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Dynamics of a feline retrovirus (FeLV) in host populations with variable spatial structure

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The predictions of epidemic models are remarkably affected by the underlying assumptions concerning host population dynamics and the relation between host density and disease transmission. Furthermore, hypotheses underlying distinct models are rarely tested. Domestic cats (*Felis catus*) can be used to compare models and test their predictions, because cat populations show variable spatial structure that probably results in variability in the relation between density and disease transmission. Cat populations also exhibit various dynamics. We compare four epidemiological models of Feline Leukaemia Virus (FeLV). We use two different incidence terms, i.e. proportionate mixing and pseudo-mass action. Population dynamics are modelled as logistic or exponential growth. Compared with proportionate mixing, mass action incidence with logistic growth results in a threshold population size under which the virus cannot persist in the population. Exponential growth of host populations results in systems where FeLV persistence at a steady prevalence and depression of host population growth are biologically unlikely to occur. Predictions of our models account for presently available data on FeLV dynamics in various populations of cats. Thus, host population dynamics and spatial structure can be determinant parameters in parasite transmission, host population depression, and disease control.

Keywords: host–parasite dynamics; model; population; FeLV; domestic cat; *Felis catus*

1. INTRODUCTION

Models are widely used to study parasite dynamics in non-human hosts (Anderson 1995; Grenfell & Dobson 1995), particularly to investigate two seminal questions. Do parasites have the potential to depress the growth rate of host populations? How can we control diseases? As the number of models increases, important questions arise concerning the relevance of models to field data. The considerable variability of predictions among quite similar models suggests that choice of the underlying assumptions is a critical step in disease modelling (Getz & Pickering 1983).

Biological data are necessary to build realistic models, and to test model predictions (Grenfell & Dobson 1995). However, data are often lacking and assumptions underlying models are generally not tested. Furthermore, in a specific host–parasite system, a single field situation is generally examined for population dynamics and structure. Thus, outcomes of different models cannot be compared, and the choice of a given model can be hazardous. A notable exception is rabies, for which data on the spread of rabies among low-density red foxes, *Vulpes vulpes*, have been used to predict rabies propagation in high-density urban populations (Smith & Harris 1991). However, rabies in urban foxes remains a speculative scenario.

There are two central assumptions that dramatically affect model predictions concerning the two questions raised, either when considering microparasites (viruses, bacteria, protozoa and fungi), or macroparasites (helminths and arthropods). First, the manner in which host density influences birth and death rates has important consequences on the predicted relative efficiency of vaccination and culling (Barlow 1995). Second, the function describing parasite incidence (i.e. the number of new infections per time unit) governs the predicted possibility for parasites to limit host population growth (Getz & Pickering 1983), and the degree to which host population size must be reduced to control disease (Barlow 1995). Incidence itself depends on the relation between population size, measured as a number or a density, and rate of contact for efficient disease transmission. Thus, modellers must integrate data on host home range size and contact behaviour as influenced by host density (Barlow 1995; Diekmann *et al.* 1995).

The domestic cat *Felis catus* provides a good example for evaluation of models describing the influence of population characteristics (dynamics, spatial organization) on parasite dynamics. As in other carnivores, the density and spatial structure of cats depend on the abundance and distribution of resources (Macdonald 1983). Cats are found in a wide variety of ecological conditions and have developed highly variable population structures, ranging from solitary-living individuals in low-density populations to social grouping in high-density populations (Liberg & Sandell 1988).

An important pathogen of felids is Feline Leukaemia Virus (FeLV), a retrovirus causing immunodepression

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Received 2 February 1998 *Accepted* 9 March 1998

and tumoral disorders. The transmission, pathogenicity and epidemiology of the virus have been studied extensively (Hardy 1993). FeLV causes significant depression of cat population growth through direct mortality and reduction of queen fecundity. There are two models of FeLV dynamics in cat populations which have been built (Lubkin *et al.* 1996; Fromont *et al.* 1997). Both models assumed cat population growth to follow a logistic curve and FeLV incidence to be frequency-dependent (proportionate mixing model). Both models predicted that FeLV is endemic in cat populations, and this was actually observed (Fromont *et al.* 1997). However, these assumptions are valid only in a fraction of cat populations. In fact, situations that were not predicted by the models have also been observed, for example in a small rural population where FeLV viraemia was repeatedly absent while opportunities for virus introduction were numerous (Fromont *et al.* 1998). The prevalence of FeLV is also highly variable in dense urban populations (Xémar 1997).

Here we aim to build four models, using different parasite incidence terms and host population dynamics, and to describe predictions concerning virus propagation, host population depression, and virus control. To know whether the dynamics predicted by different models reflect actual differences in the field, we will compare the dynamics predicted with field data found in the literature and in our epidemiological survey (Fromont *et al.* 1996, 1998).

2. MATERIAL AND METHODS

(a) *Host population growth*

We first present the elements of cat populations' dynamics that are necessary to model FeLV dynamics. Owners exert a relative control on the demography of cats living in dwellings. Most litters are unplanned, but elimination of kittens at birth acts as a limitation. Population size of pet cats has been shown to be stable over time (Pontier *et al.* 1995; Patronek *et al.* 1997). For this reason, both previous models considered logistic growth of cat populations (Lubkin *et al.* 1996; Fromont *et al.* 1997).

