MATHEMATICAL MODELLING OF
POPULATION DYNAMICS
BANACH CENTER PUBLICATIONS, VOLUME 63
INSTITUTE OF MATHEMATICS
POLISH ACADEMY OF SCIENCES
WARSZAWA 2004

# AN AGE-DEPENDENT MODEL DESCRIBING THE SPREAD OF PANLEUCOPENIA VIRUS WITHIN FELINE POPULATIONS

W. E. FITZGIBBON

*College of Technology, University of Houston, Houston, Texas 77204-4021, U.S.A.*
*E-mail: fitz@uh.edu*

M. LANGLAIS

*UMR CNRS 5466, case 26, V. Segalen Bordeaux 2 University, 33076 Bordeaux Cedex, France*
*E-mail: langlais@sm.u-bordeaux2.fr*

J. J. MORGAN

*Department of Mathematics, Texas A & M University*
*College Station, Texas 77843-3368, U.S.A.*
*E-mail: jmorgan@math.tamu.edu*

D. PONTIER

*UMR CNRS 5558, C. Bernard Lyon 1 University, 69622 Villeurbanne Cedex, France*
*E-mail: dpontier@biomserv.univ-lyon1.fr*

C. WOLF

*UMR CNRS 5466, case 26, V. Segalen Bordeaux 2 University, 33076 Bordeaux Cedex, France*
*E-mail: wolf@sm.u-bordeaux2.fr*

**Abstract.** Global existence results and long time behavior are provided for a mathematical model describing the propagation of Feline Panleucopenia Virus (FPLV) within a domestic cat population; two transmission modes are involved: a direct one from infective cats to susceptible ones, and an indirect one from the contaminated environment to susceptible cats. A more severe impact of the virus on young cats requires an age-structured model.

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2000 *Mathematics Subject Classification*: Primary 92D30; Secondary 35L45, 35B40, 35B45.

This work is partially supported by a joint N.S.F.-C.N.R.S. grant DMS 0089590, "Heterogeneous Reaction Diffusion Advection Systems" and by the French Polar Institute (IPEV, Program n$^o$279).

The paper is in final form and no version of it will be published elsewhere.

**1. Introduction.** We shall be concerned by models describing the spread of infectious disease through distributed populations. In recent times there has been much activity concerning the development and analysis of mathematical models describing the transmission of infectious disease, cf Busenberg and Cooke [5], Brauer and Castillo-Chavez [4], Diekmann and Heesterbeek [7] and the references contained therein. However, the vast majority of this work concerns the direct transmission of disease (horizontal, vertical, or both) among populations. In the work at hand we account both for the direct horizontal spread of the disease from infected individuals to susceptible individuals within the population and the indirect spread of the disease to the host population via a contact with a contaminated environment. A prime example of such a disease is Feline Panleucopenia Virus FPLV. FPLV, a member of the parvoviridae family, infects all Felidae and some other carnivore species as well. One of the distinguishing features of the disease is its ability to survive outside the host for a long period of time. Indeed studies show that it remains infectious outside of the host for over a year. Clinically the virus is described as highly contagious with severe gastroenteritis with fever, anorexia, vomiting, diarrhoea and marked leucopenia leading to the death of the animal, cf Berthier *et al.* [1]. The incubation is short (one to four days) and the infection lasts approximately two weeks. Infected cats are viremic for a few days and can excrete the virus in there feces, vomit, urine, saliva, and nasal and ocular discharges. The mortality is quite high for kittens (80%) and lower but significant for adults (20%). There is no sexual differentiation in receptivity. Recovered cats do not become excreting carriers and develop immunity lasting at least six years (effectively lifelong). Transmission of the disease to susceptible cats from infected cats occurs via normal social contact or by mechanical transmission with an infected environment (primarily feces). Vertical transmission between parents and offspring does not appear to be a factor.

