Feline Infectious Peritonitis: A Worldwide Serosurvey

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SUMMARY

Feline sera from 13 countries were assayed for coronavirus antibody, using a heterologous indirect immunofluorescence test. Significantly higher percentages of antibody carriers were obtained during testing randomly collected sera from mature males (> 1 year old) than in testing females of the same age. Antibodies were infrequently found in immature cats (< 6 months old); at 1 year of age or older, a plateau was reached and little change in the percentage of seroconverted animals was observed. Differences were not detected between purebred cats vs mixed-breed cats or household vs stray cats. In animals showing clinical signs of feline infectious peritonitis (FIP), antibodies were encountered with higher frequency than in clinically healthy cats. Significant differences in antibody incidence were found between countries, with a range between < 10% and > 50% of seropositive individuals. Antibodies were detected in sera from an isolated cat population (Marion Island) and from wild-caught cheetahs (*Acinonyx jubatus*). The antibody specificity for FIP virus was confirmed by neutralization tests. The antibody pattern in randomly collected solitary cats, in catteries, and cats with clinical FIP showed characteristic differences in titer and incidence. The implications of these results for the epizootiology of FIP are discussed.

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The frequency of antibody to feline infectious peritonitis (FIP) virus in the feline population may vary considerably; Pedersen[1] reported an occurrence of 20% seropositive cats in samples collected at random in the Davis, Calif area, and Loeffler et al[2] reported an occurrence rate of 41% in the Pullman, Wash area. We detected an occurrence rate of 16% in the Dutch cat population,[3] using a heterologous indirect immunofluorescence test with TGE virus-infected cells as an antigen source.

The purpose in the present report was to compare the incidence of antibody carriers in different parts of the world; the age, sex, and breed frequency distribution of antibody; and its correlation with disease signs other than those of peritonitis. Noncat felidae were also tested, as well as samples from catteries, multiple cat households, and animal hospitals with and without a history of clinical FIP cases.

Materials and Methods

Sera—In October 1977, a letter was sent to the small animal departments of about 70 veterinary schools or faculties in the five continents, explaining the aim of a geographic seroepizootiologic survey of FIP, and requesting a serum sample from each department. Letters were not sent to the United States. The specimens collected by June 1978 were assigned to four groups designated: (i) random sample, 505 (ii) noncat felines, 12 from cheetahs, 9 from other wild feline species; (iii) catteries, hospitals, multicat households, 86; and (iv) suspect and confirmed FIP cases, 16 from abroad, and 21 from our Dutch collection. A random sample was defined as a serum sample from a single household or stray cat without a suspicion or history of FIP; animals with conditions other than peritonitis were also considered as random. All available data on the serum donors were keyed into punch cards for convenient sorting; their distribution is given (Tables 1 and 2; Fig 1).

A special comment should be made on the sera indicated "South Africa" in Table 2 and Figure 2; these samples were collected on Marion Island (46°55′ South, 37°45′ East, size 18 × 22 km), a member of the Prince Edward Islands group which has belonged to South Africa since 1947 (the island is currently used as a weather station). Three female and two male cats were brought to the island in 1948 by a team of scientists and have multiplied to a current population of 4,000 to 6,000 animals. Genetic studies indicate that these cats originated from southern Africa, although there is limited evidence to suggest the animals might have originally been introduced to that country by oceangoing vessels in the middle of the last century. Whatever the origin of this cat population, its isolation is unique; apart from the visit once or twice a year of a supply ship, the island has remained isolated.[a]

TABLE 1—Comparative Listing of Antibody Incidence (Present Serosurvey)

Item	Titer < 10	10	30	60
Breed				
Pure-bred	49 (16)	21 (7)	21 (7)	9 (3)
Mixed-breed	53 (250)	19 (91)	17 (81)	11 (50)
Habitat				
Household	54 (249)	18 (81)	17 (80)	11 (49)
Stray	37 (17)	37 (17)	17 (8)	9 (4)

Values expressed as percentage of antibody incidence; (n) = absolute values.

Indirect Immunofluorescence Test (IFT)—The procedure followed was essentially the same as reported elsewhere[3]; instead of transmissible gastroenteritis (TGE) virus-infected porcine thyroid cells, however, antigen-containing cells of the porcine PD5 line[b] were dried onto epoxy-coated microprint slides.[c] Titers of 30 and higher were considered positive.

