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Modelling the Feline Leukemia Virus (FeLV) in Natural Populations of Cats (*Felis catus*)

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A compartmental model was built in order to study the circulation and impact of Feline Leukemia Virus (FeLV) in populations of domestic cats. The model was tested with data from a long-term study of several feline populations. The study of stability shows that FeLV is maintained in the population with a stable equilibrium and a slight reduction of population size. Estimation of the transmission rate allows us to make a comparison with the values previously estimated in the literature. We compare the impact of mass vaccination or removal programmes in controlling FeLV infection, and conclude that vaccination is more efficient. © 1997 Academic Press

INTRODUCTION

During recent years, knowledge and prediction of the epidemiology of parasitic diseases have been greatly improved by modelling techniques. Special interest is devoted to the Retroviridae family, due to the importance of Human Immunodeficiency Virus (HIV) infection. Numerous models have been proposed for this infection (Bailey, 1994), whereas the circulation of animal retroviruses has not been modelled until recently (Courchamp *et al.*, 1995).

Our interest is focused here on Feline Leukemia Virus (FeLV), a feline retrovirus (Jarrett *et al.*, 1973), and its impact in natural populations of domestic cats (*Felis catus*). For the study we built a deterministic model of FeLV circulation. The first aim of this model is to analyse the importance of transmission parameters, which were previously estimated from experimental studies, on disease persistence and stability. We also evaluate the impact of infection on cat population growth.

Our second goal concerns the practical problem of eradication of infections. Mass vaccination or removal (culling) of infected individuals are the two main ways that are usually proposed to control parasitic or infectious diseases. For most diseases, both methods can be used, but the question remains which is better for economic and efficiency reasons. A classical example is fox rabies control in Europe, where a controversy remains over theoretical results (Anderson *et al.*, 1981; Smith and Harris, 1991; Aubert, 1995).

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In the case of FeLV infection, vaccination of susceptible cats and removal of infected ones are possible and practised. Removal was done as soon as the first detection tests were available (Hardy *et al*., 1976b). A large removal programme was applied in catteries in the Netherlands, which reduced the prevalence of cats positive for FeLV from 9% in 1974 to 3% in 1985 (Weijer *et al*., 1986). The first FeLV vaccine was commercialised in 1985 (Pedersen *et al*., 1985). All commercial vaccines were proved to protect cats from subsequent infection (Panel Report on the Colloquium on Feline Leukemia Virus/Feline Immunodeficiency Virus: tests and vaccination, 1991), but they require maintaining the vaccination indefinitely. No longitudinal survey has yet compared the prevalence of FeLV infection before and after a vaccination programme, thus the efficiency of vaccination is not known at the population level. We investigate the question by studying the stability of models including either vaccination or removal measures. Then we discuss the interest of both measures taking into account their cost and benefit and comparing control of FeLV and other diseases.

MATERIAL AND METHODS

Host Populations

The spatial and social organization of populations of domestic cats, depends on their habitat (Pontier *et al*., 1995). In rural as in urban areas, growth of cat populations is limited by environmental resources, such as food and shelters from human origin (Calhoon and Haspell, 1989).

TABLE I

Main Characteristics of the Studied Populations

Population	Habitat	Density (cats/km2)	Population size (cats)
St-Just-Chaleyssin (STJ)	Rural	250	299
Aimargues (AIM)	Rural	120	203
Barisey la-Côte (BLC)	Rural	200	60
Lyon Croix-Rousse (LCR)	Urban	1100	40

Note. The sample size is given considering all four samplings per population. The sample size may be higher than the population size, as the sample size includes all cats sampled during the four years of study. FeLV antigen-positive cats were detected with the ELISA test. Data from Pontier and Artois unpublished.

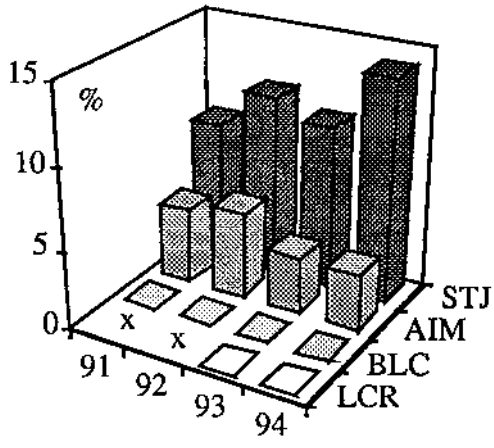

FIG. 1. Annual prevalence of FeLV antigenemia, assessed by ELISA test, in the four studied populations (STJ = Saint-Just Chaleyssin, AIM = Aimargues, BLC = Barisey-la-Côte, LCR = Lyon Croix-Rousse), for the 4 years of study (1991 to 1994). x = not determined.

Three rural cat populations have been monitored since 1982 in Aimargues and Saint-Just Chaleyssin and since 1990 in Barisey-la-côte. One urban cat population, Lyon Croix-Rousse, has been monitored since 1992 (Pontier, unpublished). The main characteristics of the populations are given in Table I. Annual birth and death rates have been estimated from the Saint-Just Chaleyssin population (Legay and Pontier, 1985; Pontier, 1993). As the population size, sex ratio, and age structure remain stable, mean fecundity and mortality rates were averaged over individuals of all ages and were estimated at 2.4 and 0.6 per year, respectively (Pontier, unpublished).

In 1991 we undertook an epidemiological study of the four cat populations. Serological status of a sample of cats is determined yearly to assay occurrence of FeLV p27 antigen. results are summarized in Fig. 1. Few cats from these populations are followed by veterinarians (less than 5%, Pontier and Artois, unpublished), and almost none have been vaccinated or tested for FeLV; thus we assume that there is no direct human intervention on virus circulation.

Virus

The Feline Leukemia Virus (FeLV) is an oncogenic and immunosuppressive virus of the domestic cat (for a review see Hardy, 1993). FeLV belongs to the Retroviridae family and the Oncovirinae subfamily, like Human T-Lymphotropic Viruses (Jarrett *et al*., 1973, Wong-Staal and Gallo, 1985). Its transmission mode may be epigenetic, from mother to foetus during

pregnancy (Hoover *et al*., 1983), or horizontal, mainly by saliva (by biting, grooming, or sharing the same feeding plates; Francis *et al*., 1977; Hoover *et al*., 1977; Rojko and Olsen, 1984). The force of infection f.i. (Capasso, 1993) depends on the transmission rate, that is, on the number of effective contacts per unit of time when one infectious host is present (here called σ). In particular, in our model, f.i. $= \sigma Y/N$, where Y/N is the proportion of infected individuals (see below: modelling of disease transmission). A possible way to calculate σ is to measure the time between the first contact with a contagious individual and the onset of infection, that is, the inverse of f.i. In the case of FeLV, the development of infection (assessed by serological tests) was studied among susceptible cats living in catteries, where around one-third of the cats were excreting carriers. The mean duration before infection was between 3.4 and 13 months, according to the age of the cats (Petersen *et al*., 1977; Grant *et al*., 1980), which means a transmission rate between 3.08 and 11.76 per year.

