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Population Dynamics of Feline Immunodeficiency Virus within Cat Populations

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A deterministic model was constructed for studying the circulation of Feline Immunodeficiency Virus (FIV), a feline retrovirus homologous to Human Immunodeficiency Virus (HIV), within populations of domestic cats. The model has been tested with data generated by a long-term study of several natural cat populations. Simulations and a study of stability show that once introduced, the retrovirus is maintained within the population, with a stable equilibrium stage reached by both numbers of susceptible and infected individuals. An estimation of parameters indicates that the transmission rate is low and depends of the structure of the population. In addition, FIV has a low impact on the population in that the total number of cats at equilibrium when this virus is present is almost always equal to the habitat carrying capacity in the absence of the virus. Those results, in agreement with other observations, suggest that FIV originally arose in the distant past.

1. Introduction

Despite the large number of existing models describing the influence of microparasites on host populations, to our knowledge no model of the circulation of retroviruses within populations of mammals (except the special case of humans) is available. Nevertheless, the retroviruses are very interesting because of their particular circulation pattern. Indeed, the very long seropositivity period, as well as the succession of different clinical stages, induce the need for specific models. Moreover, the functioning of host populations may be very specific, hence their modelling requires a good understanding of the patterns of the hosts' spatial and social structures. We present here a simple model of the circulation of the Feline Immunodeficiency Virus (FIV), a feline retrovirus, within a population of domestic cats (*Felis catus*). Because they live in a variety of ecological conditions, domestic cat populations show a high degree of variability in their spatial and social structures which can be compared to other populations of FIV-infected non-domestic felids that are more difficult to study in natural habitats. The wide prevalence of FIV infection in natural populations of cats provides an opportunity to analyse the consequences of population structures on the circulation of the virus. Although the nature of this model is qualitative, the values of parameters such as transmission rate and carrying capacity may give information on the impact (in terms of reduction of number of cats due to the disease) of the lentivirus within cat populations, and on the influence of cat population functioning on the spread of the virus.

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2. Materials and Methods

2.1. HOST POPULATION

The domestic cat is present in large numbers on all continents and on numerous islands (Todd, 1977; Legay, 1986). The spatial organization of female cats, like other carnivorous species, is determined by both the abundance and dispersion of feeding resources (Macdonald, 1983; Liberg & Sandell, 1988). The spatial distribution of female cats is principally determined by the spatial organization of human habitats in rural areas, as females are associated with human shelters (for kitten rearing, Liberg, 1981; Pontier, 1984). The spatial distribution of males, at least during the reproductive season, seems to be governed instead by the distribution of receptive females (Liberg, 1980, 1981; Liberg & Sandell, 1988). Stray cats in urban areas are organized in large social groups around shelter and feeding resources (Dards, 1979; Calhoon & Haspel, 1989). Density is high in these groups (up to 2000 cats per km^2, Natoli & De Vito, 1988, 1991) in contrast with rural areas where the density is much lower (from less than 1 cat per km^2 to 30 cats per km^2, Corbett, 1979 in MacDonald *et al*., 1987; Liberg & Sandell 1988). Also, different mating systems have been observed in relation to the spatial and social organization of populations: from promiscuity in high-density urban areas (Natoli & De Vito, 1988), to polygyny with monopolization of females in low-density rural areas (Macdonald *et al*., 1987; Liberg & Sandell, 1988). Thus the pattern of human habitation is the main factor which determines the social and reproductive patterns of the domestic cat populations.

We monitored each year three rural cat populations in France: Barisey-la-Côte (from 1990), Aimargues (from 1982), Saint-Just Chaleyssin (from 1982), and one urban stray cat population in Lyon (from 1991). These cat populations are characterized by different densities, survival parameters, and mating systems (Table 1, data in Pontier, 1993). We observed a promiscuous mating system in the urban stray cats, while a polygynous mating system was observed in the three rural cat populations (Pontier, 1993). From these studies, fecundity, averaged over females of all ages, has been estimated in rural areas of 4.8 offsprings per year (i.e. 2.4 per cat), and the mortality rate, per cat, at 0.6 year. In urban areas, preliminary observations led us to a value of 1.85 for fecundity and between 0.6 and 0.8 for mortality (unpublished data). An epidemiological study has been carried out each year since 1991 in the four cat populations on a representative sample of cats. Partial results of the epidemiological survey are given Table 1.