FIP Virus-Neutralization Test—The virus-neutralizing activity in selected cat and cheetah sera was assayed as described elsewhere,[4] using 100 mouse ID_{50} units of mouse brain-adapted FIP virus and a 1:10 dilution of the heat inactivated (30 minutes at 56 C) serum. At 7 days after intracerebral inoculation of the mixtures into

[a] Howell PG, Onderstepoort, South Africa: Personal communication, 1978.

[b] Philips Duphar, Weesp, The Netherlands.

[c] Cooke Engineering Co, Alexandria, Va.

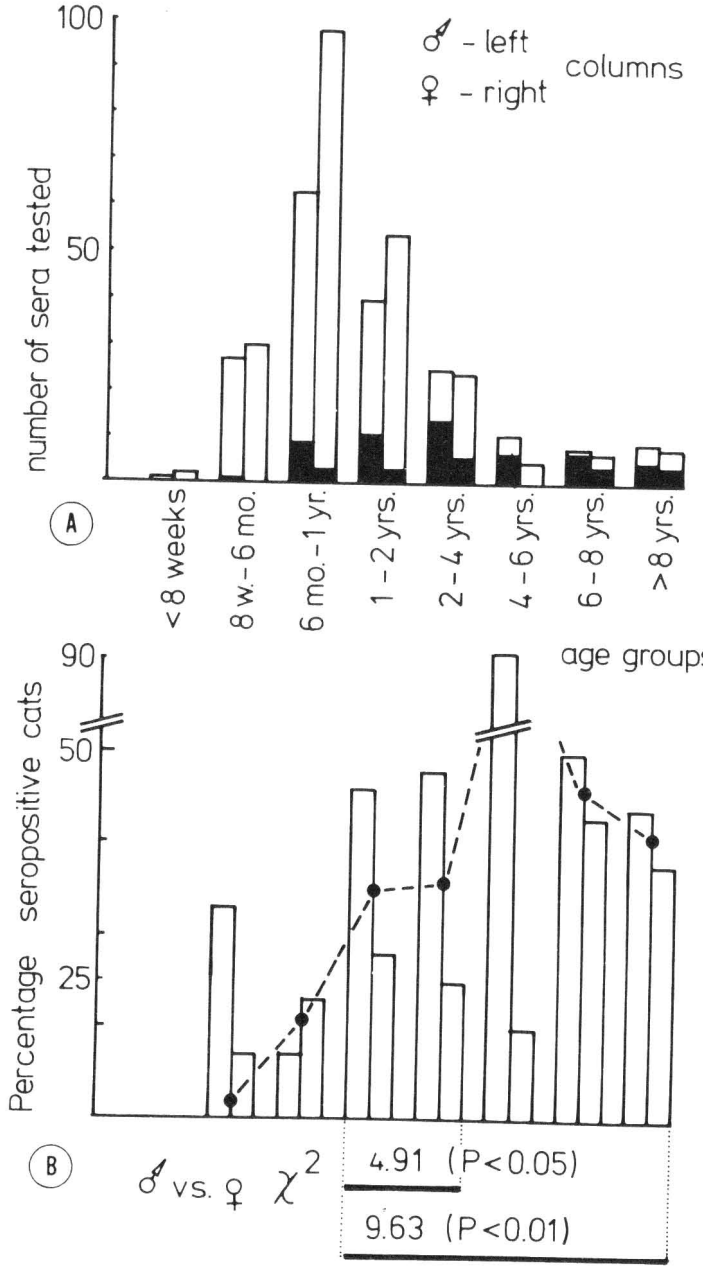

Fig 1—The sex and age distributions of randomly collected feline sera (A) and the incidence of antibody (titers ≥ 30), as determined by indirect IFT (B).

(A) The black columns represent castrated males (left) and spayed females (right).

(B) The interrupted line represents the total seroconversion percentages per age group. The X^2 values for the male:female differences are given for animals between 1 and 4 years of age and older than 1 year. The X^2 value for the male:female comparison of the whole sample volume was 1.99 (difference not significant).