FLPV was recently involved in a recent experiment in animal control. The virus was introduced on Marion Island with the aim of eradicating or drastically reducing a population of feral cats that was having a devastating impact on the indigenous avian population. Marion is an island of 290 $m^2$ located in the sub-Antartic area of the Southern Indian Ocean. In the absence of natural predators, five cats introduced gave rise to population estimated to be 3400 in 1977. In 1997, 96 cats were trapped and inoculated with FLPV and released into the population [2]. Six years later (in 1982) the population had dropped to an estimated 615 with a corresponding annual rate of decline of 29%. Subsequent to 1982, the rate of decline slowed to 8%. In 1991, the remaining cats were eliminated with an intensive culling effort [3].

**2. A simple model.** Once the virus has been introduced our total population $P$ can be subdivided into three classes $S$, $I$, and $R$, with $P = S + I + R$, representing the sub-populations of susceptible, infective, and removed individuals. Susceptibles are individuals who have not contracted but are capable of contracting the disease. Infectives are individuals fully infected and capable of transmitting the disease. The removed class consists of individuals who have recovered from the disease thereby gaining immunity. As such they affect the population dynamics but are not involved in the transmission of the disease.

We model the horizontal transmission of the disease from infectives to susceptible individuals by an incidence term. Two forms of incidence terms are standard, although their choice is somewhat problematic, [5], [6], [11]. The first type is a so called mass action term of the form

$$\sigma(S, I, R) = \sigma SI, \tag{1}$$

with $\sigma > 0$ a constant. The second choice is a proportionate mixing term

$$\sigma(S, I, R) = \sigma_{pm} SI/P = \sigma_{pm} SI/(S + I + R) \tag{2}$$

with $\sigma > 0$. The analysis in [1] shows that an appropriate choice for FPLV direct transmission in the Marion Island environment is a mass action term; we choose the form of the incidence term given by (1).

The final state variable, $C$, represents the proportion of the habitat that is infected. In the case of FPLV the following equation is derived in [1]:

$$dC/dt = \phi\,(1 - C)\,I - \delta C. \tag{3}$$

Here $\phi > 0$ is a scaling constant and $\delta > 0$ is the decontamination rate of the environment. Because $C$ is a proportion we can assume that $0 \leq C(0) \leq 1$. Given this assumption, one can readily employ the comparison principle to observe that $0 \leq C\,(t) \leq 1$ for all $t > 0$. Finally we let $\rho > 0$ denote the rate of indirect transmission of the disease from the environment to susceptible individuals. This produces a loss term for the susceptible class of the form $\rho CS$. Along the lines of [1], we employ Malthusian dynamics, corresponding to the actual demography of the feral cat population before introduction of FPLV on Marion Island. In the absence of disease we assume that the population satisfies the equation

$$dP/dt = bP - mP \tag{4}$$

This incorporates a natural birth rate, $b$, and a natural mortality rate, $m$. For present purposes, we assume no vertical disease transmission with a natural birth of susceptible individuals from all three classes, reduced by a fraction $\theta$, $0 \leq \theta \leq 1$, in the infective class. This assumption is consistent with FPLV, cf. [1] where $\theta = 1$; here, a $0 \leq \theta < 1$ takes into account early death of infective mothers caused by FPLV. We let $\alpha > 0$ be the inverse of the length of the infectious period and $\varepsilon$, $0 < \varepsilon < 1$, be the proportion of individuals surviving the infection. This yields a disease induced mortality rate of $\alpha\,(1 - \varepsilon)$. The foregoing considerations produce the following four-component system of ordinary differential equations:

$$\begin{aligned} dS/dt &= b(S + \theta I + R) - mS - \sigma SI - \rho CS, \\ dI/dt &= -(m + \alpha)I + \sigma SI + \rho CS, \\ dR/dt &= -mR + \varepsilon\alpha I, \\ dC/dt &= \phi\,(1 - C)\,I - \delta C, \end{aligned} \tag{5}$$

with initial conditions

$$S(0) \geq 0,\ I(0) \geq 0,\ R(0) \geq 0,\ P(0) > 0;\ \ 0 \leq C(0) \leq 1. \tag{6}$$

We readily obtain the following result:

PROPOSITION 1. *System (5)-(6) admits a globally defined solution on* $[0,\infty)$. *Each of the solution components remains nonnegative,* $0 \le C(t) \le 1$, *for all* $t > 0$.