2.2. MICROPARASITE

FIV is a recently identified virus inducing AIDS in cats (Pedersen *et al*., 1987; Pedersen *et al*., 1993). It belongs to the lentiviruses subfamily of retroviruses (Olmsted *et al*., 1989), which also includes Human and Simian Immunodeficiency Viruses (HIV and SIV). However, the transmission mode of FIV seems to be through by bites inflicted during fights (Yamamoto *et al*., 1989) instead of sexual contact, in contrast to HIV. Indeed, the major parameters influencing FIV infection seem to be behaviour (roaming or not), sex and age (Lutz, 1989; Sparger *et al*., 1993). In addition to providing a relevant model for HIV studies, FIV is of particular interest in veterinary science, as infection with FIV gives rise to a wide range of clinical signs (Brown *et al*., 1991; O'Neil *et al*., 1991), with a clinical staging very similar to human AIDS (Ishida & Tomoda, 1990). An attempt has been made to delineate the clinical course of FIV infection in a series of five stages analogous to those of HIV infection in humans (Pedersen & Barlough, 1991; Sparger, 1993). First, the infected cat suffers from an acute stage occurring several weeks after infection and lasting 4–16 weeks. Despite recovering from the primary stage of illness, virtually all cats infected with FIV become lifelong carriers of the virus. The first stage is followed by an asymptomatic carrier phase lasting months to years, during which the behaviour of the cat does not seem to be affected. There follows persistent generalized lymphadenopathy (PGL), AIDS-related complex (ARC) and AIDS, characterized by miscellaneous disorders and opportunistic infections. Because of the very long period separating infection from death due to AIDS, and the low survival rate of roaming

TABLE 1
Main characteristics of the four studied populations

Population	Habitat	FIV rate (y)	N (cats)	Sampling (cats)	Mating system	Density (cats · km^{-2})
Barisey-La-Côte (BC)	rural	9.1	60	33	Polygynous	200
Aimargues (AI)	rural	9.5	203	42	Polygynous	120
St-Just-Chaleyssin (SJ)	rural	24.4	299	45	Polygynous	250
Lyon (LY)	urban	33.33	40	18	Promiscuous	1100

cats (Legay & Pontier, 1983; Pontier, 1993), the majority of infected cats would be expected to die from "natural causes" (road accidents, hunting, poisoning, Hamilton *et al*., 1969) before reaching the terminal stages of FIV infection. They should have time to transmit the virus, but not to die from it— hence we expect the virus to have a low impact within natural populations of domestic cats. The impact of FIV is one aspect of the virus which we attempt to study with the model, the other being the viral transmission rate.

2.3. THE MODEL

The model we present here is based on the work of Anderson and May (e.g. Anderson & May, 1991). The dynamics of a cat population were represented by a set of differential equations. Let N be the total number of cats at time t, and K be the carrying capacity of the habitat at equilibrium. With regard to the demographic parameters, we assumed that the density dependence acts primarily on mortality. Hence the birth rate is constant (Pontier, 1984; 1993). The death rate is linearly related to N, and has the form $(m+rN/K)$, where m is the natural death rate and $r=b-m\geqslant 0$ is the population growth rate in the absence of resource limits. When the population is free from FIV, the dynamics of the population is given by the familiar logistic equation (Verlhust 1838):

$$\frac{\mathrm{d}N}{\mathrm{d}t}=rN\left(1-\frac{N}{K}\right). \tag{1}$$

Next, in order to describe the spread of FIV through the population, we introduce susceptible cats denoted by $X(t)=X$, and infected ones denoted by $Y(t)=Y$, so that $X+Y=N$.

For the sake of simplicity, we assume here that there is only one stage of illness combining acute, seropositivity, PGL, ARC and AIDS stages. Indeed, we can assume that infected sick individuals no longer participate in social life as they are too weak in the last stages to compete for territories or mates. We can also assume that the time they remain at these three last stages is too short, and their roaming habits become too restricted, to die from "natural causes". Thus, as they do not contribute to the reproductive effort of the population, or the transmission of FIV, the infected/sick class can be ignored.