However, when a cat population settles out of human control, no density-dependence occurs as long as the carrying capacity of the environment is much higher than the population size, and a phase of exponential growth is expected. The establishment of new cat populations is frequent both in sites where clumped resources favour high-density populations (such as in urban areas, see Natoli & De Vito 1991), and in non-anthropized habitats (such as islands where cats were released from boats). Exponential growth of island populations has been shown to last several decades (Van Rensburg *et al.* 1987). In contrast, in urban sites, the phase of exponential growth is probably of short duration because the carrying capacity is low and growth rate is high.

In our model, $P = P(t) \geqslant 0$ equals the total number of cats at time t. Let $\beta(P) \geqslant 0$ and $\delta(P) \geqslant 0$ be the fertility and mortality functions, respectively.

$$g(P) = \beta(P) - \delta(P), \quad P \geqslant 0,$$

equals the intrinsic growth rate for a total population of size P. When the cat population is free from FeLV and no external supply is considered, cat dynamics are governed by the ordinary differential equation:

$$\frac{\mathrm{d}P}{\mathrm{d}t} = (\beta(P) - \delta(P))P, \quad t < 0; P(0) < 0. \tag{1}$$

We shall consider two types of intrinsic growth rates. First, we consider a logistic-type form, i.e. $\beta(P) \geqslant 0$ and $\delta(P) \geqslant 0$ are density-dependent:

$$\begin{cases} \beta(P) = b, \ 0 > m > b, \ r = b - m; \ K < 0, \\ \delta(P) = m + rP/K, \end{cases}$$

where b and m are the natural birth and death rates, respectively. K is the unique positive stationary solution of equation (1), asymptotically stable for $P(0) > 0$; K equals the carrying capacity of the environment. Second, we consider a Malthusian growth, i.e. $\beta(P) = b$ and $\delta(P) = m$.

(b) *Host population structure*

We now introduce some features of host population structure and describe the relation between density and contact rate. Cat populations show variable density, from 1 cat km^{-2} in places where food resources are scarce, to 2000 cats km^{-2} around abundant, clumped food resources (Liberg & Sandell 1988). At very low density (less than 10 cats km^{-2}, as in non-anthropized areas), home ranges are large (up to 4 km^2 for males, Liberg & Sandell 1988) and encounters among adults are uncommon (Kerby & Macdonald 1988). However, a minimal contact rate is maintained to ensure reproduction. At intermediate density (10–100 cats km^{-2}, as in rural areas), the frequency of contacts increases with density. However, the increase in the frequency of contacts among cats is limited by a reduction in home range size (Liberg & Sandell 1988). Finally, when food resources are highly clumped, cats congregate at very high density (more than 1000 cats km^{-2}, such as in urban stray cat groups). Home ranges of both sexes are small (0.005–0.02 km^2) and overlap considerably, and contacts are frequent (Natoli & De Vito 1991). Thus, we can propose a qualitative relation between host density and contact rate (figure 1). Parts 1, 2 and 3 of figure 1 were proposed by Diekmann *et al.* (1995). We added part 4 to account for increased contact rate at very high density, as was also hypothesized for urban foxes (Smith & Harris 1991). Figure 1 gives a first approximation of variability in cat population structure.

There are two classical models that account for stable or increasing contact rate with density. Let c be the average number of contacts between two cats. In the proportionate mixing model (parts 1 and 3 of figure 1), one assumes that c is a constant (Hethcote & Yorke 1984). In the mass action model (parts 2 and 4), c is proportional to population size, say $c = kP$ (Anderson & May 1979).

(c) *The virus*

For a description of the propagation of FeLV within host populations we refer to Lubkin *et al.* (1996), Fromont *et al.* (1997) and references therein. The course of FeLV infection begins with a viral replication phase that lasts up to four months; this stage may have two possible outcomes. First, about two-thirds of infected cats produce an efficient immune response and stop viral replication. These cats are clinically recovered. They are no longer infectious and have a normal life expectancy. Although the duration of immunity is not ascertained, we shall consider these cats as immune. Immune females have a normal birth rate and their kittens become rapidly susceptible. On the contrary, about one-third of infected cats fail to develop the appropriate immune response and become persistently viraemic. Viraemia causes immunosuppressive and proliferative disorders that lead to death within a few weeks to several years. The fertility of viraemic females is strongly reduced: most pregnancies lead to abortion, and kittens born to viraemic mothers are mostly