*Proof.* Local existence of solutions having nonnegative components is guaranteed by Picard Theorem and the fact the vector field defined by the righthand side of the system of differential equations (5) points outwards from the positive cone of $\Re^4$. Then, from the nonnegativity of $I$ it follows that the interval $[0,1]$ is forward invariant by (3). If we add the first three components and set $P = S + I + R$ we obtain

$$dP/dt = bP - mP - [(1-\varepsilon)\alpha + (1-\theta)b]I \le (b-m)P; \tag{7}$$

we obtain a uniform exponential growth for $P(t)$ and we conclude our proof. ∎

Questions concerning the longtime behavior are considerably more difficult. Based on numerical simulations, some partial results are outlined in [1]. More precisely, when $b > m$ only two dynamics are numerically observed: either a regulation of the host population by FPLV, or no impact of the virus on the malthusian growth. The goal of the remaining part of this section is to supply analytical evidences supporting these numerical observations. From (7) one gets that $b-m<0$ implies $P(t) \to 0$ as $t \to \infty$; hence one may now assume

$$b - m > 0. \tag{8}$$

PROPOSITION 2. *Assume (6) and (8) hold. Then,*

**(i)** *when* $m(\alpha+m) < b(\varepsilon\alpha + \theta m)$ *one has* $P(t) \to +\infty$ *as* $t \to \infty$, *exponentially;*
**(ii)** *when* $m(\alpha+m) > b(\varepsilon\alpha+\theta m)$ *there exists a unique stationary state* $(S^\sharp, I^\sharp, R^\sharp, C^\sharp)$ *with positive components and* $0 < C^\sharp < 1$.

*Proof.* We first look at case **(i)**. Let $\eta$ and $\varpi$ be small positive numbers. A linear combination of the first three equations in (5) yields

$$\begin{aligned}&((m-\eta)S + mI + (b-\varpi)R)' \ge \\ &\quad (m-\eta)(b-m)S + [b(\varepsilon\alpha+\theta m) - m(\alpha+m) - (\varepsilon\alpha\varpi + b\theta\eta)]\, I + (m\varpi - b\eta)R;\end{aligned}$$

choosing $\eta = m\varpi/2b$, and next

$$\varpi < \varpi_0 = \max\left(\frac{b(\varepsilon\alpha+\theta m) - m(\alpha+m)}{\theta m + 2\varepsilon\alpha}, b\right),$$

one gets $m-\eta>0$, $b-\varpi>0$ and finds a $\lambda = \lambda(\varpi) > 0$ such that

$$[(m-\eta)S + mI + (b-\varpi)R]' \ge \lambda\,[(m-\eta)S + mI + (b-\varpi)R].$$

As a consequence, $P(t)$ experiences an exponential growth as $t \to \infty$.

Next, we look at case **(ii)**. Let $(S^\sharp, I^\sharp, R^\sharp, C^\sharp)$ be a stationary state for (5) with nonnegative components. From the equation for $C$ one gets $C^\sharp = \frac{\phi I^\sharp}{\delta + \phi I^\sharp}$, and from the equation for $R$ it follows that $R^\sharp = \frac{\varepsilon\alpha}{m} I^\sharp$. Looking at (7) one also has

$$P^\sharp = \frac{(1-\varepsilon)\alpha + (1-\theta)b}{b-m} I^\sharp,$$

where $P^\sharp = S^\sharp + I^\sharp + R^\sharp$. One may now compute $S^\sharp$ as a function of $I^\sharp$, and find

$$S^\sharp = P^\sharp - I^\sharp - R^\sharp = \frac{m(\alpha+m) - b(\varepsilon\alpha+\theta m)}{m(b-m)} I^\sharp.$$