We call ρ the encounters rate within the population, β the frequency of an aggressive contacts resulting in a bite (when an encounter occurs), and c the efficiency of the FIV transmission by biting. It is feasible to estimate

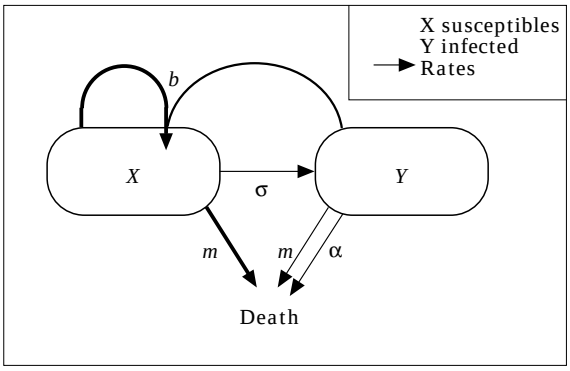

FIG. 1. Flowchart of the FIV/cat model, with susceptible individuals (X) and infected individuals (Y). There is no possibility of recovery, and no vertical transmission. b is the birth rate, m the intrinsic death rate, σ the effective bite rate, α the mortality rate due to AIDS.

c through experimental bites, but it is not feasible to estimate either the encounters rate or the probability of being bitten, when an encounter occurs. This would require intense behavioural observations, as the rate would depend heavily on the respective hierarchical positions of the two protagonists. Moreover, the results would not be generalizable to populations other than the one studied. Thus, as it is not yet possible to estimate separately ρ, β and c, and to reduce the complexity of the model, we decided to consider $\rho\beta c$ as a composite parameter, σ, which may be defined as the rate of effective bites (for transmission of FIV). The death rate due to AIDS is α ($1/\alpha$ is the length of the infectious period), and is independent of population density.

For the following biological reasons, the transmission rate will not be characteristic of mass action models but rather of proportionate mixing models (Busenberg & Cooke, 1993). We assume that an individual's infection status does not affect the probability of having direct contacts with others, and that the rate at which an individual comes into contact with others in the population is a constant, ρ. Then, of all the contacts had by a single susceptible individual, a proportion equal to $Y/(X+Y)$ is with infected individuals. Thus the rate at which contacts between susceptible and infected individuals occur is equal to $\rho XY/(X+Y)$. Then the force of horizontal transmission is $c\rho\beta XY/(X+Y)$, that is, $\sigma XY/N$. Next we assume that there is no vertical transmission (Ueland & Nesse, 1992), and there is no recovery of infected cats, by either natural or artificial means.

The compartmental representation is shown in Fig. 1. A set of first-order differential equations describing the dynamics of cat populations infected

with FIV is thus given by the proportionate mixing model:

$$\frac{dX}{dt} = b(X+Y) - mX - \frac{rNX}{K} - \frac{\sigma XY}{N} \quad (2)$$

$$\frac{dY}{dt} = \frac{\sigma XY}{N} - mY - \frac{rNY}{K} - \alpha Y. \quad (3)$$

The equation for the total population is obtained by summing eqns (2) and (3):

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) - \alpha Y. \quad (4)$$

Simulations were carried out with the computer program Dynamac (Rousseau, 1988, see Fig. 2).

3. Results

3.1. STABILITY ANALYSIS

When the effective contact rate σ is smaller or equal to the death rate α induced by FIV, the ingoing flux of the infected class is lower than the outgoing flux, and the number of infected cats decreases: this is easily seen from eqn (3). Hence

$$0 < \sigma \leqslant \alpha \text{ implies } Y(t) \to 0 \text{ as } t \to +\infty.$$

Thus, from now on, we assume $\sigma > \alpha$. This is a biologically realistic hypothesis, as the literature (Pedersen *et al*., 1993) shows the length of FIV infection to be between 2 and 8 years ($0.5 > \alpha > 0.125$), and, as we will see, σ is always larger).