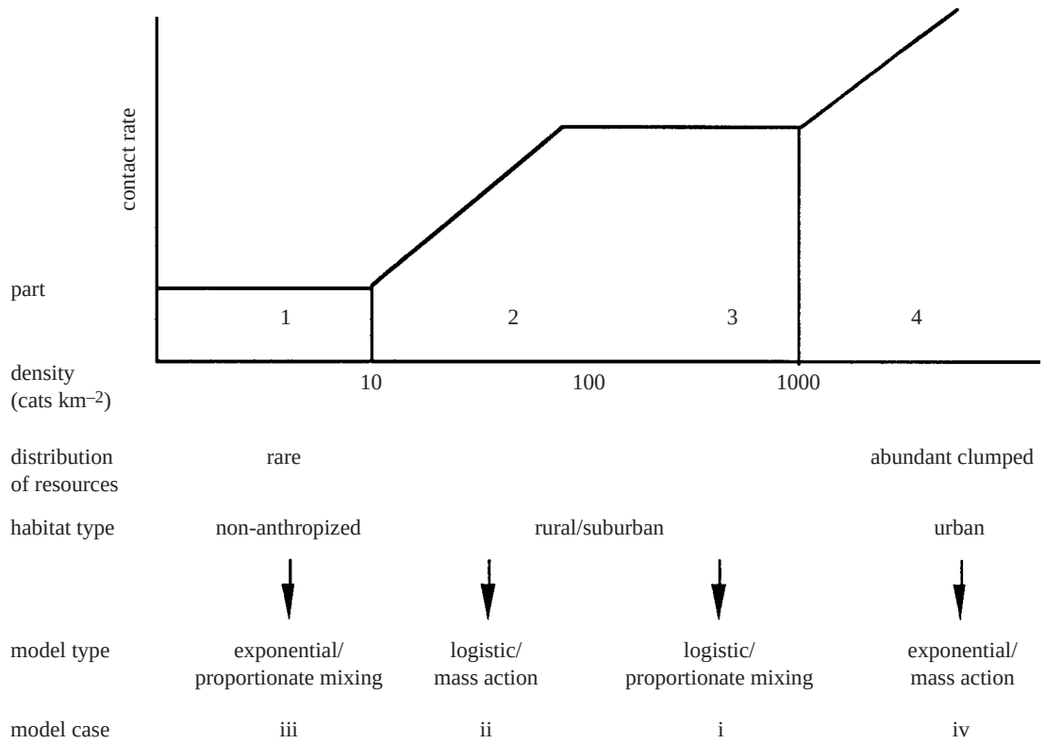

Figure 1. Qualitative features of contact rate as a function of density in domestic cats, and models used for each of four parts. Part 1 accounts for non-anthropized environments. At very low density, home ranges are large and mobility of individuals is high so as to maintain a minimal contact rate for reproduction. Parts 2 and 3 correspond to rural environments: contact rates increase as a mechanistic consequence of density (part 2); at medium density, home range size diminishes as density increases (part 3). Part 4 corresponds to highly dense populations: home ranges of all individuals overlap considerably. Orders of magnitude for threshold densities are given. Model case refers to models described in table 1.

viraemic and die at an early age. Hence, one may assume that vertical transmission is ineffective (Hardy 1993).

We denote $X=X(t)$, $Y=Y(t)$ and $Z=Z(t)$ as the respective numbers of susceptible, infectious and immune individuals, so that $P=P(t)=X(t)+Y(t)+Z(t)$ equals the total population. Let $\sigma(X,Y,Z)$ be the incidence function, i.e. the number of newly infected cats per time unit. We neglect the transient stage as being too short and let π, $0\leqslant\pi\leqslant1$, be the proportion of infected cats that develop viraemia. Lastly, $\alpha>0$ equals the specific death rate of infectious cats due to FeLV, while $1/\alpha$ equals the duration of the infectious stage.

The dynamics of FeLV within a population of cats are governed by the following set of differential equations:

$$\begin{cases} \dfrac{dX}{dt} = -\sigma(X,Y,Z) + \beta(P)(X+Z) - \delta(P)X, \\ \dfrac{dY}{dt} = \pi\sigma(X,Y,Z) - \alpha Y - \delta(P)Y, \\ \dfrac{dZ}{dt} = (1-\pi)\sigma(X,Y,Z) - \delta(P)Z, \end{cases} \tag{2}$$

together with the initial conditions $X(0)>0$, $Y(0)>0$, $Z(0)\geqslant0$.

The incidence function takes one of the following two forms: $\sigma(X,Y,Z)=\sigma_{pm}XY/P$, $\sigma_{pm}>0$ for the proportionate mixing model; or $\sigma(X,Y,Z)=\sigma_{ma}XY$, $\sigma_{ma}>0$ for the mass action model.

An important feature concerning FeLV propagation is the possible eradication of the viraemia through vaccination of susceptible cats, elimination of infectious ones, or both (Lubkin *et al.* 1996; Fromont *et al.* 1997). The efficiency of the two prophylaxis methods can be analysed upon introducing to equations (2) a vaccination rate v in the susceptible class and an elimination rate μ in the infectious class. This yields

$$\begin{cases} \dfrac{dX}{dt} = -\sigma(X,Y,Z) + \beta(P)(X+Z) - \delta(P)X - vX, \\ \dfrac{dY}{dt} = \pi\sigma(X,Y,Z) - \alpha Y - \delta(P)Y - \mu Y, \\ \dfrac{dZ}{dt} = (1-\pi)\sigma(X,Y,Z) - \delta(P)Z + vX. \end{cases} \tag{3}$$

3. RESULTS

(a) *Dynamics of the host–parasite system*

Mathematical analysis of the models is presented in an electronic appendix available at `http://www.pubs.royalsoc.ac.uk/publish/pro_bs/jun98pb2/htm`. The main results of the stability analysis are illustrated in figure 2 and summarized in table 1.