Substituting these relations into the equation for $S^\sharp$ in (5), after some algebra one gets that $I^\sharp$ is a solution to a nonlinear equation, namely $f(I^\sharp) = T^\sharp$ with

$$f(I^\sharp) = m + \sigma I^\sharp + \rho \frac{\phi I^\sharp}{\delta + \phi I^\sharp}, \;\; T^\sharp = \frac{(1-\varepsilon)\alpha + (1-\theta)m}{m(\alpha + \theta m) - b(\varepsilon\alpha + m)} bm.$$

Now, $f$ is increasing over $[0, \infty)$, $f(0) < T^\sharp$ and $f(I^\sharp) \to +\infty$ as $I^\sharp \to +\infty$. This completes the proof of **(ii)**. ∎

REMARK 1. We point out that one could obtain analogous results in the case of proportionate mixing. Recall in this case we have an incidence of the form (2).

**3. Age dependent models.** The system of ordinary differential equations model of the previous section omits many pertinent features. For example in the introduction we indicated that disease induced mortality rate for kittens was much greater than that of adults, approximately four times as much. We should also expect that the natural mortality rate and the birth rate would be highly dependent upon the age of a given cat. These comments argue for the inclusion of another independent variable, $a$. In this case we represent the time dependent age density individuals by the dependent variable $p(a,t) \geq 0$. The total population at time $t$ is given by

$$P(t) = \int_0^\infty p(a,t)da, \;\; t \geq 0.$$

From a practical standpoint one can assume the existence of an $A_\dagger$ so that $p(a,t) \equiv 0$ for $a > A_\dagger$. However, theoretically we find it convenient to let $A_\dagger = +\infty$ and assume an infinite age interval of the form $[0, +\infty)$.

The birth rate is given by $b(a) \geq 0$ for $a \geq 0$ and natural mortality is given by an expression $m(a) \geq 0$ for $a \geq 0$. The time evolution of $p(a,t)$ is governed by the celebrated Sharpe, Lotka and McKendrick equation, [13], [14], [15], [18],

$$\partial p/\partial t + \partial p/\partial a = -m(a)p, \;\; a \geq 0, \;\; t \geq 0, \tag{9}$$

subject to the age boundary condition

$$p(0,t) = \int_0^\infty b(a)p(a,t)da, \;\; t \geq 0, \tag{10}$$

and the initial condition

$$p(a,0) = p_0(a), \;\; t \geq 0. \tag{11}$$

To put things in perspective, we can assume our birth rate and natural mortality term do not depend upon $a$, make the assumption $P(\infty) = 0$, and obtain equation (4) by integrating (9)-(10)-(11) with respect to the variable $a$ on $[0, +\infty)$.

The longtime dynamics of solutions to (9), (10) and (11) is well-known since the work of [8], see [15], [18]. Set

$$\begin{aligned} &\pi(a) = \exp\left( -\int_0^a m(a')\, da' \right), \;\; a > 0; \\ &a_\dagger \;\; = \max(\, \mathrm{supp}(b)). \end{aligned} \tag{12}$$

Then,

$$
\begin{aligned}
&\text{if either } \operatorname{supp}(p_0) \subset [a_\dagger, +\infty) \text{ or } \int_0^\infty b(a)\pi(a)\,da < 1,\\
&\qquad \text{then } p(\cdot,t) \to 0 \text{ as } t \to +\infty, \text{ uniformly on } [0,A] \text{ for each } A > 0;\\
&\text{if both } \operatorname{supp}(p_0) \subset [0, a_\dagger] \text{ and } \int_0^\infty b(a)\pi(a)\,da > 1,\\
&\qquad \text{then } p(\cdot,t) \to +\infty \text{ as } t \to +\infty, \text{ uniformly on } [0,A] \text{ for each } A > 0.
\end{aligned} \tag{13}
$$

