisted for one to two weeks. Recovery occurred in all cats in which the disease was allowed to run its course. Emaciation was noted shortly after the onset of the disease and the cats did not appear normal until one month after the onset of the disease. 

Post Mortem: Inflammation of the conjunctival and nasal mucosa was seen. Inflammation of the lower respiratory tract was usually present. The larynx and trachea were inflamed and thick, cloudy mucus was present. The pneumonia was characterized by consolidation of areas of the anterior lobe of the lung. The diaphragmatic lobe was occasionally involved. The bronchial lymph nodes were not markedly enlarged. An occasional cat showed slight splenomegaly. Other internal organs were not affected.

Microscopic sections showed that the bronchial epithelium was undamaged. The alveoli were filled with a serous and cellular exudate and occasional areas of necrosis were present. The cytoplasm of the mononuclear cells contained elementary bodies. These were seen when Giemsa's stain was used.

Immunity: The complement fixation test was used to detect the presence of antibodies. Antibodies were found to be present in the serum of recovered cats more than two months after recovery.

Variants of the Feline Pneumonitis Virus

Greenland, in 1961, developed a non-lethal mutant of the feline pneumonitis virus. The virus was penicillin resistant. It was isolated by limiting diluted passages of the feline pneumonitis virus in chick embryo yolk under varying conditions. It resembled the original virus except that it was unable to kill the embryo in the presence of penicillin. The author speculated that a shift in the susceptibility of toxin production to penicillin might be involved. In other words, no toxin was produced in the presence of penicillin and the embryo remained unharmed. Another theory, advanced by the author, Greenland, was that the toxin was produced but it was not released due to changes in the cell wall permeability as a result of the presence of penicillin.

Woodroofe and Moulder also reported on non-neutralizable variants of the feline pneumonitis virus. They concluded that there are many closely related variants of the pneumonitis virus which could be isolated under selective conditions. All of these variants, however, were insensitive to penicillin and to neutralizing antisera for feline pneumonitis. These variants were termed "spread variants." They all grew to a low titer and killed embryos over a five to ten day period, as compared to one to two days for the virus from which they were derived. The authors speculated that the variants probably represented a phenotypic change controlled by an unknown number of loci affecting the surface of microorganisms and causing them to become unreactive to penicillin or neutralizing serum. The spread variants were obtained from the feline pneumonitis virus with one yolk sac passage in the presence of penicillin and chloromycetin, penicillin and quinoxaline oxide, quinoxaline oxide alone, or neutralizing antiserum. The authors concluded that when growth conditions became unfavorable the spread viruses became dominant even though the normal virus has the advantage under normal growth conditions.

Yerasimides isolated a new strain of feline pneumonitis virus from the conjunctiva of a domestic cat. The virus was compared to the feline pneumonitis virus of Baker's strain. It possessed higher toxin neutralizing and lower serum neutralizing power than the Baker virus. It was concluded that the virus was related morphologically, developmentally, and antigenically to the psittacosis-lymphogranuloma group. It was named the 111-18 strain. It could not be distinguished from the Baker virus on the basis of complement fixation, hemagglutination, developmental cycle, morphology, growth in eggs and mice, and its susceptibility to antibiotics and sulfona-
mides. Both viruses were susceptible to penicillin, chloromycetin, and Terramycin. They were not affected by sulfathiazole, sulfamerazine, streptomycin, and bacitracin. The toxins of the viruses were not affected by any of the antibiotics. The virus was presented as a new strain of the feline pneumonitis virus.

Other Feline Respiratory Viruses

Feline Viral Rhinotracheitis: The virus of feline viral rhinotracheitis can be grown in tissue culture in which it produces intranuclear inclusion bodies. The inclusion bodies are produced at the time of release of new extracellular virus. The symptomatology of the disease is as follows. The agent was readily transmitted by intranasal inoculation, as well as by natural spread. The first symptom was a slight temperature increase on the second day after inoculation. On the third day after inoculation the temperature had risen to 104°F. The temperature increased when the virus entered the cells. The affected animals were depressed. Laceration and a nasal discharge were present. Anorexia and coughing were present on the third to the sixth day after inoculation. The nasal and ocular discharges became mucopurulent. Recovery occurred in two weeks if the anorexia was overcome by the fifth day. Death occurred if the anorexia was not overcome. The postmortem lesions caused by feline viral rhinotracheitis were as follows. Inflammation of the larynx and trachea were the main lesions seen. Focal necrosis and a purulent exudate were present within six days after inoculation. The cervical lymph nodes and tonsils were enlarged. Emaciation and dehydration with the subsequent absence of subcutaneous fat were an important postmortem finding. Histopathological examination showed the presence of intranuclear inclusion bodies in the epithelium of the upper respiratory tract. The cells of the nasal turbinates, nasal septum, and the pharyngeal region contained intranuclear inclusion bodies. Most inclusion bodies were present on the second day after inoculation. Some were present on the fifth day after inoculation. Inclusion bodies were also found in the cells of the trachea. They were also demonstrated in the cells of the nictitating membrane. No other significant lesions were seen with the exception of an acute septicemia of short duration of the liver.

Immunity against feline viral rhinotracheitis was investigated using the serum neutralization test. When the animals were challenged one month later a slight immune response was obtained. The virus was found to persist in the upper respiratory tract for up to fifty days after inoculation.

The most obvious differences between the diseases caused by the feline viral rhinotracheitis virus and those caused by the feline pneumonitis virus are the absence of lung involvement with the former and the presence of intracytoplasmic inclusion bodies with feline pneumonitis and intranuclear inclusion bodies with feline viral rhinotracheitis. Also, the inclusion bodies of feline pneumonitis are in the monocytes in the alveolar exudate while those of feline viral rhinotracheitis are in the mucosal cells. Both diseases were characterized by a rather long course and recovery generally occurred with both diseases.