The clinical course of FeLV infection begins with a phase of transient infection lasting on average three weeks and up to four months (Hardy *et al*., 1973; Rojko *et al*., 1979). Although the virus is present, this stage is generally asymptomatic, and transiently infected cats are considered a minor source of infection, as their viremia is low (Jarrett *et al*., 1982) and the virus excretion only lasts a few days (Francis *et al*., 1979; Jarrett *et al*., 1982). This stage may lead to two possible outcomes.

First, a large proportion of the infected cats (65%, Hoover and Mullins, 1991) stops viral replication and becomes immune to subsequent infection (Rojko *et al*., 1979). These cats may be considered as clinically recovered and seem to stay immunised for life, although this question is still discussed (Hardy *et al*., 1976a; Pacitti and Jarrett, 1985; Charreyre and Pedersen, 1991). Immune cats are no longer contagious and have a normal life expectancy (McClelland *et al*., 1980; Francis and Essex, 1980; Rojko *et al*., 1982). Immunised females give birth to kittens that are passively protected by maternal antibodies during a few weeks, and then become susceptible (Hoover *et al*., 1983).

Thirty to 40% of the infected cats fail to develop the appropriate immune response and become persistently viremic (Hardy *et al*., 1976a). The viremia remains lifelong and causes various proliferative or immunosuppressive disorders which lead to death within a few weeks to several years. Francis and Essex (1980) estimated the mean duration between infection and death of viremic cats at 20.9 months, which means a mortality rate of 0.57 per year. Data from three other authors give annual mortality rates of 0.51, 0.53, and 0.51 (Weijer and Daams, 1976; Hardy, 1980; Ishida *et al*., 1981), calculated on 20, 36, and 14 months respectively, with a constant mortality rate. Moreover, the fertility of viremic queens is strongly reduced: 80% of their pregnancies lead to abortion, and the few live kittens are viremic at birth and die early (Hoover *et al*., 1983).

Our model takes into account population parameters (birth and death rates, evaluated at 2.4 and 0.6 per year in the Saint-Just Chaleyssin population) and parameters characterizing infection dynamics. the transmission rate (we use here the value of 2 per year), the proportion of infected cats that become persistently viremic (0.33), and the specific mortality rate of persistently viremic cats (0.53 per year, which is the value estimated with the largest sample size) are included in the model.

Model of FeLV Circulation

The model developed and analysed here is based on Anderson and May's work (e.g., Anderson and May, 1991). The total number of cats at time t is denoted N. Let b be the natural birth rate and let m be the natural death rate in the absence of infection, so that $r = b - m$ is the intrinsic growth-rate of the cat population in the absence of resource limitation and epidemic. According to what is known from the populations we monitored (Pontier, 1993), we assumed that resource limitation acts through density-dependence on the death rate which takes from $(m + r(N/K))$, where K is the carrying capacity of the habitat, while the birth rate remains constant at b.

When the population is free from FeLV infection, the dynamics of the total population is governed by the logistic model (Verhulst, 1838):

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right). \qquad (1)$$

When the virus is present, we assumed that cats may be divided into three categories, according to the clinical stage: susceptible (denoted X), persistently viremic (Y), and immune (Z) cats. For simplicity, we do not considered transient viremic cats as a category, as we assumed that this stage (lasting several weeks) is short enough to be neglected, compared to the duration of life or infection (several years). The compartmental representation is presented Fig. 2.

σ is annual transmission rate of the infection. As for other retroviruses, we considered that infectious contacts, depending on the social behaviour of individuals, do not increase with population size (Capasso, 1993; Courchamp *et al*., 1995). The force of infection was

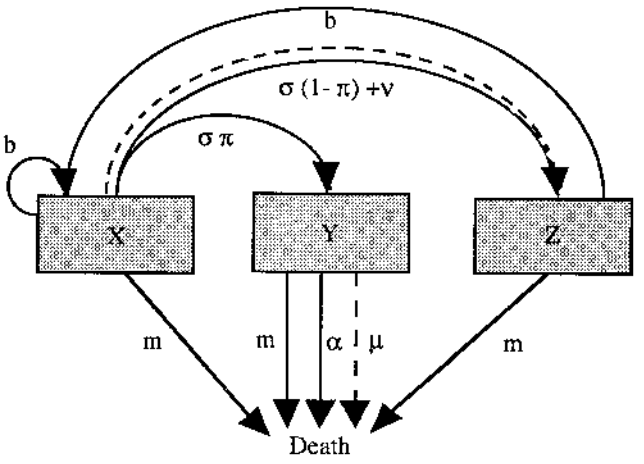

FIG. 2. Susceptible individuals are denoted X, persistently viremic ones Y, and immune cats Z. Transient infected cats are not considered as a category, and only susceptible and immune cats take part in reproduction. Viremia or immunity stages are not reversible and immune cats have the same life expectancy as susceptible ones. b is the birth rate, m is the intrinsic death rate, σ is the transmission rate, π is the proportion of infected cats that become persistently viremic, and α is the mortality rate due to FeLV infection. Dotted lines show prophylaxis measures, with v = vaccination rate of susceptible cats and μ = removal rate of infected cats.

formulated according to the proportionate mixing model, which considers that the incidence of infection is a function of the proportion, rather than the number, of infected individuals (Hethcote and Yorke, 1984; Capasso, 1993; Hethcote and Van Ark, 1987).

When cats are infected, they become either persistently viremic (in proportion π), or immune $(1-\pi)$. Immune and susceptible cats have a normal life expectancy and reproduction. We assumed that kittens born from immune females are as susceptible as adult cats. We also assumed that infected queens do not participate in reproduction, as their progeny die as foetuses or kittens (Hoover *et al.*, 1983). The persistently viremic cats have a specific mortality rate α due to FeLV infection.