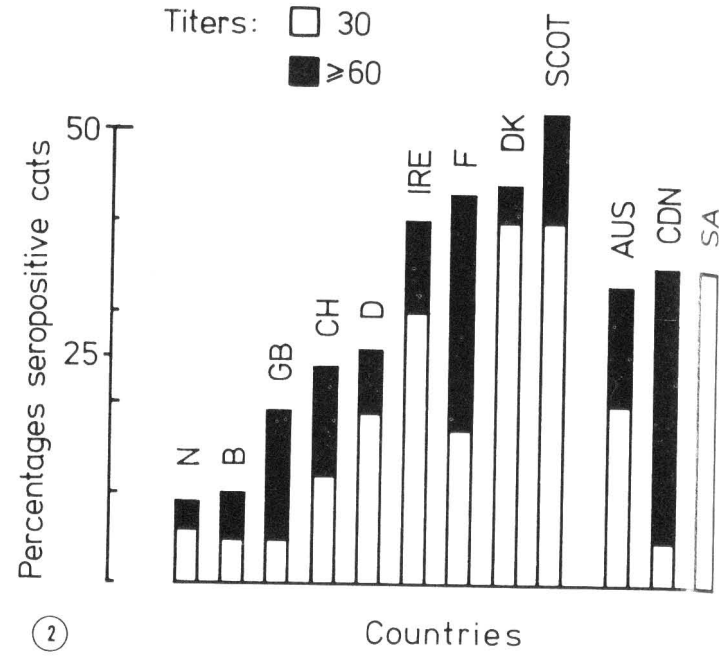

Fig 2—Incidence of seropositive cats in different countries. N = Norway; B = Belgium; GB = England; CH = Switzerland; D = Germany, Federal Republic; IRE = Ireland; F = France; DK = Denmark; SCOT = Scotland; AUS = Australia; CDN = Canada; SA = Marion Island, South Africa.

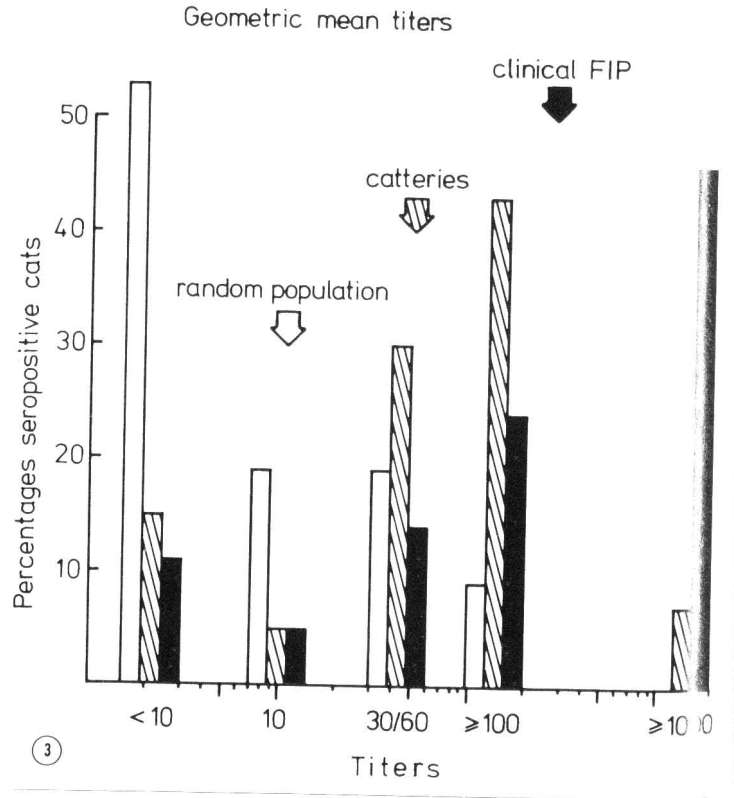

Fig 3—Frequency distribution of antibody titers in the feline random population (n = 505), in crowded cat groups (n = 86) and in animals with clinical FIP (n = 37).

1-day-old mice, the animals were killed and the brain was examined for the presence of viral antigen, using a direct IFT with fluorescein isothiocyanate-conjugated ascites fluid from an animal with FIP.

Results

Correlation of Sex, Age, Breed, Habitat, and Symptoms with Seroconversion (Random Sample)—The sex/age distribution of the collected cat sera is shown (Fig 1A). The prevalence of samples from females between 8 weeks and 2 years of age is due to animals submitted for spaying (103 females as compared with 32

TABLE 2—The Geographic Distribution of FIP (Published and Unpublished Evidence) and of Seropositive Cats