Setting the time derivatives to zero in eqns (2) and (3), we deduce that the following equilibrium solutions are possible:

$$\begin{cases} X_1^* = 0 \quad \text{and} \quad Y_1^* = 0 \\ X_2^* = K \quad \text{and} \quad Y_2^* = 0 \\ X_3^* = \dfrac{bK(\alpha^2 - \alpha\sigma + b\sigma + \alpha m - \sigma m)}{(\sigma - \alpha)^2(b - m)} \end{cases}$$

and

$$Y_3^* = \frac{K(\sigma - \alpha - b)(\alpha^2 - \alpha\sigma + b\sigma + \alpha m - \sigma m)}{(\sigma - \alpha)^2(b - m)},$$

provided the latter is positive. The first two equilibrium points correspond to trivial and uninteresting solutions: disappearance of either the disease or the population. The third one is non-trivial and interesting, and has also been obtained directly from computer program analyses (Mathematica Wolfram Research, Inc.). A detailed stability analysis, which we summarize here, is found in the Appendix.

To study the conditions of stability of those equilibria, we introduce two threshold parameters, both of them having a biological significance. According to the value of these parameters, one or the other equilibrium states will operate at a given time. According to Jacquez *et al*. (1991) in a population at the equilibrium stage, the reproductive number, R_0,

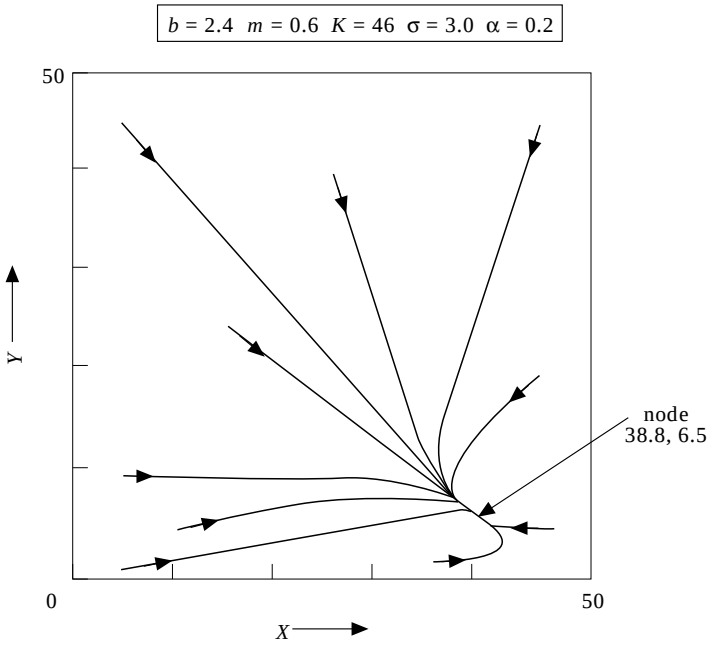

FIG. 2. Phase portrait for different initial conditions of susceptible and infected individual numbers (X and Y individuals). Dynamics of FIV reach in any case an equilibrium point, or node, illustrating the stability of the model.

is "the number of infected individuals generated by one infective when all contacts of the infective are susceptibles" (Jacquez *et al*., 1991). The disease will spread if and only if this number is greater than one. R_0 is defined as the average number of effective infectious contacts per cat per unit time multiplied by the mean duration of life of an infected cat when the population is at equilibrium stage. Hence, from eqns (2) and (3), we obtain:

$$R_0 = \frac{\sigma}{\alpha + b}. \tag{5}$$

In fact, at equilibrium $N = K$; thus $\alpha + r(N/K) + m = \alpha + b$ is the death rate induced by both disease and demography, $1/(\alpha + b)$ being the life expectancy of an infected cat. When $R_0 \leqslant 1$, one has $Y(t) \to 0$ and $X(t) \to K$ over time: the epidemic disappears and the population will settle at its disease-free equilibrium number K (Anderson *et al*., 1981; Bentil & Murray, 1993).

When $R_0 > 1$, then the prevalence $y(t) = Y(t)/N(t)$ of infected individuals stabilizes to

$$y^* = 1 - \frac{b}{\sigma - \alpha}. \tag{6}$$

A second threshold parameters arises (see Appendix). Set

$$R_1 = \frac{b}{m + \alpha y^*}. \tag{7}$$

This is the "net reproductive coefficient of the population when the disease is endemic" (Busenberg & Cooke, 1993). Still assuming $R_0 > 1$, when $R_1 \leqslant 1$ the population becomes extinct ($N(t) \to 0$), while when $R_1 > 1$, the disease remains endemic and approaches a unique equilibrium ($X(t) \to X_3^*$ and $Y(t) \to Y_3^*$).