We first assumed that there is no vaccination nor removal of any cat, which is close to what is observed in our cat populations (Pontier and Artois, unpublished). Finally, the cat population dynamics may be described by the following set of differential equations:

$$\frac{dX}{dt} = b(X+Z) - mX - r\frac{N}{K}X - \sigma\frac{XY}{N} \qquad (2)$$

$$\frac{dY}{dt} = \sigma\frac{XY}{N}\pi - mY - r\frac{N}{K}Y - \alpha Y \qquad (3)$$

$$\frac{dZ}{dt} = \sigma\frac{XY}{N}(1-\pi) - mZ - r\frac{N}{K}Z. \qquad (4)$$

The equation for the total population N is obtained by adding the three previous ones:

$$\frac{dN}{dt} = rN\left(1-\frac{N}{K}\right) - (b+\alpha)\ Y. \qquad (5)$$

Model simulations have been carried out with the computer programme Dynamic (Rousseau, 1988), using parameter values in the literature.

Modelling FeLV Prophylaxis

Our aim is to compare the efficiency of control programmes, since two types of measures have been recommended to control FeLV infection, removal of infected cats and vaccination of susceptible ones.

The above model was modified to take into account the two prophylaxis programmes. First, let v be the instantaneous vaccination rate of susceptible cats. We assumed that only susceptible cats are vaccinated and that the vaccine is completely efficient for life, which is consistent with what is known about vaccination. Equations (2) and (4) were respectively modified as follows:

$$\frac{dX}{dt} = b(X+Z) - mX - r\frac{N}{K}X - \sigma\frac{XY}{N} - vX \qquad (2')$$

$$\frac{dZ}{dt} = \sigma\frac{XY}{N}(1-\pi) + vX - mZ - r\frac{N}{K}Z. \qquad (4')$$

Equations (3) and (5) are not modified.

For removal, we assumed that persistently viremic cats are removed at a per capita rate μ. Only Eqs. (3) and (5) are changed from the first model and become

$$\frac{dY}{dt} = \sigma\frac{XY}{N}\pi - mY - r\frac{N}{K}Y - \alpha Y - \mu Y \qquad (3')$$

$$\frac{dN}{dt} = rN\left(1-\frac{N}{K}\right) - (b+\alpha+\mu)\ Y. \qquad (5')$$

The model with both possible measures is represented Fig. 2. In order to compare both prophylactic measures, we assumed that they are not applied together. A stability analysis of the model with both vaccination and removal is performed in the Appendix.

RESULTS

Stability Analysis

Setting the time derivatives to zero in Eqs. (2), (3), (4), and (5), we find after some calculations three stationary points:

$$X_1^* = Y_1^* = Z_1^* = 0$$

$$X_2^* = K,\ Y_2^* = Z_2^* = 0$$

$$X_3^* = x^*N^*,\ Y_3^* = y^*N^*,\ Z_3^* = N^* - X_3^* - Y_3^*,$$

wherein

$$x^* = \frac{(\sigma-\alpha)(b+\alpha)-\sigma\pi b}{\sigma\pi(\sigma-b-\alpha)},$$

$$y^* = \frac{b(\sigma\pi-b-\alpha)}{(b+\alpha)(\sigma-b-\alpha)},$$

$$N^* = K\left[1-\frac{b(\sigma\pi-b-\alpha)}{r(\sigma-b-\alpha)}\right]$$

provided $0 < x^*,\ y^* < 1$.

The first two possibilities correspond to trivial solutions: eradication of either the cat population or the disease. The third one is the most interesting equilibrium point, where the disease persists within the population. As expected, the equilibrium size of the population N^* is less than the carrying capacity K. The depression of the population size is $b(\sigma\pi-b-\alpha)/r(\sigma-b-\alpha)$.

We show (Appendix) that there is a first threshold parameter, the reproductive number

$$R_0 = \sigma\pi/(b+\alpha).$$

The trivial state (X_2^*, Y_2^*, Z_2^*) is stable of and only if $R_0 < 1$; furthermore, introducing a secondary threshold parameter

$$R_1 = \frac{b}{m+(b+\alpha)\ y^*},$$

when $R_0 > 1$,

$$R_1 \leqslant 1 \text{ implies } N(t) \to 0 \text{ as } t \text{ goes to} + \infty$$

$$\text{and so do } X(t),\ Y(t),\ Z(t)$$

while

$$R_1 > 1 \text{ implies } X(t) \to X^*,\ Y(t) \to Y^* \text{ and } N(t) \to N^*.$$

Parameter Estimation

With the above condition for the existence of a nontrivial solution, it is possible to re-estimate the value of σ, from the other parameter values in the literature. We assume that birth and death rates ($b = 2.4,\ m = 0.6$), proportion of infected cats becoming persistently viremic ($\pi = 0.33$), and specific mortality rate ($\alpha = 0.53$) are the "less unknown" parameters, as they have been estimated from large samples, and by several authors, for α. The condition $1 < \sigma\pi/(b+\alpha)$ implies $\sigma > 8.9$, whereas the experimentally estimated value for σ is 3.08 to 11.76 according to age of cats. This will be discussed below.

Simulations

For birth rate, death rate, proportion of infected cats that become persistently viremic, and specific mortality rate for persistently viremic cats, we first used parameter values estimated in the literature (that is, respectively $b = 2.4,\ m = 0.6,\ \pi = 0.33$, and $\alpha = 0.53$). A simulation using the literature-estimated value for transmission rate σ (3.08 to 11.76) may lead to the second trivial solution, that is, extinction of the disease from the population. Then we arbitrarily chose for σ a value higher than 8.9, ($\sigma = 10$), in order to fill the condition for a nontrivial solution. In this case, the FeLV prevalence y^* is 4.28% and the depression of host population size is 6.98%. If we use the extreme values estimated for π and α (that is, 0.3 to 0.4 for π and 0.51 to 0.57 for α), the estimated prevalence ranges from 0.8 to 12.4%, and the depression of host population size from 1.34 to 20.18%.

Impact of Prophylaxis

In the first prophylaxis programme, vaccination, the stability analysis performed in the Appendix yields modified threshold parameters

$$R_0^V(v) = R_0(0, v) = \frac{\sigma\pi b}{(b+\alpha)(b+v)},$$

$$R_1^V(v) = R_1(0, v) = \frac{b}{m+(b+\alpha)\ y_3^*}.$$

The dynamics are similar to that without any prophylaxis programme with modified values x_3^*, y_3^*, and N_3^* given in (A8), (A9) and (A10).