An age dependent model describing the circulation of FPLV will define time dependent age densities $s(a,t)$, $i(a,t)$, $r(a,t)$ as state variables. As before, $C(t)$ denotes the proportion of infected habitat. We can compute the time dependent susceptible, infective and removed classes by integrating on $[0,+\infty)$ with respect to $a$, i.e. we have

$$S(t) = \int_0^\infty s(a,t)da,\ I(t) = \int_0^\infty i(a,t)da,\ R(t) = \int_0^\infty r(a,t)da.$$

The total population is given by $P(t) = S(t) + I(t) + R(t)$.

We introduce an age dependent incidence term $(\sigma(a)I(t))\,s$ and an age dependent environmental incidence term $(\rho(a)C(t))\,s$. In order to differentiate between the recovery rates of adults and juveniles we introduce an age dependent recovery rate $\varepsilon(a)$, $0 \le \varepsilon(a) \le 1$; $\alpha > 0$ is still the inverse of the length of the infectious period. We should expect a lowered birth rate from the class of infectives. The function $\theta(a)$ will represent the fractional lowering of the birth rate from infective class. We will have $0 \le \theta(a) \le 1$, for $a \ge 0$.

We make the following assumptions on coefficient functions and initial data:

**(A1)** the functions $b, \theta, m, \sigma, \rho, \varepsilon$ are nonnegative, and belong to $L_\infty(\Re^+) \cap C(\Re^+)$; also $0 \le \theta(a) \le 1$, $0 \le \varepsilon(a) \le 1$.

**(A2)** the initial functions $s_0, i_0, r_0$ are nonnegative, and belong to $L_\infty(\Re^+) \cap C^1(\Re^+)$; $0 \le C_0 \le 1$.

The foregoing conditions yield the following coupled semilinear system of differential equations:

$$
\begin{cases}
\partial s/\partial t + \partial s/\partial a = -m(a)s - \sigma(a)I(t)\,s - \rho(a)Cs,\\
\partial i/\partial t + \partial i/\partial a = -\left[m(a) + \alpha\right] i + \sigma(a)I(t)\,s + \rho(a)Cs,\\
\partial r/\partial t + \partial r/\partial a = -m(a)r + \alpha\varepsilon(a)i,\\
\partial C/\partial t = \phi(1-C)I(t) - \delta C,
\end{cases} \tag{14}
$$

subject to boundary conditions

$$
\begin{cases}
s(0,t) = \displaystyle\int_0^{A_\dagger} \left(b(a)s(a,t) + \theta(a)b(a)i(a,t) + b(a)r(a,t)\right) da,\\
i(0,t) = r(0,t) = 0,
\end{cases} \tag{15}
$$

and initial conditions

$$
\begin{cases}
s(a,0) = s_0(a) \ge 0,\ i(a,0) = i_0(a) \ge 0,\ r(a,0) = r_0(a) \ge 0,\\
0 \le C(0) = C_0 \le 1.
\end{cases} \tag{16}
$$

We point out that the assumption of no hereditary transmission of the disease is incorporated in the age boundary condition (15). Moreover if we assume the functions

$b, \theta, m, \sigma, \rho, \alpha, \varepsilon$ are positive constants then (14)-(15)-(16) may be formally integrated for age $a \in [0, \infty)$ to produce a system of ordinary differential equations of the form (5).