3.2. PARAMETER ESTIMATIONS

Equilibria have been observed *in natura*, from a long-term study of three domestic cat populations in rural areas (12 years for Saint-Just-Chaleyssin (SJ) and Aimargues (AI), and three years for Barisey-La-Côte (BC), Pontier, 1984; 1993). Thus, we assume that $dN/dt = 0$. As the serological state of individuals is only followed for two years, there is not indication whether we also have $dY/dt = 0$. Nevertheless, as we know that an equilibrium state is dictated, only the three equilibrium points are possible solutions. Serological data indicate that $Y \neq 0$, hence we deduce that the only possible equilibrium state is when the disease is present within the population: $X = X_3^*$ and $Y = Y_3^*$. Thus, we have $dX/dt = dY/dt = 0$.

At equilibrium, $dN/dt = 0$, hence, from eqn (4): $rN(1 - N/K) - \alpha Y = 0$.

Thus, $K = rN^2/(rN - \alpha Y)$. And from eqn (3), we have $\sigma = N(\alpha + m + (rN/K))/\mathrm{X}$.

We thus have five parameters to consider (b, m, K, σ and α), two being deduced from biological data (b and m). Indeed, as mentioned above, the values of b and m are 2.4 and 0.6, respectively, for rural populations, and 1.85 and 0.7 for the urban one. From the three remaining parameters, α is the "less unknown". The total length of infection ($1/\alpha$) is still not known with precision, and may be highly variable: however, it is thought to be on average between 3 and 6 years (Moraillon, 1990; Pedersen & Barlough, 1991; Sparger, 1993). Thus, we can express K and σ as functions of α, with values of α falling within a narrow range. Figure 3 shows values of σ and K for the four studied populations and for values of $1/\alpha$ varying more than expected in reality (from 2 to 10 years). The variation of K and σ with values of α is not large, showing that α is not a major parameter of the model, after it reaches 1 year. The figure also shows the variation of R_0 and R_1 for different values of α: R_0 and R_1 are always greater than 1, leading to the third equilibrium point: (X_3^*, Y_3^*). The transmission rate, (λ), may be defined as the rate of effective bites received by a cat per unit of time (σ), multiplied to the proportion of infected cats in the population ($\lambda = \sigma y^*$). Thus $1/\lambda$ is the average time between two infections. Figure 3 also shows that the variation of values of $1/\lambda$, according to the variation of α, is not important and that there is a great difference in values of transmission rates for the four populations, from an average time between infections of around 1 year, to one of around 4 years (LY and SJ on the one hand, and AC and BC on the other). We can also see that values of N are always less than but very close to K.

4. Discussion

The main results of the model are given below.

(i) When FIV is introduced in a population of cats, infection inevitably develops and is maintained ($R_0 > 1$ and $R_1 > 1$). We have no means of verifying this experimentally (i.e. by introducing viruses within a natural population). However, FIV is encountered world-wide even on some islands, and at relatively high rates (11.04% on average, on 59 FIV serosurveys (for a review, see Courchamp & Pontier, 1994). Moreover, we have serological proof of its presence in different parts of the world as far back as stored sera are available, which is 1974, 1972 or 1968, in Europe, Australia, Japan and the United States, respectively (Gruffydd-Jones *et al*., 1988; Sabine *et al*., 1988; Furuya *et al*., 1990; Moraillon *et al*., 1990; Shelton *et al*., 1990).