For removal of infected cats, the modified threshold parameters now are

$$R_0^R(\mu) = R_0(\mu, 0) = \frac{\sigma\pi}{b+\alpha+\mu},$$

$$R_1^R(\mu) = R_1(\mu, 0) = \frac{b}{m+(b+\alpha+\mu)\ y_3^*}.$$

The dynamics is modified accordingly; see the Appendix.

Comparing Prophylaxis Programmes

Assume that $R_0 = \sigma\pi/(b+\alpha) > 1$ so that the disease-free trivial equilibrium is unstable. For v or μ large enough this equilibrium becomes stable again. More specifically, in the vaccination programme, the minimum value for the vaccination rate v is v_0 so that $R_0(0, v_0) = 1$, say

$$v_0 = \frac{b}{b+\alpha}\sigma\pi - b = b\left[\frac{\sigma\pi}{b+\alpha} - 1\right] = b(R_0 - 1).$$

v_0 can be interpreted as the minimum vaccination coverage required to eradicate a beginning infection from an homogeneous population. In the elimination programme, the minimum value for μ is μ_0 so that $R_0(\mu_0, 0) = 1$, say

$$\mu_0 = \sigma\pi - (b+\alpha) = (b+\alpha)\left[\frac{\sigma\pi}{b+\alpha} - 1\right]$$
$$= (b+\alpha)(R_0 - 1).$$

Clearly $0 < v_0 < \mu_0$ as long as $\alpha > 0$. Hence, if we are only interested in the respective magnitude of the effort parameters v and μ, the vaccination programme is more efficient than the removal of infected cats.

One may also check that when $R_0 = \sigma\pi/(b+a) > 1$ then

$$R_0^V(x) < R_0^R(x), \quad 0 < x < v_0.$$

DISCUSSION

Circulation of the FeLV Virus

When introduced in an homogeneous cat population, with the condition that $1 < R_0$, the model predicts that the infection is maintained, leading to a stable equilibrium. The predicted prevalence, using the extreme parameter values proposed in the literature for π and α, ranges from 0.8 to 12.4%. The host population depression (6.98%) is weak but detectable. These results suggest a stable (and rather low) prevalence when the disease is present, which actually was observed in numerous countries. FeLV instantaneous prevalence was measured as 13.6% in the United States (Ehrlund and Adams, 1984), 2.6% in Australia (Wilkinson and Thompson, 1987), 23.1% in Chile (Correa *et al.*, 1989), 4.5% in the United Kingdom (Hosie *et al.*, 1989), 3.2% in Japan (Ishida *et al.*, 1981), or 12.5% in Senegal (Alogninouwa *et al.*, 1992). In our populations, where FeLV is present, observed prevalence ($4.84 \pm 1.93\%$ for Aimargues and $11.11 \pm 3.02\%$ for Saint-Just Chaleyssin) lie in the same range of values as predicted by the model, with no significant differences over years (Fig. 1, Fromont *et al.*, 1996). These results suggest that the parameter values are realistic ones. However, such a comparison must be only semiquantitative, as the parameter evaluation is not accurate.

Parameter Estimation

Among the three parameters describing disease transmission, two (proportion of infected cats becoming persistently viremic π and specific death rate for persistently viremic cats α) are measurable directly. Such measures were made experimentally (Hardy *et al.*, 1976a; Weijer and Daams, 1976; Francis and Essex, 1980; Hardy, 1980; Ishida *et al.*, 1981), and, although only a few studies are available, we assume the results are accurate.

Estimation of the transmission rate σ, is an interesting result of the model. Although the model does not allow extensive study of this parameter, the first estimate shows that σ must be close to the upper limit of the range observed before (3.08 to 11.76) to ensure maintenance of the infection. However, the observed value should be considered as an approximation since the method used may lead to several biases. First, the measured duration is confounded with the latency period, when FeLV is present but viremia is not yet detectable (Hardy *et al.*, 1973). On the other hand, the observed cats were confined in households which is very different from the living conditions in natural populations. The first bias underestimates σ while the second one tends to overestimate it.

Impact of the Prophylaxis

In terms of quantitative results, the analysis of the model including prophylaxis permits concluding that vaccination of susceptible individuals is more efficient than removal of infected ones. Using the same kind of model, divergent results have been obtained, according to the disease. Vaccination has been found to be the best way to control rabies in red foxes (*Vulpes vulpes*), particularly in low and medium density fox populations (Anderson, *et al.*, 1981), while Barlow (1991) showed that bovine tuberculosis in New Zealand possum (*Trichosurus vulpecula*) populations is better controlled by culling. The differences in the pathogenesis of the diseases may explain this discrepancy. The type of model used to represent virus circulation also helps to explain the different results obtained. Using a spatial stochastic model and taking into account the role of fox population heterogeneity on the transmission of rabies, Smith and Harris (1991) found that vaccination may increase the

total fox density, and subsequently rabies incidence. Field results showed that vaccination is much more efficient than culling alone (Aubert *et al*., 1994), although it may suffer a relative failure in some places (Aubert, 1995). Thus characteristics of both population and disease are important parameters in modelling the impact of prophylaxis measures.

Moreover, the quantitative results concerning FeLV infection are not comparable to rabies or tuberculosis. The FeLV tests reveal whether a cat is contagious or not, so that it is possible to eliminate only seropositive cats and to immunise only negative cats, while vaccinating or culling programmes for fox rabies involve healthy as well as rabid animals, so that the proportion of individuals to be treated is not directly comparable.

Finally, a good measure of efficiency must take into account the cost and benefit of each proposal. The cost of FeLV prophylaxis must take into account several aspects. First, the cost of both FeLV tests and vaccines is supported by volunteer cat owners. Second, FeLV infection is not considered as dangerous for public health. Consequently, there is no obligatory prophylaxis for FeLV control, contrary to fox rabies or bovine tuberculosis (but we can image that a prophylaxis plan would start in order to protect wild felids like the European wild cat *Felis silvestris*, which may be threatened by FeLV infection Artois and Remond, 1994). Lastly, the psychological cost of a removal programme for FeLV control is heavy, because of the affective importance of cats. In conclusion, for all of these reasons, and for its higher efficiency, vaccination seems to be the best way to eradicate FeLV infection. A prophylaxis programme using both vaccination and elimination would be necessarily more efficient, but, as discussed above, in the field, the two measures do not have the same cost, and removal may only be considered as complementary to a vaccination plan.