Country	Published data	Present serosurvey: Correspondent or source of test samples	Town or region	Serosurvey test result (%)	Additional information of FIP frequency
Australia	Jones and Hogg[5]	VP Studdert	Melbourne	19/47 (40)	< 1 case/year
	Watson, Huxtable, and Bennett[6]	ADJ Watson	Sydney	4/22 (18)	1-2 cases/year (600 admissions)
Belgium	Pastoret, Gouffaux, and Henroteaux[7]	PP Pastoret	Liège	6/55 (11)	Commonly diagnosed
		D Mattheeuws	Ghent	—	Occasionally diagnosed
Canada	Stevenson, Tilt, and Purdy[8]		Quebec	1/6	
			British Columbia	1/4	
		RC Povey, Guelph		(33)	Repeatedly diagnosed
			Newfoundland	3/5	
			Ontario	2/5	
Denmark	Flagstad and Larsen[9] Mortensen and Steensborg[10]	A Flagstad	Copenhagen	11/28 (39)	Increasingly diagnosed (1977)
England	Ishmael and Howell[11] Wilkinson[12]	JE Jones	Liverpool	4/21 (19)	Occasionally diagnosed
France	Auclair-Semere and Groulade[13]	JF Guelfi	Toulouse	13/30 (43)	Rarely diagnosed
	Chappuis, Brun, and Corbran[14]	B Toma	Paris (Maison Alfort)	—	Not uncommon
		C Duret	Lyon	—	Diagnosed once
Germany	Tuch, Witte and Wüller[15]	LF Müller	Berlin	7/12 (58)	Not frequently diagnosed
		I Schütt	Hannover	7/42 (17)	Not frequently diagnosed
Hungary	—	B Lomniczi Z Horváth	Budapest	6/10 (60)	Not diagnosed
Iran	—	B Yamini	Teheran	—	Diagnosed once
Ireland	Hartigan and Wilson[16]	WR Kelly	Dublin	12/30 (40)	Not frequently diagnosed
Japan	Konishi, Takahashi, and Ogata[17]	—	Tokyo	—	About 6 cases/year
Kenya	—	JE Price	Nairobi	—	Not diagnosed
Mexico	de Aluja[18]	AS de Aluja	Mexico D.F.	—	Diagnosed once
Netherlands	Mieog and Richter[19]	E van Ooyen	Utrecht	11/69 (16)	~12 cases/year
New Zealand	—	BR Jones	Palmerston	—	About one case/year (since 1972)
Nigeria	—	JB Adeyanju	Zaria	—	Two doubtful cases
Norway	—	B Hyllseth	Oslo	3/34 (9)	Not frequently diagnosed
Portugal	—	A Mendes	Lisbon	—	Not diagnosed
Scotland	—	AS Nash	Glasgow	16/31 (52)	Not frequently diagnosed
	—	PGG Darke	Edinburgh	—	Not frequently diagnosed
South Africa	Chantal and Deschamps[20]	PG Howell	Onderstepoort	—	Not uncommon
	Bland van der Berg and Botha[21]	PG Howell	Marion Island	6/17 (35)	—
Sweden	—	M Stavenborn	Uppsala	seropositive cattery	Not frequently diagnosed
Switzerland	Stünzi and Grevel[22]	M Lazarowicz	Basel	seropositive cattery	—
		U Freudiger	Bern	18/79 (23)	10 cases/year
		W Leeman	Zürich	8/26 (31)	12 cases/year
United States	Holzworth[23]	—	Davis, Calif	7/33 (21)[1]	About 1.3% of hospital admissions
	Feldmann and Jortner[24]	—	Pullman, Wash	9/22 (41)[2]	—
	Robison, Holzworth, and Gilmore[25]				
Yugoslavia	—	S Cvetnić	Zagreb	—	Not frequent, diagnosed first in 1972

nales submitted for castration), when blood samples could have been taken conveniently during anesthesia.

The distribution by sex of animals having antibody titers ≥ 30 gives the impression of more seropositive males in most age groups (Fig 1); however, the difference is not significant[d] when a comparison is made between the whole volume of samples from males: females. A comparison of the animals 1 to 4 years of age showed higher percentages of male seroconvertants, and the difference was highly significant ($P < 0.01$) when all males older than 1 year were compared with the seroconverted females. A minimum of antibody carriers was found in animals younger than 1 year ($P < 0.01$, as compared with all older cats); from the 2nd year of life onward, no significant changes occurred. When statistical analysis was applied to purebred cats vs mixed-breed cats and household vs stray cats, no differences in antibody incidence were detected (Table 1).

The clinical signs observed in the random-cat sample (no FIP anamnesis) which correlated with titers exceeding 30 are shown (Table 3).

Geographic Distribution (Random Sample)—Data from published reports and personal communications on the occurrence of clinical FIP in different countries are summarized (Table 2). The numbers of seropositive samples in our tests are also given.

