New Feline Viruses: A Review of Their Designations and Significance

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The purpose of this communication is to clarify the present confusion existing in the designation of the newly reported feline viruses and comment briefly on their significance in regard to feline respiratory diseases. Unfortunately, during the exchange of many of these viruses between investigators, their original laboratory designations were maintained and eventually appeared in various publications.

It is not our intention to classify these viruses. rather to review them and associate each isolate with its proper letter-number designation. Since there is renewed interest in some of these viruses, it is important that their true identity be known. It is hoped that this report will prevent further confusion in the identity of these agents.

For convenience in discussing these newer viruses, we have arbitrarily placed them into two groups according to their cytopathology in tissue culture.

1. Those associated with inclusion bodies (feline herpesvirus).
2. Those in which inclusion bodies or elementary bodies have not been demonstrated.

Only those viruses reported since 1957 are discussed in this paper. Prior to that time only four confirmed viruses of the cat were described; these are: rabies, pseudorabies, feline panleukopenia and feline pneumonitis. Although reports of other virus diseases appeared in the literature, it is our opinion that their true identity has yet to be established.

There are 20 reported isolates in the American literature in which inclusion bodies are not associated with their cytopathology (Table I). Of these 20 viruses in Table I, nine (KCD, FR, FC, FS, FI, FJ, FPL, F-17 and C-14) have been shown by serum neutralization studies to be distinct viruses. The remaining 11 have not been completely tested with all members of this group.

Of the numerous feline virus isolates listed in Table I, all but three were obtained from the oropharngeal region or the conjunctival membranes of the domestic cat. These three were isolated from the spleens of cats ill with panleukopenia. The KCD virus was the first to be reported from the spleen by Fastier (1) in 1957 when he was attempting to propagate panleukopenia virus in tissue culture. Bolin (2) recovered his agent from the spleen under similar circumstances. The F-20 agent was also isolated from the spleen. It is not uncommon to isolate many of these viruses from both the upper respiratory tract and conjunctiva from the same animal.

Experimental transmission studies with these viruses have been very limited and their importance as disease producing agents is unknown. Fastier, in studies to

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TABLE I. New feline cytopathogenic viruses reported in the American literature which are not associated with inclusion bodies

Designation Name	Letter-Number	Original Reference	Isolated by
Kidney Cell Degenerating Virus	KCD	(1)	Fastier
Bolin Virus	FVI, FPL, FPL-FM	(2)	Bolin
Feline Respiratory Isolate	FRI-14, C-14	(14)	Crandell
Feline Respiratory Isolate	FRI-29, C-29	(14)	Crandell
Feline Respiratory Isolate	FRI-278	(14)	Crandell
Feline Respiratory Isolate	FRI-12	(14)	Crandell
Feline Respiratory Isolate	FRI-6	(14)	Crandell
California Feline Isolate	CFI, FIV, FI	(5)	Crandell
Feline Respiratory Virus	FR, FRV	(15)	Madin
Feline Conjunctivitis Virus	FC, FCV	(16)	McKercher
Feline Stomatis Virus	FS, FSV	(16)	McKercher
Respiratory Virus	TC-15*	(16)	York
Enteritis Virus	FPV	(16)	Jen Sal
	FJ, F-20	(15)	Emery
	F-5	(15)	Bittle
	F-9	(15)	Bittle
	F-10	(15)	Bittle
	F-11	(15)	Bittle
	F-17	(15)	Bittle
	F-19	(15)	Bittle

*The identity of this isolate has been lost.

produce illness with his KCD agent, was unsuccessful and chose to refer to his virus as an orphan virus. Although Bolin reported positive results in producing panleukopenia with his virus, it has not been confirmed as being the virus of feline panleukopenia. On the contrary, the virus has been shown by several investigators as not related to the feline panleukopenia virus.

Crandell (unpublished data) has produced clinical cases of panleukopenia by challenging cats hyperimmune to the Bolin virus with infected spleens from fatal cases of panleukopenia. In addition, immune panleukopenia antiserum did not neutralize the Bolin agent in extensive tissue culture studies. Cohen, et al (3) compared the Bolin virus with panleukopenia in vivo and vitro and found the two viruses to be different. These authors did, however, observe ascites in some kittens 7-10 days following inoculation with the Bolin virus.

In another study by Bittle, et al (4) they produced in kittens a fever of about 5 days duration with transient mild leukopenia, depression, anorexia followed by rapid recovery with the Bolin virus. The F-20 virus produced a similar but milder illness, while the KCD virus did not produce overt illness.

These authors also demonstrated no antigenic relationship between the three spleen isolates (KCD, Bolin virus, F-20) and the virus of panleukopenia.

Crandell and Madin (5) reported that their agent, designated as CFI, produced a mild upper respiratory illness in kittens. Like the other agents, further studies are needed to determine its full significance as a pathogen for the domestic cat.

The isolates in Table II have been clearly shown by neutralization tests to be antigenically similar to the original virus reported by Crandell and Maurer (6). There is sufficient evidence showing that there is no serological relationship between these viruses and those in Table I.