Crandell and Madin isolated what they considered to be a new feline virus. They named it the California feline isolate or C. F. I. virus. It was capable of producing a nonfatal respiratory involvement in cats. Three groups of cats were inoculated with the virus after increasing serial passages in tissue culture. The first group of cats was inoculated with the third tissue culture passage. The only symptom seen was an increased temperature between the fourth and fifth day with the exception of one cat which showed a nasal and ocular discharge. The second group of cats was inoculated with the twelfth tissue culture passage of the virus. The second group also showed an increased temperature. Cats which had been inoculated intranasally showed nasal and oral ulcers. The temperature peak was reached on the third day. A mucous to mucopurulent nasal and ocular discharge was present. There was some blood seen in the feces. The third group was inoculated with the thirti-
eth tissue culture passage of the virus. This group showed no clinical response except a slight temperature increase on the first day. It was thus demonstrated that virulence was increased by serial passage in tissue culture up to the twelfth passage, but after thirty passages no clinical response could be elicited even though an antibody titer was produced. No change was seen in the cytopathic effect on tissue culture cells.

The C. F. I. virus was compared with the feline pneumonitis virus of Baker and the following differences were seen. The feline pneumonitis virus formed elementary bodies characteristic of the psittacosis-lymphogranuloma group of viruses in chick embryos and mice. The C. F. I. virus would not grow in chick embryos or mice. Also, the C. F. I. virus did not form elementary bodies in tissue culture. Immune serum against feline pneumonitis had no effect against the cytopathic effect of the C. F. I. virus on feline kidney cell tissue cultures.

The C. F. I. virus was also compared with the feline viral rhinotracheitis virus. The feline viral rhinotracheitis virus produced intranuclear inclusion bodies but the C. F. I. virus did not. Also, no relationship was demonstrated in cross immunization test. (4)

Torlone(15) isolated a cytopathic agent from the eye and nasal mucosa of a kitten that had died due to an upper respiratory infection. It was isolated on a feline kidney cell tissue culture. After eight passages the stock virus was tested. It was transmitted to laboratory cats by intraocular and intranasal inoculation. Some of the cats died of pneumonitis. When the virus was tested by the serum neutralization test against other viruses the results were negative. The virus was rapidly spread in the natural environment. It was possible to make an immune serum. The virus was cytopathic in from four to forty-eight hours and formed paranuclear inclusion masses.

Bittle and his co-workers(2) isolated a series of antigenically different viruses from the feline upper respiratory tract and conjunctiva of cats showing clinical symptoms of respiratory disease, with the exception of three cats. The viruses were cytopathic. It was concluded that they were potential disease producers due to the inability of the investigators to isolate cytopathic effect producing viruses from clinically normal cats. Wide spread infection by some of these agents was believed to exist on the basis of neutralizing antibodies present in the serum of kittens and older cats tested. The viruses were antigenically separate even though a low titer cross relationship existed between a number of them.

Conclusion and Summary

The purpose of this paper has been to review the literature concerning the feline respiratory viruses. The basic plan was to describe and discuss feline pneumonitis and then describe the other feline respiratory viruses and compare them with the feline pneumonitis virus and the disease it produced.

Even such a relatively cursory study, as for this review, discloses that the feline respiratory viruses present an extremely complex picture. As investigations in this field have been carried on, more and more viruses have been discovered. The methods for investigation of these viruses have varied with the different investigators. Some stressed growth characteristics of the viruses while others stressed the antigenic relationships among viruses and still others, at least in the present stages of their investigations, stressed the clinical manifestations caused by the viruses.

The possibility of vaccine production should be considered. One group of investigators, Bittle and his co-workers, (2) suggested that because of the relationship shown by low titer cross relationship among a number of the viruses, it might be possible to produce an immunizing agent offering protection against some of the viruses.

The importance of these viruses when compared to other feline viral diseases should also be considered. The effects of the feline respiratory viruses are confined almost entirely to the respiratory system while some other feline viral diseases have more generalized manifestations. (12)

From the standpoint of diagnosis, it
would be virtually impossible to differentiate the different respiratory viruses on the basis of symptomatology. Less difficulty would be encountered in differentiating the viral respiratory diseases from other feline viral diseases. For example, the virus of panleukopenia causes a decreased leukocyte count and intestinal lesions (10) but feline pneumonitis is accompanied by leukocytosis and no intestinal manifestations are seen.

In conclusion, all of the feline respiratory viruses have not been discussed in this paper, nor was it the intention of the author to do so. Neither was one or two viruses covered in depth to the exclusion of the other respiratory viruses. The primary aim of this paper was to review the field by the device of concentrating primarily on one virus which had been comparatively well studied, the feline pneumonitis virus, and then discussing the other viruses in relation to it.

REFERENCES

Artificial Insemination of a Commercial Beef Herd

Douglas Hageman*

Although artificial insemination (A.I.) was initially organized as a means of bringing the service of superior purebred dairy sires to average dairy farms with grade cattle, it has been widely used by breeders of registered purebred cattle. The purpose of this article is to describe the successful use of artificial insemination in a commercial beef cow-calf operation in central South Dakota.

Since genetic improvement has been shown through the use of artificial insemination, the same was expected in this herd. The advantages expected when this herd was started on a program of artificial insemination were: (1) increased weaning weights, (2) genetic improvement in the herd through better replacement heifers and a more uniform set of calves, (3) advantages of using proven sires, and (4) reduction in the number of herd bulls required.

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