(i) *Proportionate mixing–logistic model*

In the proportionate mixing–logistic case, system (2) reads

$$\begin{cases} \dfrac{dX}{dt} = b(X+Z) - mX - rXP/K - \sigma_{pm}XY/P - vX, \\ \dfrac{dY}{dt} = -mY - rYP/K + \pi\sigma_{pm}XY/P - \alpha Y - \mu Y, \\ \dfrac{dZ}{dt} = -mZ - rZP/K + (1-\pi)\sigma_{pm}XY/P + vX. \end{cases} \tag{4}$$

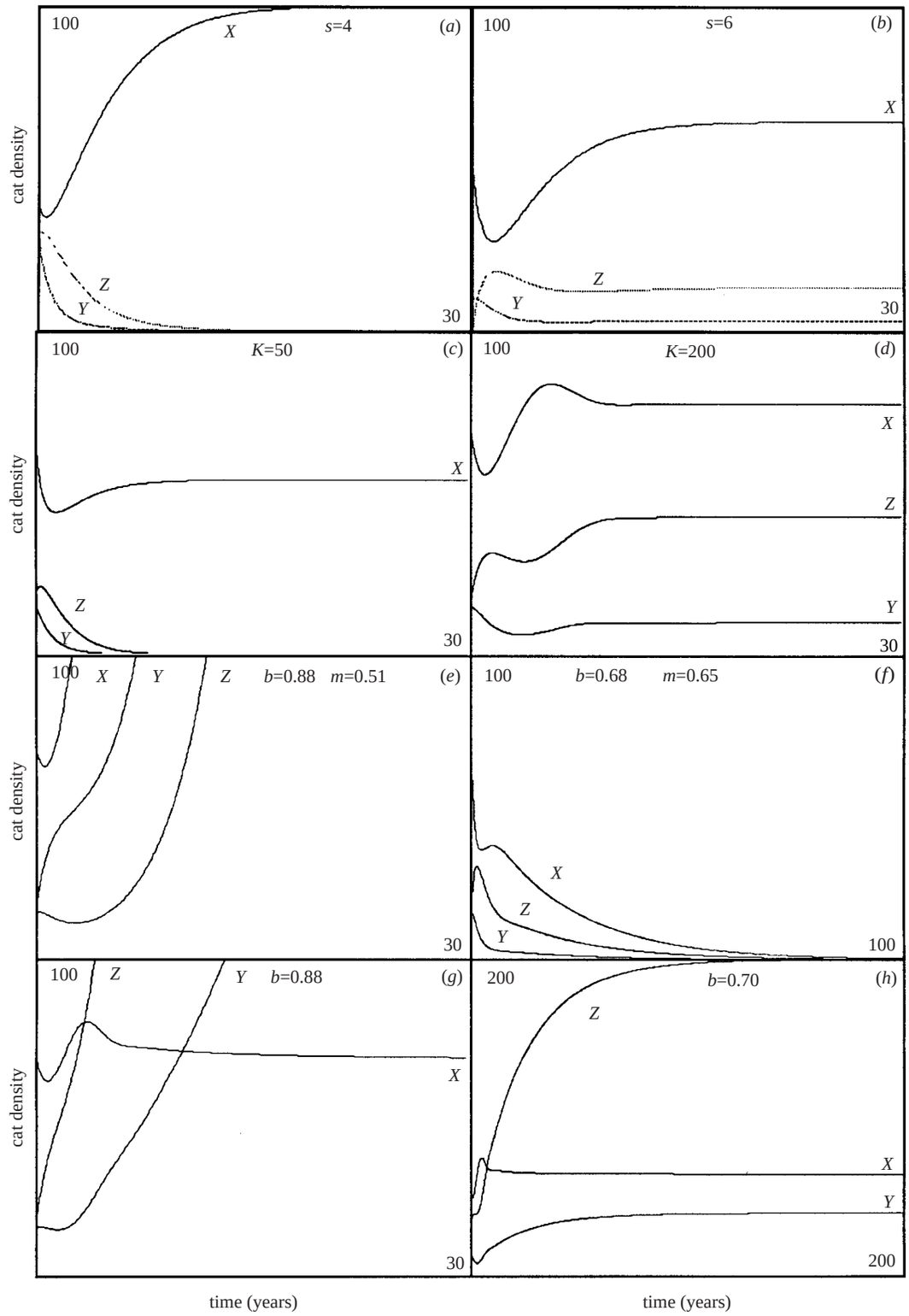

Figure 2. Simulations of the main stationary states of each of four models of FeLV infection. X, Y and Z represent numbers of susceptible, viraemic and immune cats, respectively. Unless otherwise stated, values for parameters are $b=0.88$ females female^{-1} yr^{-1}, $m=0.51$/year, $K=100$, $\pi=0.33$, $\alpha=0.76$/year, $\sigma_{pm}=6$ and $\sigma_{ma}=0.06$. (*a*,*b*) Case (i) or proportionate mixing–logistic model. (*a*) $\sigma_{pm}=4$: FeLV goes extinct ($R_0^{pm}<1$). (*b*) $\sigma_{pm}=6.0$: infection establishes in the population ($R_0^{pm}>1$). (*c*,*d*) Case (ii) or mass action–logistic model. (*c*) $K=50$: FeLV goes extinct ($R_0^{ma}<1$). (*d*) $K=200$: infection establishes in the population ($R_0^{ma}>1$). (*e*,*f*) Case (iii) or proportionate mixing–exponential model. (*e*) Basic values of parameters lead to the non-trivial stationary state: FeLV develops in the population, which maintains an exponential growth. (*f*) $b=0.68$ and $m=0.65$: host population goes extinct under the effect of the disease. (*g*,*h*) Case (iv) or mass action–exponential model. (*g*) $b=0.88$ (observed value in rural populations): no stable equilibrium can be reached. (*h*) $b=0.70$: the system tends to the non-trivial stationary state: the model predicts that host population size is stabilized by the disease after a long transient period (note time-scale).