The mean percentages of antibody titer (with the exception of the sam-

[d] The probability of error in comparisons has been calculated, using the X^2 test.

ples from the Marion Islands, South Africa) were 53 ± 16% for titers < 10; 17 ± 12% for titers of 10; and 31 ± 15% for significantly positive titers ≥ 30. However, pronounced differences have been observed in the frequency of antibody carriers between various countries. For Norway and Belgium, a percentage of seropositive samples of ≤ 10% has been found, whereas England, Switzerland, and Germany yielded 20% to 25% (Fig 2); this difference is significant ($P < 0.05$). Ireland, Scotland, Denmark, and France ranked in a group with 40% to 50% of antibody incidence ($P < 0.01$, as compared with the previous group). Ten sera from stray cats in Hungary (shot at the Budapest zoo) were also tested and six had titers ≥ 30 (data not shown).

Because of its unique character, the Marion Island collection has been studied in more detail. In contrast to other countries where seronegative samples (titer < 10) were encountered in frequencies between 27% and 91%, 100% of sera from Marion Island cats had titers between 10 and 30 and were specific for FIP virus, as evidenced by serum-neutralization assay (Table 4).

Noncat Felidae—A limited number of sera from exotic felines and members of other felidae subfamilies were collected from zoological gardens in the Netherlands[e] and tested. Of the samples from the flatheaded cat (*Felis planiceps*), Tayra (*Eira barbata*), margay cat (*Felis (Leopardalus) wiedii*), Amur leopard cat (*Prionailurus bengalensis euptilura*), two servals (*Felis (Leptailurus) serval*), lynx (*Lynx lynx*), leopard (*Panthera pardus*), Sumatran tiger (*Panthera tigris sumatrae*), and sand cat (*F margarita*), none were found to be seropositive. Of 12 samples from South African wild-caught cheetahs (*Acinonyx jubatus*), however, 11 had titers of 10 and one had a titer of 60. Again the specificity was confirmed in three samples by virus neutralization (Table 4).

Catteries, Multicat Households, Animals Hospitals—A total of 86 sera from colonies in Sydney, Copenhagen, Ontario, Liverpool, Onderstepoort (South Africa), Stockholm, Basel, and Toulouse were studied. A distinction was not made whether FIP had been diagnosed previously in the group, but only sera from clinically healthy animals were included in the survey. In Figure 3, the frequency distribution of serum antibody titers in cat collections was compared with that in the random population on one hand and in animals with clinical FIP on the other (37 sera). The three distributions show characteristic patterns; the geometric mean titers were: random population, 11; crowded groups, 50; and animals with clinical FIP, 270.

TABLE 3—Correlation of Signs of Disease and Antibody Titers in Cats Submitted to Animal Hospitals (Random Sample)

Clinical signs	Titers ≥ 30	Total	P
CNS disturbance, ocular changes	8	10	<0.01
Stomatitis, tonsillitis, (tracheo-)bronchitis	8	10	<0.01
Neoplasms, granulomas	5	8	<0.05
Osteomyelitis, periostitis, lameness	4	6	~0.05
No clinical signs	96	405	—

TABLE 4—Results of Heterologous Indirect Immunofluorescence and Neutralization Tests Done with Eight Random Feline Sera from Marion Island (South Africa) and Cheetah (*Acinonyx jubatus*) Sera from South Africa

Serum No.	Immunofluorescence titer	Results of neutralization test*
CATS		
C31	30	0/7
C45	30	0/5
C55	30	0/5
C49	30	1/5
C54	30	3/4
C53	30	5/5†
C84	10	0/5
C58	10	3/9
CHEETAHS		
A2	60	3/7
A1	10	0/10
A6	10	2/6

* Values expressed as No. of animals showing positive fluorescence, indicative of nonneutralized FIP virus/total No. of 1-day-old mice inoculated intracerebrally with the serum/virus mixture. †Assayed twice with identical results.