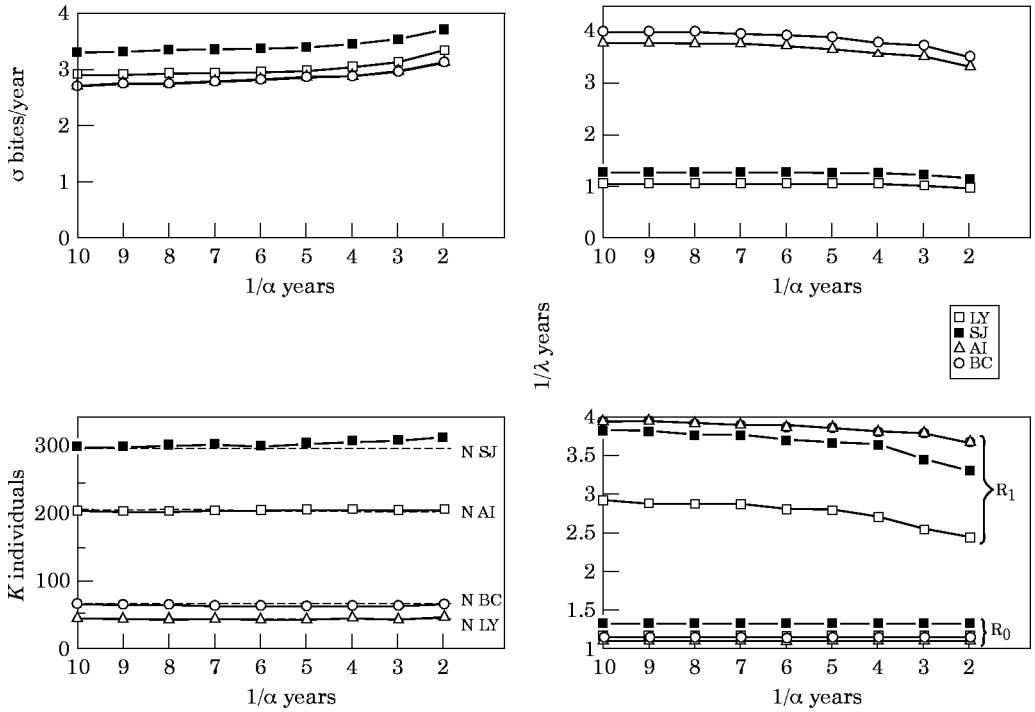

FIG. 3. Influence of α on different parameters of the model for the four studied populations (Lyon (LY), Saint-Just-Chaleyssin (SJ), Barisey-La-Côte (BC) and Aimargues-Le-Cailar (AC)). $1/\alpha$ is the infectious period length, σ the effective bite rate, $1/\lambda$ the average time between two infections, K the carrying capacity of the habitat in the absence of virus, and R_0 the reproductive number. These parameters do not seem to be affected by variations in the death rate due to AIDS.

(ii) The introduction of FIV does not lead to the extinction of susceptible or infected cats, but to a stable equilibrium stage of both categories, in proportions dependent upon parameters specific to the population ($dX/dt = dX/dt = 0$). The stable equilibrium stage of the total population dynamics is observed *in natura* in several natural populations of cats, over more than 12 years (Pontier, 1993).

(iii) The impact of the virus, in terms of reduction of the population size at equilibrium, is not significant ($K \approx N$). We have no means of verifying this result, but it seems at least intuitively sensible, if we consider the life expectancy and infection duration ratio.

(iv) The transmission rate is always low ($1/\lambda > 1$ year), confirming field observations that FIV is not very contagious (Shelton *et al*., 1990). With the long duration of infection, this again is consistent with a low-impact virus.

In addition to the general properties of FIV dynamics within domestic cat populations, we also have determined that the transmission rate (λ) differs clearly according to the population. Thus the dynamics of FIV circulation within cat populations appears to be influenced by the characteristics of the host population. This result is not surprising as the spatial distribution of the cats in the local population, and more generally the population's biological properties (survival pattern, social structures, mating system and dispersal pattern) largely differ according to the fragmentation of human habitat (Pontier, 1993). This may in turn largely influence the pattern of diffusion of cat diseases. The highest transmission rates of FIV are obtained both in a rural cat population (Saint-Just Chaleyssin) and in the urban cat population (Lyon). The former is characterized by a low cat density and a high degree of polygyny, and the latter by the highest density and a promiscuous mating system. The lowest rates of transmission are observed in the rural cat populations characterized by intermediate densities, and maybe by a lower degree of polygyny. Even if we cannot rule out the possibility that differences in FIV prevalence rates between cat populations are partially linked to geographical position of these populations (LY and SJ populations are geographically close to each other), these differences should result mainly from the differences in behavioural

mechanisms of the individuals. Thus it is crucial to investigate the behavioural mechanisms involved in the transmission of FIV.