Table 1. *Summary of model predictions for FeLV infection in domestic cats*

case	threshold population size for virus persistence?	extinction of host population under the effect of virus possible?	qualitative change in host population dynamics at endemic state
(i) proportionate mixing–logistic	no	yes (not shown, see figure 2)	decrease in population density
(ii) mass action–logistic	yes	no	decrease in population density
(iii) proportionate mixing–exponential	no	yes (but biologically unlikely)	no endemic stable state possible
(iv) mass action–exponential	no	no	limitation of population density (but biologically unlikely)

The equation for the total population thus reads

$$\frac{\mathrm{d}P}{\mathrm{d}t} = r\left(1 - \frac{P}{K}\right)P - (b + \alpha + \mu)\mathcal{Y}.$$

A total of three dynamics may be observed: eradication of the disease, destruction of the host population, or emergence of a stable endemic state. The reproduction number is

$$R_0^{\mathrm{pm}}(\mu,v) = \frac{\sigma_{\mathrm{pm}}\pi b}{(b+v)(b+\alpha+\mu)}.$$

When $R_0^{\mathrm{pm}}(\mu,v) < 1$, the host population settles down to its disease-free equilibrium,

$$X = \frac{b}{b+v}K, \quad \mathcal{Y} = 0, \quad \mathcal{Z} = \frac{v}{b+v}K.$$

When $R_0^{\mathrm{pm}}(\mu,v) > 1$, FeLV develops and a secondary threshold parameter arises:

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

with

$$y^* = \frac{(\sigma_{\mathrm{pm}}\pi - b - \alpha - \mu)b - (b+\alpha+\mu)v}{(b+\alpha+\mu)(\sigma_{\mathrm{pm}} - b - \alpha - \mu)}$$

where $R_1^{\mathrm{pm}}(\mu,v)$ is the net reproductive coefficient of the host population when the disease is endemic (Busenberg & Cooke 1993). Still assuming $R_0^{\mathrm{pm}}(\mu,v) > 1$, the asymptotic prevalence is y^* and (i) when $R_1^{\mathrm{pm}}(\mu,v) < 1$, the host population goes extinct; (ii) when $R_1^{\mathrm{pm}}(\mu,v) > 1$, a globally stable endemic state emerges:

$$P^* = K\left[1 - \frac{(b+\alpha+\mu)}{r}y^*\right],$$
$$X^* = x^*P^*, x^* = \frac{(\sigma_{\mathrm{pm}} - \alpha - \mu + v)(b+\alpha+\mu) - \sigma_{\mathrm{pm}}\pi b}{\sigma_{\mathrm{pm}}\pi(\sigma_{\mathrm{pm}} - b - \alpha - \mu)},$$
$$\mathcal{Y}^* = y^*P^*,$$
$$\mathcal{Z}^* = (1 - x^* - y^*)P^*,$$

where host population size is reduced to level P^* under the effect of the virus.

To analyse the effect of prophylaxis, assume that before starting any vaccination programme, an elimination programme is carried out at a rate $\mu \geqslant 0$; $\mu = 0$ means no elimination at all. The corresponding reproduction number equals

$$R_0^{\mathrm{pm}}(\mu,0) = \frac{\sigma_{\mathrm{pm}}\pi}{b+\alpha+\mu}.$$

Suppose moreover that this elimination programme is not efficient to eradicate FeLV, so that $R_0^{\mathrm{pm}}(\mu,0) > 1$. Thus, it becomes necessary to develop a vaccination program at a rate v. Vaccination will be efficient provided $R_0^{\mathrm{pm}}(\mu,v) < 1$, in which case, the proportion of vaccinated cats is given by $\mathcal{Z}/P = v/(b+v)$. We rewrite $R_0^{\mathrm{pm}}(\mu,v)$ as

$$R_0^{\mathrm{pm}}(\mu,v) = \frac{b}{b+v} \times \frac{\sigma_{\mathrm{pm}}\pi}{b+\alpha+\mu} = \left[1 - \frac{v}{b+v}\right] \times R_0^{\mathrm{pm}}(\mu,0).$$

It follows that vaccination will be efficient provided the proportion of vaccinated cats satisfies the inequality

$$\frac{\mathcal{Z}}{P} > 1 - \frac{1}{R_0^{\mathrm{pm}}(\mu,0)}. \tag{5}$$

Similar relations between the optimal proportion of vaccinated individuals and reproduction number in the absence of a vaccination programme are derived in Anderson *et al.* (1981) for the propagation of fox rabies and in Langlais & Suppo (1998) for generic models without vertical transmission.