Model Used

Compartment models are particularly interesting for their simplicity and they have been extensively studied (see, for example, Capasso, 1993), but they also include some constraints which may invalidate the conclusions concerning the disease studied. First, the chosen model assumes that the rates of crossing between the compartments are constant, which means that the characteristics of the population, environment, and disease do not evolve with time. However, this is a good hypothesis if we consider a short-term period so that the environment is stable, and we may consider that the population characteristics are stable, too, as this stability has been observed in our more than 10-year study period (Pontier, unpublished data). Moreover, our epidemiological study agrees with the results of the model; the prevalence for the 5 years of study, ranging from 3.45 to 5.45% for Aimargues and from 8.70 to 13.95% for Saint-Just Chaleyssin, with no significant tendency between years, which suggest that prevalence is at equilibrium (Fromont *et al*., in press).

A particular constraint is linked to the choice of the logistic model, relating to the choice of density dependent parameters. the fact that birth rate is constant at the population level is actually established in rural populations (Pontier, unpublished), but the density-dependence of the mortality rate is more difficult to observe. This density-dependence can lead to an excessive stability which may not exist in reality. However, the model results, that is, a global stability of the population size around the carrying capacity of the habitat K, seems convincing, compared to our long-term study (Pontier, unpublished) and to our epidemiological results (Fromont *et al*., 1996).

The third implicit hypothesis of the model is to consider both population and environment as homogeneous. Actually, this is not true either for the individuals, or for the environment. Differences exist in individual behaviour between cats according to their age and sex (Liberg, 1981; Turner and Bateson, 1988) and also according to the population (Liberg and Sandell, 1988; Pontier *et al*., 1995). Moreover, the spatial structure of cat populations is highly variable (Pontier, 1993), from dispersed cat groups in rural areas, where human habitats are scattered, to clustered and isolated populations, where the resources are heterogeneously distributed as in urban areas (Natoli and de Vito, 1988). Such variability may influence the transmission of infection, as was found for fox rabies (Smith and Harris, 1991). Actually, prevalence of FeLV infection differs with sex and age. Males seem to be more often infected than females (Weijer and Daams, 1976; Hosie *et al*., 1989), although contradictory results have been found (Alogninouwa *et al*., 1992) and, although kittens are more susceptible to the virus (Hoover *et al*., 1976), cats aged 3–5 years are more often infected, due to their higher exposure (Weijer and Daams, 1976; Grant *et al*., 1980; Hosie *et al*., 1989). Moreover, our epidemiological results show that two out of the four studied populations are free from FeLV infection (Table I), and a more detailed study suggests that the spatial structure of a cat population, and in particular its total size, may influence FeLV circulation (Fromont *et al*., 1996). To analyse FeLV persistence within populations according to population characteristics, it thus seems necessary to

take into account a fair amount of spatial structuring. Our future work will use another type of model which takes into account explicitly the spatial structure of cat populations.

APPENDIX: MATHEMATICAL ANALYSIS

Let $X(t)$, $Y(t)$, and $Z(t)$ be the respective numbers of susceptible, persistently viremic, and immune cats at time t. Thus $X(t)+Y(t)+Z(t)=N(t)$ represents the total population at time t. We consider the set of differential equations describing the dynamics of the cat population with both removal of infected cats and vaccination of susceptible ones, namely

$$\frac{dX}{dt}=b(X+Z)-mX-r\frac{N}{K}X-\sigma\frac{XY}{N}-\nu X, \quad \text{(A1)}$$

$$\frac{dY}{dt}=\sigma\frac{XY}{N}\pi-mY-r\frac{N}{K}Y-\alpha Y-\mu Y, \quad \text{(A2)}$$

$$\frac{dZ}{dt}=\sigma\frac{XY}{N}(1-\pi)-mZ-r\frac{N}{K}Z+\nu X. \quad \text{(A3)}$$

The differential equation for the total population N is derived upon adding (A1)–(A3), say

$$\frac{dN}{dt}=rN\left(1-\frac{N}{K}\right)-(b+\alpha+\mu)\ Y, \quad \text{(A4)}$$

with $r=b-m$ (see main text). Let us introduce the proportions $x=X/N$, the prevalence $y=Y/N$, and $z=Z/N$, so that $0\leqslant x, y, z\leqslant 1$ and $x+y+z=1$. One may express the time derivative of $x=X(1/N)$ as

$$\frac{dx}{dt}=\frac{1}{N}\frac{dX}{dt}-\frac{X}{N^2}\frac{dN}{dt}.$$

Substituting in this relation the explicit expressions of dX/dt and dN/dt given by the right-hand sides of (A1) and (A4) and using the simplification $z=1-x-y$, one gets after cautious algebraic manipulations

$$\frac{dx}{dt}=b-(b+\nu)\,x-by-(\sigma-b-\alpha-\mu)\,xy. \quad \text{(A5)}$$

Likewise one finds

$$\frac{dy}{dt}=[\sigma\pi x-(b+\alpha+\mu)+(b+\alpha+\mu)\ y]\ y. \quad \text{(A6)}$$

Lastly, the identity $Y=yN$ allows us to rewrite the differential equation (A4) as

$$\frac{dN}{dt}=N[r(1-N/K)-(b+\alpha+\mu)\ y]. \quad \text{(A7)}$$

Stability Analysis of the Prevalence

Looking for the stationary states of Eqs. (A5) and (A6) one gets two semitrivial states:

$$y_1^*=0, \quad x_1^*=\frac{b}{b+\nu},$$

$$y_2^*=1, \quad x_2^*=0;$$

and after some new algebraic manipulations or using a suitable software system for symbolic computation,

$$y_3^*=\frac{\sigma\pi b-(b+\alpha+\mu)(\nu+b)}{(b+\alpha+\mu)(\sigma-b-\alpha-\mu)}$$

$$=\frac{(\sigma\pi-b-\alpha-\mu)\,b-(b+\alpha+\mu)\,\nu}{(b+\alpha+\mu)(\sigma-b-\alpha-\mu)}, \quad \text{(A8)}$$

$$x_3^*=\frac{(\sigma-\alpha-\mu+\nu)(b+\alpha+\mu)-\sigma\pi b}{\sigma\pi(\sigma-b-\alpha-\mu)}. \quad \text{(A9)}$$

This latter is feasible, provided $0<x_3^*,\ y_3^*<1$ and $0\leqslant x_3^*+y_3^*\leqslant 1$.