Crandell and Despeaux (7) proposed the name "feline viral rhinotracheitis" as the name for the disease caused by this virus. The virus has been shown by various investigators to possess properties characteristic of the herpesvirus group.

Transmission studies have clearly shown that the FVR virus produces an upper respiratory infection in cats (6) (8). These investigators have reproduced the illness and demonstrated intranuclear inclusion bodies in the epithelial cells in the nictitating membrane, trachea and turbinates similar to those seen in tissue cultures infected with the virus. The frequency with which this virus has been recovered from clinically ill cats lends support that it is the cause of a commonly occurring disease in cats.

In addition to those feline viruses appearing in the American literature (Table

TABLE II. Feline cytopathogenic viruses (feline herpesvirus) reported in the American literature since 1958 which are associated with intranuclear inclusion bodies and have been shown to be antigenically similar

Letter-Number Designation	Original Reference	Isolated by
C-27, FVR-1	(6)	Crandell
FVR-2, NIH	(17)	Crandell
FVR-3	(17)	Crandell
P-001, PV	(17)	Gorham
F-1	(15)	Bittle
F-2	(15)	Bittle
F-14	(15)	Bittle
F-16, 3402	(15)	Bittle
F-18	(15)	Bittle
FRV	(18)	Ditchfield

I and II), several European investigators have reported the isolation of cytopathogenic agents from the conjunctiva and nasal mucosa of the cat. Because of their importance in the final grouping or classification of the feline viruses, their inclusion in this report seemed appropriate. They are also grouped according to their cytopathology in tissue culture in Table III.

Torlone (9) isolated a virus from a cat dying of an upper respiratory disease in Italy. The observed cytopathic effects in feline renal cell cultures are similar to those viruses in Table I. (The virus was compared to FVR, FS, FC, KCD, F-1, F-17 and 3402 and was shown to be serologically distinct.)

Burki (10) reported the isolation of 14 cytopathogenic agents from ill cats in Switzerland. Seven of these were grouped by Burki according to their cytopathology in tissue culture. The cytopathology resembles that produced by the viruses in Table I. These viruses were further grouped by serological means into 5 types. More recently, Burki (11) described these viruses as having characteristics of the picornaviruses.

The other seven viruses were associated with intranuclear inclusions and possessed the properties of the viruses belonging to the herpesvirus group. These were shown by Burki, et al (12) to be related serologically to the American strain of FVR.

Piercy and Prydie (13) reported the isolation of 25 virus strains from cats in England. These strains comprised more than one antigenic type. Although these authors associated their agents with "feline influenza," the viruses reported do not possess characteristics of the myxoviruses.

CONCLUSION

1. The viruses listed in Table I are probably of different serologic prototypes, although they elicit low serologic cross relationships.
2. Comparative studies with European isolates are necessary before the actual number of distinct viruses is known.
3. Additional studies are needed before their full significance as feline pathogens can be determined.
4. Additional knowledge regarding their physio-chemical properties is necessary before they can be properly classified.
5. Studies have shown that the Bolin virus is not related to feline panleukopenia virus.
6. The feline viral rhinotracheitis isolates are serologically similar and all possess characteristics common to the herpesvirus group.
7. The fact that virus isolations have

TABLE III. New feline cytopathogenic viruses reported from Europe (1960 - 1964)

Viruses Not Associated With Intranuclear Inclusions		Viruses Associated with Intranuclear Inclusions (feline herpesvirus)	
Designation	**Original Ref.**	**Designation**	**Original Ref.**
VGP	(9)	86/61	(10)
337/61	(10)	87/61	(12)
344/61	(10)	91/61	(12)
346/61	(10)	149/61	(12)
377/61	(10)	156/61	(12)
378/61	(10)	227/62	(12)
135/62	(10)	292/62	(12)
227/62	(10)	297/62	(12)
"Influenza Virus"	(13)		

been made in New Zealand, United States of America, Canada and Europe suggests that these viruses possess a wide geographic distribution.

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Isolation of an Unidentified Hemagglutinating Virus from the Respiratory Tract of Turkeys

A hitherto unrecorded virus, tentatively named Wilmot virus, has been isolated from Ontario turkey flocks. It was obtained on only one occasion from the tracheas of birds showing signs of respiratory infection accompanied by a severe drop in egg production.

Respiratory symptoms, rales and coughing, were heard when the flock was disturbed and lasted 3 to 4 days. Greenish diarrhoea was evident at this time. Mortality was low (10 birds out of 1800) but egg production dropped from 50% to a low of 20%. Egg shell quality deteriorated with the decline in egg production and hatchability dropped from 72% to 42%.

Turkeys necropsied at the height of infection showed a moderate tracheitis with excess mucus. Catarrhal enteritis was a constant finding and several of the dead birds had an egg peritonitis.

The virus was isolated in the allantoic fluid of chick embryos, for which it was lethal, and was found to hemagglutinate chicken red blood cells.

Serological evidence of its presence in 4 additional flocks was obtained. It was transmissible to chickens and apparently spreads by contact.

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