Discussion

The indirect IFT was introduced in 1976 to measure antibodies against FIP virus,[1] with frozen liver sections from infected kittens serving as an antigen source. Since the antigenic relationship between FIP virus and TGE virus of swine is established,[3,26-28] a heterologous test using TGE virus-infected porcine cells may be used. In this system, the ratio between antigen-containing and negative control cells can be standardized, and extensive absorption of the conjugated anti-feline IgG is not necessary. It may be objected to since a heterologous test is less specific and sensitive than the homologous reaction because coronaviruses show two-way cross-reactivity by IFT within antigenic clusters.[28] This criticism would also apply for FIP virus antigen with respect to feline antibody against a (potentially unknown) related coronavirus. Using such exclusive tests as virus neutralization, a one-way cross-reactivity between anti-FIP viral antibody and TGE virus was found[26,27]; in contrast, mouse brain-adapted FIP virus was not neutralized by porcine anti-TGE viral antibody.[f] The FIP virus neutralizing antibodies in the Marion Island feline sera and the cheetah sera, therefore, are most probably specific and do not reflect previous infection with TGE virus. Domestic cats are susceptible to (inapparent) infection with TGE virus by the oral, nasal, and conjunctival routes[27,f]; antibody to TGE viral antigen, however, could be detected by indirect IFT only after parenteral hyperimmunization.[f] Therefore, the data obtained using the heterologous test reflect the epizootic activity of FIP virus rather than that of TGE virus, which has been of minor importance in some of the regions studied (Ireland,[g] the Southern hemisphere[h]). Heterologous tests tend to be less sensitive than the homologous reaction. For a seroepizootiologic survey, this is acceptable since we wanted to avoid false-positive reactions resulting in overestimates of seroconverted animals. With the same rationale, we have referred to titers < 30 as negative. The reproducibility of the indirect IFT was found satisfactory after double-blind testing of 16 serum samples; in five samples the titers differed by a factor of 3, the remaining results were identical or differed by a factor of 2.

Cats are paired territorial predators; in the wild, the territory is of sufficient area to feed a pair and their offspring. Domestication only slightly modifies the territorial habit; the area

[e] Obtained from the Department of Special Animal Pathology, Veterinary Faculty, Utrecht State University, The Netherlands.

[f] Reynolds DJ, Garwes DJ: Virus isolation and serum antibody responses after infection of cats with transmissible gastroenteritis virus. *Arch Virol* 60:6-66, 197

[g] Kelly WR, Dublin, Ireland: Personal communication, 1978.

[h] Pensaert M, Ghent, Belgium: Personal communication, 1978.

hen coincides with a human habitation and its surroundings. Cattery conditions impose a further reduction in territory which may lead to a colonial habit in queens, young stock, and castrated males. This relatively unnatural situation may result in fighting, subjugation, and the establishment of a peck order.[29] Obviously, the degree of seroconversion within a group depends on the intensity and frequency of contact between the group members; therefore we have differentiated between cats from habitats where mutual contacts are spontaneous (single household cats, free roaming cats, the random sample) and from forcedly crowded communities (catteries, hospitals). It is demonstrated in Figure 3 and has been described previously,[1-3] that high-titered antibodies are more frequently detected in the crowded groups. This is not unexpected for a virus infection—it usually leads to herd immunity; with FIP, however, the shift toward higher titer values is paralleled by an increase of disease incidence. Animals with clinical FIP showed geometric mean titers which were 1.4 logs higher than those in random-sample cats and 0.7 log higher than those in catteries at risk. Differences in the frequency of antibody in the random population were not found between purebred and outbred cats or between household and stray cats. This is consistent with observations on the mortality from FIP.[30] However, we found significantly more seroconverted males than females in the random population cats above 1 year of age. Clinical FIP was also reported more frequently in males than in females,[25] although these results have been questioned.[30] Feline behavior suggests that males, upon reaching sexual maturity, would run a higher risk of infection. Movement of males from one female territory to another (as the females enter estrus) has been confirmed by observations in the colors of offspring.[29] In situations of forced crowding as in catteries, we found no male/female difference in antibody frequency ($n = 43$, $X^2 = 0.98$).

The age distribution of antibody (Fig 1) shows a steady increase of seropositive animals between 0.5 and 2 years, which is also the period of highest incidence of clinical FIP.[30] In Figure 4, we have combined our serologic data with published mortality percentages.[30] It can be seen that in the susceptible period, the ratio of FIP cases:antibody carriers in the random sample was > 0.6 with decreasing values for older animals. The median age for both antibody conversion and death from FIP was about 4 years.

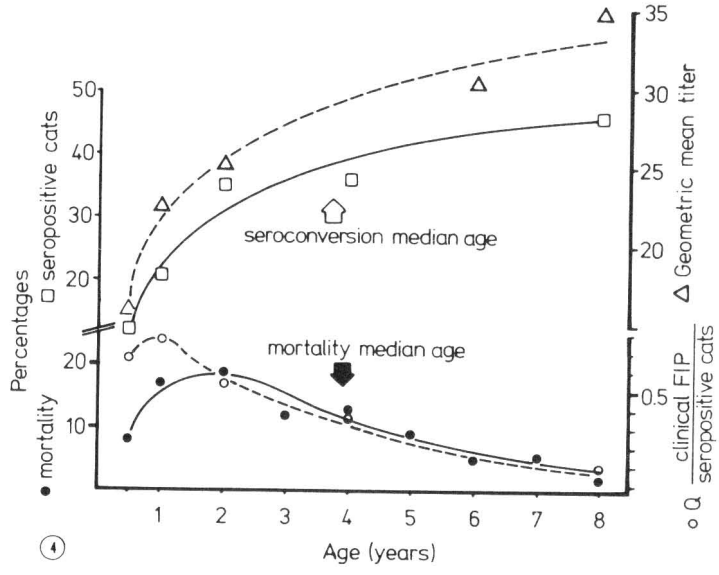

Fig 4—Synopsis of mortality[30] and serology data for FIP.