The jacobian matrix of (A5)–(A6) reads

$$J(x,y)=\begin{pmatrix}-(b+\nu)-(\sigma-b-\alpha-\mu)\ y & -b-(\sigma-b-\alpha-\mu)\,x\\ \sigma\pi y & \sigma\pi x-(b+\alpha+\mu)+2(b+\alpha+\mu)\ y\end{pmatrix}.$$

The first stationary state supplies the threshold parameter R_0. One has

$$J(x_1^*,\ y_1^*)=\begin{pmatrix}-(b+\nu) & -b-(\sigma-b-\alpha-\mu)\dfrac{b}{b+\nu}\\ 0 & \sigma\pi\dfrac{b}{b+\nu}-(b+\alpha+\mu)\end{pmatrix},$$

hence $(x_1^*,\ y_1^*)$ is (locally) stable if and only if

$$R_0(\mu,\nu)=\frac{\sigma\pi b}{(b+\nu)(b+\alpha+\mu)}<1.$$

The second stationary state is always unstable. Actually the determinant and the trace of the jacobian matrix at $(x_2^*,\ y_2^*)$ are given by

$$\det J(x_2^*, y_2^*) = \sigma\pi b - (b+\alpha+\mu)(\sigma+\nu) + (b+\alpha+\mu)(\alpha+\mu),$$
$$\operatorname{Tr} J(x_2^*, y_2^*) = b+\alpha+\mu-(\sigma-\alpha-\mu+\nu).$$

Assuming first that $R_0(\mu, \nu) \geqslant 1$ it follows that $b+\alpha+\mu < \sigma$. Using this inequality in the last term on the right-hand side of the equation for the determinant, one finds

$$\det J(x_2^*, y_2^*) \leqslant (\sigma\pi - \sigma - \nu)(b+\alpha+\mu) < 0,$$

because $0 \leqslant \pi < 1$, yielding instability.

Next, when $R_0(\mu, \nu) < 1$ one gets $\sigma\pi b < (b+\nu)(b+\alpha+\mu)$ and, thus, from the equation for the determinant

$$\det J(x_2^*, y_2^*) < (b+\alpha+\mu)(b-\sigma+\alpha+\mu).$$

If $\operatorname{Tr} J(x_2^*, y_2^*) > 0$ the stationary state is unstable while $\operatorname{Tr} J(x_2^*, y_2^*) \leqslant 0$ implies $b-\sigma+\alpha+\mu \leqslant \nu-\alpha-\mu$ so that

$$\det J(x_2^*, y_2^*) < (b+\alpha+\mu)(\nu-\alpha-\mu)$$

and (x_2^*, y_2^*) is unstable as soon as $\nu \leqslant \alpha+\mu$.

Lastly, irrespective of $R_0(\mu, \nu)$ if $\alpha+\mu < \nu$ then

$$\det J(x_2^*, y_2^*) < \sigma\pi b - (b+\alpha+\mu)\,\sigma = \sigma(\pi b - \alpha - \mu) \leqslant 0.$$

Hence (0, 1) cannot be stable.

As far as the nontrivial stationary state is concerned one first shows that it is feasible if and only if $R_0(\mu, \nu) > 1$. Actually when this holds $y_3^* > 0$, the numerator of the first fraction in (A8) is positive and so is its denominator because $b+\alpha+\mu < \sigma$, as noted earlier in this case. Next, removing the negative term from the numerator of the second fraction in (A8) one gets $y_3^* < 1$. From (A6), any positive stationary state is a solution of

$$0 = \sigma\pi x^* - (b+\alpha+\mu)(1-y^*). \qquad \text{(A10)}$$

Thus $R_0(\mu, \nu) > 1$ implies

$$0 < x_3^* \leqslant \frac{b+\nu}{b}\frac{b+\alpha+\mu}{\sigma\pi}(1-y_3^*) < 1$$

and (x_3^*, y_3^*) is feasible, since $0 \leqslant x_3^* + y_3^* \leqslant 1$. Now assuming $R_0(\mu, \nu) \leqslant 1$ and $b+\alpha+\mu < \sigma$, one has $y_3^* \leqslant 0$. The remaining case is $R_0(\mu, \nu) \leqslant 1$ and $b+\alpha+\mu > \sigma$; if (x_3^*, y_3^*) is feasible then $0 \leqslant x_3^* \leqslant 1-y_3^*$ and it follows from Eq. (A10) that

$$0 \leqslant (\sigma\pi - b - \alpha - \mu)(1-y_3^*) < 0,$$

a contradiction.

Going back to the stability of (x_3^*, y_3^*) one has

$$\operatorname{Tr} J(x_3^*, y_3^*) = -\nu\frac{b+\alpha+\mu}{\sigma-b-\alpha-\mu} - \frac{\sigma\pi b}{b+\alpha+\mu} + \frac{\sigma\pi - b - \alpha - \mu}{\sigma-b-\alpha-\mu}\,b.$$

Now $R_0(\mu, \nu) > 1$ implies $\sigma > \sigma\pi > b+\alpha+\mu$ so that

$$0 < \frac{\sigma\pi - b - \alpha - \mu}{\sigma - b - \alpha - \mu} < 1$$

while

$$\frac{\sigma\pi}{b+\alpha+\mu} > \frac{\sigma\pi b}{(b+\alpha+\mu)(b+\nu)} = R_0(\mu, \nu) > 1.$$

Thus $\operatorname{Tr} J(x_3^*, y_3^*) < 0$. Lastly, from (A5) and (A6) (x_3^*, y_3^*) is a solution of

$$(b+\nu) + (\sigma - b - \alpha - \mu)\,y_3^* = b\frac{1-y_3^*}{x_3^*} = b\frac{\sigma\pi}{b+\alpha+\mu}.$$

Using these relations the determinant can be written

$$\det J(x_3^*, y_3^*) = \sigma\pi(\sigma - b - \alpha - \mu)\,x_3^* y_3^* > 0.$$

Hence, (x_3^*, y_3^*) is (locally) stable as long as it exists.

Stability with Prophylaxis

In this setting when $R_0(\mu, \nu) < 1$ the prevalence $y(t)$ goes to 0 as t goes to $+\infty$, Eq. (A7) for N is asymptotically equivalent to the logistic equation (1), so that as t goes to $+\infty$

$$N(t) \to K,\ X(t) \to \frac{b}{b+\nu}K, \quad Y(t) \to 0, \quad Z(t) \to \frac{\nu}{b+\nu}K.$$

Next, if $R_0(\mu, \nu) > 1$, Eq. (A7) is asymptotically equivalent to the differential equation of the logistic type

$$\frac{dP}{dt} = P\left[\, b - m - (b+\alpha+\mu)\,y_3^* - \frac{r}{K}P\right].$$

Introducing the second threshold parameter $R_1(\mu, \nu)$,

$$R_1(\mu, \nu) = \frac{b}{m + (b+\alpha+\mu)\,y_3^*},$$

one obtains as t goes to $+\infty$

$R_1(\lambda, \mu) \leqslant 1$, which implies $N(t) \to 0$,

as well as $X(t)$, $Y(t)$, and $Z(t) \to 0$

while

$$R_1(\lambda, \mu) > 1 \text{ implies } N(t) \to N_3^*(\mu, \nu)$$

with

$$N_3^*(\mu, \nu) = \frac{K}{r}\left[b - m - (b + \alpha + \mu)\, y_3^*\right] \qquad \text{(A11)}$$

and $X(t) \to x_3^* N_3^*$, $Y(t) \to y_3^* N_3^*$.