We specify the birth function

$$B(t) = \int_0^{A_\dagger} [b(a)s(a,t) + \theta(a)b(a)i(a,t) + b(a)r(a,t)]\, da,$$

and introduce functional notations

$$\begin{aligned}
&H(a,t) = m(a) + \sigma(a)I(t) + \rho(a)C(t),\\
&K_1(a,t) = m(a) + \alpha,\\
&K_2(a,t) = m(a),\\
&F(a,t) = \sigma(a)I(t)s(a,t) + \rho(a)C(t)s(a,t),\\
&J(a,t) = \alpha\varepsilon(a)i(a,t),\\
&L(a,t) = \phi I(t) + \delta.
\end{aligned}$$

If we adapt the characteristic methods outlined in [18], [15], [9] we may observe that solutions to (14)-(15)-(16) have integrated forms

$$s(a,t) = \begin{cases} s_0(a-t)\exp\left(-\displaystyle\int_0^t H(a-t+s,s)\, ds\right), & \text{for } a > t,\\ B(t-a)\exp\left(-\displaystyle\int_0^a H(s,t-a+s)\, ds\right), & \text{for } t > a, \end{cases} \tag{17}$$

$$i(a,t) = \begin{cases} i_0(a-t)\exp\left(-\displaystyle\int_0^t K_1(a-t+s,s)\, ds\right) + \\ \quad \displaystyle\int_0^t \exp\left(-\int_s^t K_1(a-t+u,u)\, du\right) F(a-t+s)\, ds, & \text{for } a > t,\\ 0, & \text{for } t > a, \end{cases} \tag{18}$$

$$r(a,t) = \begin{cases} r_0(a-t)\exp\left(-\displaystyle\int_0^t K_2(a-t+s,s)\, ds\right) + \\ \quad \displaystyle\int_0^t \exp\left(-\int_s^t K_2(a-t+u,u)\, du\right) J(a-t+s)\, ds, & \text{for } a > t,\\ 0, & \text{for } t > a, \end{cases} \tag{19}$$

and

$$C(t) = C_0 \exp\left(-\int_0^t L(s)\, ds\right) + \phi \int_0^t \exp\left(-\int_s^t L(u)\, du\right) I(s)\, ds, \text{ for } t > 0. \tag{20}$$

A quadruple of functions $(s(a,t), i(a,t), r(a,t), C(t))$ is said to be a mild solution to (14)-(15)-(16) if it satisfies the system of integral equations (17)-(18)-(19)-(20). We have the following result:

THEOREM 3. *If conditions **(A1)** and **(A2)** are satisfied, then there exists a unique non-negative quadruple of functions providing a mild solution to (14)-(15)-(16). These functions are continuous except possibly along the line $t = a$, continuously differentiable for $a > t$ and $a < t$, and satisfy the differential equations and the boundary conditions.*

*Proof.* The arguments establishing this result are quite lengthy, involving a concatenation of semigroup theory and fixed point arguments. Indeed over a chapter of [18] is devoted to the case of a scalar Gurtin and McCamy equation. For this reason we shall not include a

proof of this result but we shall briefly overview arguments establishing solution positivity and a priori bounds; see [19] for details on a related problem.

Arguments appearing in [16] develop comparison principles for age dependent operators of the form $\partial\psi/\partial t + \partial\psi/\partial a$ and the comparison principle in turn allows us to establish a theory of invariant rectangles for age transport systems which parallels the theory of invariant rectangles in [17] for reaction diffusion systems. With this in mind we can imagine the vector field defined by the right hand side of (14)-(15)-(16) in $\Re^4_+$, $(s, i, r, C)$ state space. We may readily observe that this vector field does not point out of the positive cone $\Re^4_+$ and conclude that if the initial data is nonnegative the functions remain nonnegative.

From the nonnegativity of $i(a,t)$ and $I(t)$ one may conclude that $0 \leq C(t) \leq 1$ provided $0 \leq C_0 \leq 1$.