Of all clinical signs reported, highly significant correlations with antibody presence were determined for CNS disturbance and ocular changes and for respiratory tract signs. While the former have been documented frequently,[30] the respiratory tract distress may represent signs of the primary infection with FIP virus. During our testing program, a healthy colony in Australia was found seropositive; 2 months later, FIP was first diagnosed and a problem of neonatal mortality was observed.[1] The transplacental transmission of FIP virus[31] and neonatal FIP[32,33] have been described.

The worldwide distribution of FIP is summarized in Table 3 and the incidence of antibody for each country is summarized in Figure 2. The significant differences ($P < 0.01$) encountered between neighboring countries (eg, England/Scotland) may reflect divergence in the local population density, and the breeding regimen. In countries with comparable percentages of seroconverted animals (eg, France/Denmark) the ratio of high-titer to low-titer sera can be significantly different. It remains to be shown whether prognostic conclusion on the incidence of clinical FIP can be drawn from these data.

The FIP virus specificity of antibody was established by neutralization tests for the isolated Marion Island population. It must be concluded that FIP virus (or an unknown, but closely related coronavirus) circulates among these cats and that the virus was probably introduced as early as 1948, which was 15 years before the first description of FIP was published.[23] The complete isolation of this group is further illustrated by the fact that feline panleukopenia virus has not been found, although herpesvirus and calicivirus antibodies are present.[a] Neutralizing antibodies against FIP virus were also detected in sera from cheetahs, which have not yet been described as susceptible to FIP.

The results of the present report indicate that clinical FIP correlates in frequency with cat-to-cat contacts on one hand and with the height of antibody titer on the other hand. The cat is an increasingly popular pet and the diagnosis of FIP can be expected to become more important. High antibody titer coinciding with clinical disease is no paradox; the suggested immune pathogenesis of FIP[34] has been supported by our recent finding of marked decreases in the serum com-

[a] Watson ADJ, Sydney, Australia: Personal communication, 1978.

plement concentration of diseased cats.[j] Hypocomplementemia, nonneutralizing antibody, and virus replication in mononuclear phagocytes are found also in the immune pathogenesis of the dengue shock syndrome, which occurs at high frequency in persons with preinfection heterotypic immunity.[k]

[j] Horzinek MC, Daha M, van Dam RH, et al: Arguments in favour of an immune pathogenesis of feline infectious peritonitis. Fourth Munich Symp Microbiol, June 1979

[k] Halstead SB: Cellular and molecular pathogenesis of dengue shock syndrome (abstr), in *Proceedings*, Fourth Int Congr Virol, 1978, p. 71.

References

1. Pedersen NC: Serologic studies of naturally occurring feline infectious peritonitis. *Am J Vet Res* 37:1449–1453, 1976.

2. Loeffler DG, Ott RL, Evermann JF, et al: The incidence of naturally occurring antibodies against feline infectious peritonitis in selected cat populations. *Feline Pract* 8:43–45, 1978.

3. Osterhaus ADME, Horzinek MC, Reynolds DJ: Seroepidemiology of feline infectious peritonitis virus infections using transmissible gastroenteritis virus as antigen. *Zentralbl Veterinaermed [B]* 24:835–841, 1977.

4. Horzinek MC, Osterhaus ADME, Wirahadiredja RMS, et al: Feline infectious peritonitis (FIP) virus. III. Studies on the multiplication of FIP virus in the suckling mouse. *Zentralbl Veterinaermed [B]* 25:806–815, 1978.

5. Jones BR, Hogg GG: Feline infectious peritonitis. *Aust Vet J* 50:398–402, 1974.

6. Watson ADJ, Huxtable CRR, Bennett AM: Feline infectious peritonitis. *Aust Vet J* 50:393–397, 1974.

7. Pastoret PP, Gouffaux M, Henroteaux M: Description et étude expérimentale de la péritonite infectieuse féline. *Ann Med Vet* 118: 479–492, 1974.