Stability without Prophylaxis

The conclusion follows from the previous case, upon setting $\mu = \nu = 0$.

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REFERENCES

Alogninouwa, T., Kamara, B., Kaboret, Y., and Parent, R. 1992. Prévalence estimée de l'infection par le virus leucémogène félin (FeLV) en milieu urbain à Dakar (Sénégal), *Rev. Méd. Vét.* **143**, 553–555.

Anderson, R. M., Jackson, H. C., May, R. M., and Smith, A. M. 1981. Population dynamics of fox rabies in Europe, *Nature* **289**, 765–771.

Anderson, R. M., and May, R. M. 1991. "Infectious Diseases of Humans: Dynamics and Control," Oxford Univ. Press, Oxford.

Artois, M., and Remond, M. 1994. Viral diseases as a threat to free-living wild cats (*Felis silvestris*) in continental Europe, *Vet. Rec.* **134**, 651–652.

Aubert, M., Flamand, A., and Kieny, M. P. 1994. La rage bientôt éradiquée en Europe occidentale?, *La Recherche* **265**, 582–584.

Aubert, M. 1995. Rage en Europe occidentale—Une concertation difficile, *Sem. Vét.* **766**, 38.

Bailey, N. T. J. 1994. Prediction and validation in the public health modelling of HIV/AIDS, *Stat. Med.* **13**, 1933–1943.

Barlow, N. D. 1991. Control of endemic bovine Tb in New Zealand possum populations: Results from a simple model, *J. Appl. Ecol.* **28**, 794–809.

Calhoon, R. E., and Haspell, C. 1989. Urban cats populations compared by season, subhabitat and supplemental feeding, *J. Appl. Ecol.* **58**, 321–328.

Capasso, V. 1993. Mathematical modelling of transmission mechanisms of infectious diseases, *in* "Mathematics Applied to Biology and Medicine" (V. Demongeot and V. Capasso, Eds.), pp. 171–186, Wuerz, Winnipeg.

Charreyre, C., and Pedersen, N. C. 1991. Study of Feline Leukemia Virus immunity, *J.A.V.M.A* **199**, 1316–1324.

Correa, J. A., Segovia, P., and Rojas, J. 1989. Detection of infection by Feline Leukemia Virus through the Elisa test in Santiago, Chile, *Arch. Med. Vet.* **21**, 48–50.

Courchamps, F., Pontier, D., Langlais, M., and Artois, M. 1995. Population dynamics of Feline Immunodeficiency Virus within populations of cats, *J. Theor. Biol.* **175**, 553–560.

Ehrlund, L., and Adams, A. 1984. A seroepidemiologic study of Feline Leukemia Virus in San Antonio, *Vet. Met.* **79**, 625–628.

Francis, D. P., Essex, M., and Hardy, W. D., Jr. 1977. Excretion of FeLV by naturally infected pet cats, *Nature* **269**, 252–254.

Francis, D. P., Essex, M., Cotter, S., and Hardy, W. D., Jr. 1979. Feline Leukemia Virus infections: The significance of chronic viremia, *Leukemia Res.* **3**, 435–441.

Francis, D. P., and Essex, M. 1980. Epidemiology of feline leukemia, *in* "Feline Leukemia Virus" (W. D. Hardy, Jr., M. Essex, and A. J. McClelland, Eds.), pp. 127–131, Elsevier North Holland, New York.

Fromont, E., Pontier, D., and Artois, M. 1996. Spatial structure of cat populations and circulation of feline viruses, *Acta Oecologica* **17**, 609–620.

Grant, C. K., Essex, M., Gardner, M. B., and Hardy, W. D., Jr. 1980. Natural Feline Leukemia Virus infection and the immune response of cats of different ages, *Cancer Res.* **40**, 823–829.

Hardy, W. D., Jr., Hirshaut, Y., and Hess, P. 1973. Detection of Feline Leukemia Virus and other mammalian oncornaviruses by immunofluorescence, *in* "Unifying Concepts of Leukemia" (R. M. Dutcher and L. Chieco-Bianchi, Eds.), pp. 778–799, Karger, Basel.

Hardy, W. D., Jr., Hess, P. W., and McEwen, E. G. 1976a. The biology of Feline Leukemia Virus in the natural environment, *Cancer Res.* **36**, 582–588.

Hardy, W. D., Jr., McClelland, A. J., Zuckermann, E. E., Hess, P. W., Essex, M., Cotter, S. M., McEwen, E. G., and Hayes, A. A. 1976b. Prevention of the contagious spread of Feline Leukemia Virus and the development of leukemia in pet cats, *Nature* **263**, 326–328.

Hardy, W. D., Jr. 1980. The virology, immunology and epidemiology of the Feline Leukemia Virus, *in* "Feline Leukemia Virus" (W. D. Hardy, Jr., M. Essex, and A. J. McClelland, Eds.), pp. 33–78, Elsevier North Holland, New York.

Hardy, W. D., Jr. 1993. Feline oncoretroviruses, *in* "The Retroviridae, Vol. 2" (J. A. Levy, Ed.), pp. 109–180, Plenum, New York.

Hethcote, H. W., and Yorke, J. A. 1984. "Gonorrhea Transmission Dynamics and Control," Lecture Notes in Biomathematics, Vol. 56, Springer-Verlag, Berlin.

Hethcote, H. W., and Van Ark, J. W. 1987. Epidemiological models for heterogeneous populations: Proportionate mixing, parameter estimation, and immunization programs, *Math. Biosci.* **84**, 85–118.

Hoover, E. A., Olsen, R. G., Hardy, W. D., Jr., Schaller, J. P., and Mathes, L. E. 1976. Feline leukemia virus infection: Age-related variation in response of cats to experimental infection, *J. Natl. Cancer Inst.* **57**, 365–369.