(ii) *Mass action–logistic model*

In the mass action case, system (2) reads:

$$\begin{cases} \dfrac{\mathrm{d}X}{\mathrm{d}t} = b(X+\mathcal{Z}) - mX - rXP/K - \sigma_{\mathrm{ma}}X\mathcal{Y} - vX, \\ \dfrac{\mathrm{d}\mathcal{Y}}{\mathrm{d}t} = -m\mathcal{Y} - r\mathcal{Y}P/K + \pi\sigma_{\mathrm{ma}}X\mathcal{Y} - \alpha\mathcal{Y} - \mu\mathcal{Y}, \\ \dfrac{\mathrm{d}\mathcal{Z}}{\mathrm{d}t} = -m\mathcal{Z} - r\mathcal{Z}P/K + (1-\pi)\sigma_{\mathrm{ma}}X\mathcal{Y} + vX. \end{cases} \tag{6}$$

Two different dynamics may be observed. The trivial stationary state $X = \mathcal{Y} = \mathcal{Z} = 0$ is unstable, thus FeLV cannot yield a destruction of the cat population. The reproduction number is

$$R_0^{\mathrm{ma}}(\mu,v) = \frac{\sigma_{\mathrm{ma}}\pi K b}{(b+v)(b+\alpha+\mu)}.$$

Contrary to the previous case, the reproduction number depends on K. When $R_0^{\mathrm{ma}}(\mu,v) < 1$, the host population settles down to its disease-free equilibrium:

$$X = \frac{b}{b+v}K, \quad \mathcal{Y} = 0, \quad \mathcal{Z} = \frac{v}{b+v}K.$$

When $R_0^{\mathrm{ma}}(\mu,v) > 1$, the disease-free equilibrium is unstable. In the non-trivial case, the disease is maintained in the population and population size is reduced as for the previous case.

The reproduction number corresponding to the elimination programme is

$$R_0^{\mathrm{ma}}(\mu,0)\frac{\sigma_{\mathrm{ma}}\pi K}{b+\alpha+\mu}.$$

If $R_0^{\mathrm{ma}}(\mu,0) > 1$ and a vaccination programme is developed at rate v, vaccination will be efficient provided $R_0^{\mathrm{ma}}(\mu,v) < 1$, in which case the proportion of vaccinated cats is given by $\mathcal{Z}/P = v/(b+v)$. We rewrite $R_0^{\mathrm{ma}}(\mu,v)$ as

$$R_0^{\mathrm{ma}}(\mu,v) = \frac{b}{b+v}\times\frac{\sigma_{\mathrm{ma}}\pi K}{b+\alpha+\mu} = \left[1-\frac{v}{b+v}\right]\times R_0^{\mathrm{ma}}(\mu,0).$$

It follows that vaccination will be efficient provided the proportion of vaccinated cats satisfies the inequality

$$\frac{\mathcal{Z}}{P} > 1-\frac{1}{R_0^{\mathrm{ma}}(\mu,0)}. \tag{7}$$

This formula is identical to the relation derived in the previous case (equation (5)).

(iii) *Proportionate mixing–exponential model*

Now the dynamics are governed by

$$\begin{cases} \dfrac{\mathrm{d}X}{\mathrm{d}t} = b(X+\mathcal{Z}) - mX - \sigma_{\mathrm{pm}}X\Upsilon/P - vX, \\ \dfrac{\mathrm{d}\Upsilon}{\mathrm{d}t} = -m\Upsilon + \pi\sigma_{\mathrm{pm}}X\Upsilon/P - \alpha\Upsilon - \mu\Upsilon, \\ \dfrac{\mathrm{d}\mathcal{Z}}{\mathrm{d}t} = -m\mathcal{Z} + (1-\pi)\sigma_{\mathrm{pm}}X\Upsilon/P + vX. \end{cases} \tag{8}$$

The differential equation for the total population reads

$$\frac{\mathrm{d}P}{\mathrm{d}t} = rP - (b+\alpha+\mu)\Upsilon.$$

The threshold parameters are the same as in case (i). When $R_0^{\mathrm{pm}}(\mu,v) < 1$, the epidemic is eradicated and the cat population experiences a Malthusian growth as $t \to +\infty$:

$$P(t) \to +\infty,\ X(t) \to +\infty,\ \mathcal{Z}(t) \to +\infty, \text{ and } y(t) \to 0.$$

When $R_0^{\mathrm{pm}}(\mu,v) > 1$, FeLV develops with an asymptotic prevalence y^* and (i) when $R_1^{\mathrm{pm}}(\mu,v) < 1$, the cat population goes extinct; (ii) when $R_1^{\mathrm{pm}}(\mu,v) > 1$, the cat population experiences a Malthusian growth as $t \to +\infty$:

$$P(t)\to+\infty,\ X(t)\to+\infty,\ \Upsilon(t)\to+\infty,\ y(t)\to y^*,\ \mathcal{Z}(t)\to+\infty.$$

As a conclusion, FeLV cannot stabilize an exponentially increasing feline population where incidence follows a proportionate mixing law. Analysis of prophylaxis programmes would be similar to that described in case (i).