If we add the differential equations for $s$, $i$ and $r$, and set $p = s+i+r$ we may observe that $p(a,t)$ satisfies a differential inequality of the form

$$\begin{aligned} &\partial p/\partial t + \partial p/\partial a \leq -m(a)p,\ t>0,\ a>0,\\ &p(a,0) = p_0(a) = s_0(a) + i_0(a) + r_0(a),\ a>0,\\ &p(0,t) \leq \textstyle\int_0^\infty b(a)p(a,t)\,da,\ t>0. \end{aligned} \tag{21}$$

If we integrate (21) on $[0,+\infty)$ with respect to $a$ we have for $P(t) = \|p(\cdot,t)\|_{1,[0,\infty)}$

$$P'(t) \leq (b_{\max} - m_{\min})P(t),\ t>0;\ P(0) = \int_0^\infty p_0(a)\,da,$$

wherein $b_{\max} = \max(b(a), a>0)$ and $m_{\min} = \min(m(a), a>0)$. We have an exponential growth estimate for $P(t)$. Going back to (21) one gets an exponential growth estimate for $\|p(\cdot,t)\|_{\infty,[0,\infty)}$; this together with nonnegativity produces uniform exponential growth estimates for $\|s(\cdot,t)\|_{\infty,[0,\infty)}$, $\|i(\cdot,t)\|_{\infty,[0,\infty)}$, $\|r(\cdot,t)\|_{\infty,[0,\infty)}$ and $I(t)$. ∎

We have obtained some preliminary results concerning the longtime behavior of solutions to (14)-(15)-(16). From the set of inequalities in (21) and a comparison principle, it follows that when

$$\text{either } \int_0^\infty b(a)\pi(a)\,da < 1,\ \text{or}\ \ \operatorname{supp}(p_0) \subset [a_\dagger, +\infty)$$

then, $p(\cdot,t) \to 0$ as $t \to +\infty$, uniformly on $[0,A]$ for $A>0$; see (13), [18], [15]. Hence, one may now assume

$$\int_0^\infty b(a)\pi(a)\,da > 1. \tag{22}$$

Set

$$\mathcal{T}_0(\varepsilon,\theta) = \int_0^\infty b(a)\pi(a)\left[e^{-\alpha a}\theta(a) + \alpha\int_0^a \varepsilon(a')e^{-\alpha a'}\,da'\right]da. \tag{23}$$

THEOREM 4. *Assume (22) hold. Then,*

**(iii)** *when $\mathcal{T}_0(\varepsilon,\theta) < 1$, then there exists an age dependent stationary state with positive components, $(s^\sharp(a), i^\sharp(a), r^\sharp(a), C^\sharp)$, and $0 < C^\sharp < 1$.*

*Proof.* Along the lines of the proof of Proposition 2, the idea is to compute stationary states. First, one still has

$$C^\sharp = \frac{\phi I^\sharp}{\delta + \phi I^\sharp}, \quad I^\sharp = \int_0^\infty i^\sharp(a)\, da.$$

Second, at equilibrium, one gets

$$d_a s^\sharp + m(a)s^\sharp = -\varphi_1(a, I^\sharp)s^\sharp, \quad \varphi_1(a, I^\sharp) = \sigma(a)I^\sharp + \rho(a)\frac{\phi I^\sharp}{\delta + \phi I^\sharp},$$

so that for some positive initial condition $s^\sharp(0)$

$$s^\sharp(a) = s^\sharp(0)\pi(a)\Phi_1(a, I^\sharp), \ \Phi_1(a, I^\sharp) = \exp\left(-\int_0^a \varphi_1(a', I^\sharp)da'\right). \tag{24}$$

The equation for $i^\sharp$ reads $d_a i^\sharp + m(a)i^\sharp = -\alpha i^\sharp + \varphi_1(a, I^\sharp)s^\sharp$; therefore,

$$i^\sharp(a) = s^\sharp(0)\pi(a)e^{-\alpha a}\Phi_2(a, I^\sharp), \quad \Phi_2(a, I^\sharp) = \int_0^a e^{\alpha a'}\varphi_1(a', I^\sharp)\Phi_1(a', I^\sharp)\, da', \tag{25}$$

because $i^\sharp(0) = 0$. The equation for $r^\sharp$ reads $d_a r^\sharp + m(a)r^\sharp = \alpha\varepsilon(a)i^\sharp$; therefore,