8. Stevenson RG, Tilt SE, Purdy JG: Feline infectious peritonitis and pleurisy. *Can Vet J* 12:97–99, 1971.

9. Flagstad A, Larsen S: The occurrence of feline infectious peritonitis. *Nord Vet Med* 28: 577–584, 1976.

10. Mortensen VA, Steensborg K: Infektiøs peritonitis hos katte (F.I.P.). *Dansk Vet Tijdskr (kbh)* 59:761–764, 1976.

11. Ishmael J, Howell JMcC: Observations on the pathology of the spleen of the cat. *J Small Anim Pract* 9:7–13, 1968.

12. Wilkinson GF: Feline infectious peritonitis. *Vet Rec* 86:674, 1970.

13. Auclair-Semere G, Groulade P: Péritonite infectieuze chez une chatte. *Bull Acad Vet (Toulouse)* 48:289–292, 1975.

14. Chappuis G, Brun A, Corbran JP: Péritonite infectieuse féline: Mise en évidence et reproduction expérimentale de la maladie. *Récd Méd Vét* 152:239–242, 1976.

15. Tuch K, Witte KH, Wüller H: Feststellung der felinen infektiösen Peritonitis (FIP) bei Hauskatzen und Leoparden in Deutschland. *Zentralbl Veterinaermed [B]* 21:426–441, 1974.

16. Hartigan PJ, Wilson P: Feline infectious peritonitis. *Ir Vet J* 26:8–9, 1972.

17. Konishi S, Takahashi E, Ogata M, et al: Studies on feline infectious peritonitis (FIP). I. Occurrence and experimental transmission of the disease in Japan. *Jpn J Vet Sci* 33:327–333, 1971.

18. de Aluja AS: Peritonitis infecciosa felina (informe de un caso). *Veterinaria (Mexico City)* 3:106–108, 1972.

19. Mieog WHM, Richter JHM: Feline infectious peritonitis. Tijdschr *Diergeneeskd* 96: 85–98, 1971.

20. Chantal J, Deschamps B: Un cas de péritonite sérofibrineuse chronique chez le chat. *Rev Méd Vét (Paris)* 125:1208–1209, 1974.

21. Bland van der Berg P, Botha WS: Feline infectious peritonitis in South Africa. *J S Afr Vet Assoc* 48:109–116, 1977.

22. Stünzi H, Grevel V: Die ansteckende fibrinöse Peritonitis der Katze. Vorläufige Mitteilung über die ersten spontanen Fälle in der Schweiz. *Schweiz Arch Tierheilkd* 115:579–585, 1973.

23. Holzworth J: Some important disorders of cats. *Cornell Vet* 53:157–160, 1963.

24. Feldmann BM, Jortner BS: Clinicopathological conference. *J Am Vet Med Assoc* 144:1409–1420, 1964.

25. Robison RL, Holzworth J, Gilmore C: Naturally occurring feline infectious peritonitis: signs and clinical diagnosis. *J Am Vet Med Assoc* 158:981–986, 1971.

26. Reynolds DJ, Garwes DJ, Gaskell C: Detection of transmissible gastroenteritis virus neutralizing antibody in cats. *Arch Virol* 55:77–86, 1977.

27. Witte KH, Tuch K, Dubenkropp H, et al: Untersuchungen über die Antigenverwandtschaft der Viren der Felinen Infektiösen Peritonitis (FIP) und der Transmissiblen Gastroenteritis (TGE) des Schweines. *Berl Münch Tieraerztl Wochenschr* 90:396–401, 1977.

28. Pedersen NC, Ward J, Mengeling WL: Antigenic relationship of the feline infectious peritonitis virus to coronaviruses of other species. *Arch Virol* 58:45–53, 1978.

29. Scott PP: The cat, in *The UFAW Handbook on the Care and Management of Laboratory Animals*. Edinburgh 1976, pp 330–356.

30. Pedersen NC: Feline infectious peritonitis: something old, something new. *Feline Pract* 6:42–51, 1976.

31. Pastoret PP, Henroteaux M: Epigenetic transmission of feline infectious peritonitis. *Comp Immun Microbiol Infect Dis (Oxford)*, 1:67–70, 1978.

32. Norsworthy GD: Neonatal feline infectious peritonitis. *Feline Pract* 5:34, 1974.

33. Pastoret PP, Gouffaux M, Henroteaux M, et al: Feline infectious peritonitis. *J Am Vet Med Assoc* 171:740–741, 1977.

34. Horzinek MC, Osterhaus ADME: The virology and pathogenesis of feline infectious peritonitis. *Arch Virol* 58:253–266, 1978.