(iv) *Mass action–exponential model*

Now

$$\begin{cases} \dfrac{\mathrm{d}X}{\mathrm{d}t} = b(X+\mathcal{Z}) - mX - \sigma_{\mathrm{ma}}X\Upsilon - vX, \\ \dfrac{\mathrm{d}\Upsilon}{\mathrm{d}t} = -m\Upsilon + \pi\sigma_{\mathrm{ma}}X\Upsilon - \alpha\Upsilon - \mu\Upsilon, \\ \dfrac{\mathrm{d}\mathcal{Z}}{\mathrm{d}t} = -m\mathcal{Z} + (1-\pi)\sigma_{\mathrm{ma}}X\Upsilon + vX. \end{cases} \tag{9}$$

There are two dynamics possible. The only trivial stationary state $X = \Upsilon = \mathcal{Z} = 0$ is unstable, hence the cat population cannot go extinct under this model. There is a unique non-trivial stationary state:

$$\begin{aligned} X^* &= \frac{m+a+\mu}{\pi\sigma_{\mathrm{ma}}},\quad \Upsilon^* = \frac{r(m+v)}{\sigma_{\mathrm{ma}}[m-(1-\pi)b]}, \\ \mathcal{Z}^* &= \frac{(b+a+\mu)[\pi v + r(1-\pi)]}{\sigma_{\mathrm{ma}}\pi[m-(1-\pi)b]}. \end{aligned} \tag{10}$$

This state is admissible, i.e. $\Upsilon^* > 0$ and $\mathcal{Z}^* > 0$, if, and only if, $b(1-\pi) < m < b$, in which case it is also (locally) asymptotically stable.

This condition (previous paragraph) does not depend on the prophylaxis parameters v and μ. This means that any such prophylaxis programmes are ineffective when fertility is high. Hence, a preliminary sterilization programme may prove useful in eradicating FeLV within an exponentially increasing cat population where incidence follows a mass action law.

The following points are clear from equations (10).

1. An elimination programme alone at a rate μ will have no effect on the infectious class, but will increase both susceptible and immune classes. Thus, the total population of cats will increase, while the prevalence

$$\frac{\Upsilon^*}{X^*+\Upsilon^*+\mathcal{Z}^*} = \frac{r}{b+\alpha+\mu}$$

will decrease.
2. A vaccination programme alone at a rate v will increase both infectious and immune classes, but will have no effect on the susceptible class. The prevalence will be unaffected, while the total population of cats will increase.
3. A joint programme involving both vaccination and elimination will increase the total population at level

$$\frac{(m+v)(b+a+\mu)}{\sigma_{\mathrm{ma}}[m-(1-\pi)b]},$$

with a decreased prevalence owing to elimination alone.

(b) *Application to biological data*

Several previous studies have provided estimates of model parameters (Fromont *et al.* 1997). Demographic parameters were derived from the long-term monitoring of cat populations (Pontier *et al.* 1995). From this survey, b and m were estimated to be 0.88 females female^{-1} yr^{-1} and 0.51 per year, respectively (Fromont *et al.* 1997). Several estimates of α and π were available: the specific mortality rate α was taken as 0.76 per year (Weijer & Daams 1976; Hardy 1980; Ishida *et al.* 1981) and the proportion of cats that develop viraemia after infection π was estimated to be 0.33 (Hardy *et al.* 1976). Direct estimates of incidence σ are not available in the field, but for R_0 to be greater than 1, definitions of R_0 imply $\sigma_{\mathrm{pm}} > 4.97$ and $K\sigma_{\mathrm{ma}} > 4.97$. We did simulations with $\sigma_{\mathrm{pm}} = K\sigma_{\mathrm{ma}} = 6$ (figure 2).

Relevance of the proportionate mixing–logistic model (case (i), figure 2*a*,*b*) has been studied in Fromont *et al.* (1997). That model accounted for large, stable populations of cats. In two large rural populations (200 and 300 cats),

we previously observed persistence of infection over several years. Observed prevalences ($4.84 \pm 1.93\%$ and $11.11 \pm 3.02\%$) were in the range of predicted values using extreme estimates for parameter values, and we did not detect significant differences among annual prevalences.

In a third rural population (Barisey-la-Côte), however, no viraemic cat was ever detected during seven consecutive years, although opportunities for virus introduction were probably frequent. Barisey-la-Côte is a small population (around 50 cats) and the village is distant from adjacent populations. In a detailed study of FeLV risk, we hypothesized that small population size may be responsible for too few contacts among cats to maintain persistent infection (Fromont *et al.* 1998). This is the situation modelled here in the mass action–logistic model (case (ii)), and simulations with realistic parameter values suggest that viral extinction occurs under a critical community size of around 100 cats (figure 2*c*,*d*). This case may also account for another situation, small urban groups of feral cats (unless population size is not yet stable, see below). Several recent studies on FeLV prevalence in urban populations suggest that FeLV is not endemic. A high spatial and temporal variability was found: several populations were free of FeLV, whereas prevalence reached exceptionally high levels in others (up to 70%: Fromont *et al.* 1996; Yamaguchi & Macdonald 1996; Xémar 1997). Demography of the cat populations studied was not precisely assessed, however.