Hoover, E. A., Olsen, R. G., Mathes, L. E., and Schaller, J. P. 1977. Relationship between Feline Leukemia Virus antigen expression and viral infectivity in blood, bone marrow, and saliva of cats, *Cancer Res.* **37**, 3707–3710.

Hoover, E. A., Rojko, J. L., and Quackenbush, S. L. 1983. Congenital Feline Leukemia Virus infection, *Leukemia Rev. Int.* **1**, 7–8.

Hoover, E. A., and Mullins, J. I. 1991. Feline leukemia virus infection and diseases, *J.A.V.M.A* **199**, 1287–1297.

Hosie, M. J., Robertson, C., and Jarrett, O. 1989. Prevalence of Feline Leukemia Virus and antibodies to Feline Immunodeficiency Virus in cats in the United Kingdom, *Vet. Rec.* **125**, 293–297.

Ishida, T., Kawai, S., and Fujiware, K. 1981. Detection of Feline Leukemia Virus infection in Tokyo area by enzyme-linked immunosorbent assay (ELISA), *Jpn. J. Vet. Sci.* **43**, 871–874.

Jarrett, O., Golder, M. C., and Stewart, M. F. 1982. Detection of transient and persistent Feline Leukemia Virus infection, *Vet. Rec.* **110**, 225–228.

Jarrett, W. F. H., Jarrett, O., McKey, L., Laird, H., Hardy, W. D., Jr., and Essex, M. 1973. Horizontal transmission of leukemia virus and leukemia in the cat, *J. Natl. Cancer. Inst.* **51**, 833–841.

Legay, J. M., and Pontier, D. 1985. Relation âge-fécondité dans les populations de chats domestiques, *Felis catus*, *Mammalia* **49**, 395–402.

Liberg, O. 1981. "Predation and Social Behaviour in a Population of Domestic Cats. An evolutionary Perspective," Ph.D. thesis, University of Lund.

Liberg, O., and Sandell, M. 1988. Spatial organization and reproductive tactics in the domestic cat and other felids, *in* "The Domestic Cat: The Biology of Its Behaviour" (D. C. Turner and P. Bateson, Eds.), pp. 83–98, Cambridge Univ. Press, Cambridge.

Macdonald, D. W. 1980. "Rabies and Wildlife. A Biologist's Perspective," Oxford Univ. Press, Oxford.

McClelland, A. J., Hardy, W. D., Jr., and Zuckerman, E. E. 1980. Prognosis of healthy FeLV infected cats, *in* "Feline Leukemia Virus" (W. D. Hardy, Jr., M. Essex, and A. J. McClelland, Eds.), pp. 121–126, Elsevier North Holland, New York.

Natoli, E., and De Vito, E. 1988. The mating system of feral cats living in group, *in* "The Domestic Cat. The Biology of Its Behaviour" (D. C. Turner and P. Bateson, Eds.), pp. 99–108, Cambridge Univ. Press, Cambridge.

Pacitti, A. M., and Jarrett, O. 1985. Duration of the latent state in Feline Leukaemia Virus infections, *Vet. Rec.* **117**, 472–474.

"Panel Report on the Colloquium on Feline Leukemia Virus/Feline Immunodeficiency Virus: Tests and Vaccination." 1991. *J.A.V.M.A.* **199**, 1273–1469.

Pedersen, N. C., Theilen, G., Keane, M. A., Fairbanks, L., Mason, T., Orser, B., Chen, C. H., and Allison, C. 1977. Studies of naturally transmitted Feline Leukemia Virus infection, *Am. J. Vet. Res.* **38**, 1523–1531.

Pedersen, N. C., Johnson, L., and Ott, R. L. 1985. Evaluation of a commercial Feline Leukemia Virus vaccine for immunogenicity and efficacy, *Feline Pract.* **15**, 7–20.

Pontier, D. 1984. "Contribution à la biologie et à la génétique des populations de chats domestiques (*Felis catus*)," Ph.D. thesis, Université Claude Bernard, Lyon I.

Pontier, D. 1993. Analyse des traits d'histoire de vie chez les Mammifères. Mémoire d'habilitation à diriger les recherches, Université Claude Bernard, Lyon I.

Pontier, D., Rioux, N., and Heizmann, A. 1995. Differences in body weight associated with coat color genes in domestic cat *Felis catus*: evidence of selection on the orange allele, *Oikos* **73**, 299–308.

Rojko, J. L., Hoover, E. A., Mathes, L. E., Olsen, R. G., and Schaller, J. P. 1979. Pathogenesis of experimental Feline Leukemia Virus infection, *J. Natl. Cancer Inst.* **63**, 759–760.

Rojko, J. L., Hoover, E. A., Quackenbush, S. L., and Olsen, R. G. 1982. Reactivation of latent Feline Leukaemia Virus infection, *Nature* **298**, 385–388.

Rojko, J. L., and Olsen, R. G. 1984. The immunobiology of the Feline Leukemia Virus, *Vet. Immunol. Immunopathol* **6**, 107–165.

Rousseau, B. 1988. "Vers un environnement de résolution de problèmes en biométrie, apport des techniques de l'intelligence artificielle et de l'interaction graphique," Ph.D. thesis, Université Claude Bernard, Lyon I.

Smith, G. C., and Harris, S. 1991. Rabies in urban fox (*Vulpes vulpes*) in Britain: the use of a spatial model to examine the pattern of spread and evaluate the efficacy of different control regimes, *Phil. Trans. R. Soc. Lond.* **334**, 459–479.

Turner, D. C., and Bateson, P. 1988. "The domestic Cat: The Biology of its Behaviour," Cambridge Univ. Press, Cambridge.

Verhulst, P. F. 1838. Notice sur la loi que la population suit dans son accroissement, *Corr. Math. Phys.* **10**, 113.

Weijer, K., and Daams, J. H. 1976. The presence of leukaemia (lymphosarcoma) and Feline Leukaemia Virus (FeLV) in cats in the Netherlands, *J. Small Anim. Pract.* **17**, 649–659.

Weijer, K., Uytdehaag, F., and Osterhaus, A. 1986. Control of Feline Leukemia Virus infection by a removal programme, *Vet. Rec.* **119**, 555–556.

Wilkinson, G. T., and Thompson, H. L. 1987. A survey of the prevalence of Feline Leukaemia Virus infection in cats in south-east Queensland, *Aust. Vet. Pract.* **17**, 195–197.

Wong-Stall, R., and Gallo, R. C. 1985. Human T-lymphotropic retroviruses, *Nature* **317**, 395–403.