In conclusion, it should be noted that, by design, the purpose of this model is more the conceptualization of the mechanisms of FIV transmission than predictions or precise parameter estimations. Nevertheless, the four points highlighted by the model, as well as the obtained values for the estimated parameters, are in agreement with field observations and the literature. It is also interesting to note that these points support the hypothesis that the virus originally arose in the distant past. Considering both the world-wide repartition of FIV strains, and that many non-domestic felid species are infected by specific but very similar strains of FIV (Courchamp & Pontier, 1994), it can be hypothesized (Olmsted *et al*., 1992) that FIV might have been present even before the speciation of felids, giving an interesting example of host–parasite coevolution.

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APPENDIX

Mathematical Analysis

When $\sigma \leqslant \alpha$, one gets $\sigma XY/N \leqslant \alpha Y$ because $X \geqslant 0$, $Y \geqslant 0$ and $X+Y=N$. Hence,

$$\frac{dY}{dt} \leqslant -\left[m+\frac{(b-m)}{K}\right)N\Bigg]Y(t)<0 \quad \text{if } Y(t)>0,$$

so that $Y(t) \to 0$ as $t \to +\infty$ when $\sigma \leqslant \alpha$.

Assuming $\sigma > \alpha$ to hold, let us introduce the prevalence $y(t)$ and $x(t)=1-y(t)$:

$$y(t)=\frac{Y(t)}{N(t)}, \quad x(t)=\frac{X(t)}{N(t)}.$$

One finds after some calculations two logistic equations:

$$\frac{dy}{dt}=[(\sigma-\alpha-b)-(\sigma-\alpha)y]y$$

$$\frac{dx}{dt}=[b-(\sigma-\alpha)x](1-x).$$

For the dynamics of the prevalence, one has two equilibria:

$$y=0 \quad \text{and} \quad y^*=1-\frac{b}{(\sigma-\alpha)}.$$

Now, $0 \leqslant y(t) \leqslant 1$, so that the latter is pertinent if and only if $b/(\sigma-\alpha)$ lies in [0, 1]; from $\sigma > \alpha$ this is possible if and only if $R_0 \geqslant 1$. As a first conclusion, it follows that

(i) if $R_0 \leqslant 1$ then $y(t) \to 0$ and $x(t) \to 1$ as $t \to +\infty$,

(ii) if $R_0 \geqslant 1$ then $y(t) \to y^*$ and $0 < y^* < 1$ as $t \to +\infty$.

Let us now look at the dynamics of the total population N, given by (4).

When $R_0 \leqslant 1$ the equation for N is asymptotically equivalent to (1); thus $N(t) \to K$, $Y(t)=y(t)N(t) \to 0$ and $X(t) \to K$ as $t \to +\infty$.

If $R_0 > 1$ the equation for N is asymptotically equivalent to

$$\frac{dN}{dt}=\left[(b-m-\alpha y^*)-\frac{(b-m)}{K}N\right]N.$$

Reintroducing the threshold parameter R_1, one gets

(i) if $R_1 \leqslant 1$ then $N(t) \to 0$ and the population gets extinct: $X(t) \to 0$ and $Y(t) \to 0$ as $t \to +\infty$,

(ii) if $R_1 > 1$ then $N(t) \to K(b-m-\alpha y^*)/(b-m)$ and an endemic state emerges: $X(t) \to X^*$ and $Y(t) \to Y^*$ as $t \to +\infty$,

with

$$X^*=\frac{bK(\alpha^2-\alpha\sigma+b\sigma+\alpha m-\sigma m)}{(\sigma-\alpha)^2(b-m)}$$

and

$$Y^*=\frac{K(\sigma-\alpha-b)(\alpha^2-\alpha\sigma+b\sigma+\alpha m-\sigma m)}{(\sigma-\alpha)^2(b-m)}.$$

These results may be summarized in the following table:

		$y(t)$	$N(t)$	$X(t)$	$Y(t)$
$R_0 \leqslant 1$		0	K	K	0
$R_0 > 1$	$R_1 \leqslant 1$	y^*	0	0	0
$R_0 > 1$	$R_1 > 1$	y^*	N^*	X^*	Y^*