The proportionate mixing–exponential model (case (iii)) accounts for populations growing in non-anthropized habitats. It is noteworthy that host population size cannot be stabilized under the effect of disease, and extermination of the population only occurs when the intrinsic growth rate is extremely low (simulations show that r must be less than 0.05, figure 2*f*). Estimates of r in subantarctic populations were found to be much higher, either on the Kerguelen archipelago (as high as $r = 0.51$, Wetzel *et al.* 1982), or on Marion Island ($r = 0.21$, Van Rensburg *et al.* 1987) even 30 years after the release of founder cats. Thus, the most biologically realistic case is exponential growth of host populations with asymptotic FeLV prevalence, y^* (figure 2*e*). Few field data are available to assess the prediction of this model. In a preliminary study done at the Kerguelen archipelago, none of 54 feral cats sampled was viraemic, suggesting that FeLV is either absent or present at low prevalence levels (M. Artois, E. Fromont & D. Pontier, unpublished data).

Finally, the mass action–exponential model (case (iv)) would be the most appropriate for urban groups of stray cats while they are establishing: mass action is preferred because home ranges are overlapping and contact rate increases with population size. Exponential growth accounts for the rapid increase in population size at the beginning of population growth. Biologically realistic values for parameters lead to no stable state (figure 2*g*). The model predicts that a birth rate lower than that observed ($b = 0.88$) would lead to stabilization of population size under the effect of disease (figure 2*h*). Stabilization would only occur after a long period, however, probably longer than the period of exponential growth. This model also shows that prophylaxis would produce paradoxical effects in terms of the number of infectious and immune versus susceptible cats, and be ineffective for disease control.

4. DISCUSSION

The four models predict different dynamical outcomes following virus introduction to a cat population: stable virus persistence for case (i); stable virus persistence if population size is greater than a certain threshold in case (ii); exponential growth of infected population in case (iii); and no stationary state in case (iv). These predictions correctly account for data presently available on FeLV dynamics. We argue that a single model is insufficient to describe population dynamics of FeLV in cats, and, more generally, to describe specific host–parasite relations where the dynamics and structure of the host species vary. This study confirms that the impact of a parasite may vary widely according to host population characteristics (Grenfell & Dobson 1995), and stresses the need to do detailed field studies on host–parasite interactions before building epidemic models.

Our models help to define what needs to be measured in field studies. First, population growth must be assessed accurately. Even in a long-term follow-up, logistic growth may be confounded by exponential growth with a low value for r. The evolution of contact rate as a function of density is also disputable. For example, mass action models make the assumption that contact rate decreases with population density. Contact rates should be assessed in the field by gathering behavioural data on encounter rates among cats in dense and sparse populations. It is also probable that the increase in contact rate with density is shaped by individual behaviour. In cats, besides the change of home range size with density, it is thought that hierarchical dominance relations establish in dense social groups. The hierarchy allows reduction in the rate of direct aggressive encounters (E. Natoli, H. A. Baggio and D. Pontier, unpublished data). Whether or not this behaviour is a selective response to disease, a hierarchy acts against linear increase of contact rate with density. However, a more detailed function than that in figure 1 may be appropriate to describe the evolution of contact rate with density.

As for disease control, cases (i) and (ii) give classical results that must be assessed considering the costs and benefits of elimination and vaccination (Fromont *et al.* 1997). Among the costs is the lack of efficacy of FeLV vaccines: the proportion of cats to be vaccinated must be corrected using the efficacy of the vaccine used. An interesting result of model (iii) is that extermination or stabilization of the host population size under the effect of the virus are biologically unlikely to occur. Thus, FeLV introduction is probably not an efficient strategy for control of feral populations, unlike in other host populations and other diseases (Van Rensburg *et al.* 1987). Some predictions concerning prophylaxis can be tested in the field, for example whether the predicted threshold proportion of vaccinated cats is sufficient to maintain a population free of disease.

Although much more refined options exist (Anderson 1995), we consider only basic disease models here, because our simple assumptions were sufficient to model available data. An alternative explanation for the absence of FeLV in some small cat populations could be to consider a stochastic process of extinction. We need more field data on this point before further analysis can be done. Another

possibility is the impact of genetic structure of the host population on parasite dynamics (Gulland *et al.* 1993). We have determined that domestic cats carrying the orange coat colour allele tend to be infected by FeLV less often than other cats (Pontier *et al.* 1998). As the orange allele occurs at different frequencies between rural and urban areas (Pontier *et al.* 1995), the proportion of orange cats may explain part of the difference between rural and urban FeLV dynamics. Likewise, age and sex have been found to be significant risk factors of FeLV infection in cats (Fromont *et al.* 1998), so that heterogeneity in population structure may act on parasite dynamics. Also, we incorporated some spatial structure when modelling FeLV incidence. However, spatial heterogeneity may exist at other scales. In conclusion, more complex models cannot be constructed before more field data are obtained.

We thank J. M. Gaillard, W. G. King and N. G. Yoccoz for their helpful comments. This work was supported by the CNRS under the grant 'Modélisation de la circulation de parasites dans des populations structurées'. Field studies on the Kerguelen archipelago are part of a programme of the French Polar Institute IFRTP.

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