$$r^\sharp(a) = \alpha s^\sharp(0)\pi(a)\Phi_3(a, I^\sharp), \quad \Phi_3(a, I^\sharp) = \int_0^a e^{-\alpha a'}\varepsilon(a)\Phi_2(a', I^\sharp)\, da', \tag{26}$$

because $r^\sharp(0) = 0$. At age $a = 0$ one must have

$$s^\sharp(0) = \int_0^\infty b(a)\left(s^\sharp(a) + \theta(a)i^\sharp(a) + r^\sharp(a)\right)\, da.$$

Using (24), (25) and (26), and simplifying by $s^\sharp(0)$ yields a nonlinear equation for $I^\sharp$

$$F(I^\sharp) \equiv \int_0^\infty b(a)\pi(a)\left[\Phi_1(a, I^\sharp) + e^{-\alpha a}\theta(a)\Phi_2(a, I^\sharp) + \alpha\Phi_3(a, I^\sharp)\right] = 1. \tag{27}$$

Now, one may check that $F(0) > 1$ by (22), and $F(I^\sharp) \to \mathcal{T}_0(\varepsilon, \theta)$ as $I^\sharp \to +\infty$. Hence there is at least a positive $I^\sharp$, a solution of $F(I^\sharp) = 1$ if $\mathcal{T}_0(\varepsilon, \theta) < 1$; then $s^\sharp(0)$ is evaluated from (25) upon integrating with respect to age to get

$$I^\sharp = s^\sharp(0)\int_0^\infty \pi(a)e^{-\alpha a}\Phi_2(a, I^\sharp)\, da.$$

Remark 2. For constant $b$, $m$, $\varepsilon$ and $\theta(a) \equiv 1$, a direct computation of $\mathcal{T}_0(\varepsilon, \theta)$ shows that (**ii**) and (**iii**) are equivalent.

**4. Conclusion.** The unstructured model, (5), with a mass action type incidence was shown to be a more appropriate choice than the analogous model with a proportionate mixing incidence to understand a recent experiment in animal control on Marion Island; see [1]. This was established through numerical simulations using field data. Our results in the present work are the continuation of [1].

From a dynamical point of view numerical simulations of (5) were also showing that as soon as a stationary state with positive entries was existing then it was numerically stable, else the host population was recovering an exponential growth. Although we do not prove any local stability result for the stationary state, Proposition 2 supports these observations upon supplying a threshold parameter yielding the existence of a unique stationary state with positive entries, indicating whether the virus can control (case **(ii)**) or not control (case **(i)**) the exponential growth.

Numerical simulations also showed more complex dynamics for the proportionate mixing incidence model (see (2)): extinction of the host population for large $\sigma_{pm}$, an exponential growth for small $\sigma_{pm}$ and $\rho$, and a unique stationary state with positive entries at intermediate values for $\sigma_{pm}$ and $\rho$; see [1]. Obviously the first part of Proposition 2 (case **(i)**) still holds for a proportionate mixing incidence; but this is not the case anymore for the second part (case **(ii)**). Reworking the proof of Proposition 2, **(ii)**, when $m(m+\alpha) > b(\theta m + \varepsilon\alpha)$ a secondary bifurcation parameter must be introduced, namely

$$\mathcal{S}_{pm} = \frac{m(m+\alpha)}{m(m+\alpha) - b(\theta m + \varepsilon\alpha)};$$

there exists a unique stationary state if and only if $\frac{\sigma_{pm}}{(1-\varepsilon)\alpha} < \mathcal{S}_{pm} < \frac{\sigma_{pm}}{(1-\varepsilon)\alpha} + \frac{\rho}{b-m}$. To be consistent with numerical simulations this threshold parameter also indicates whether the virus can drive the host population to extinction, case $\mathcal{S}_{pm} \leq \frac{\sigma_{pm}}{(1-\varepsilon)\alpha}$, or not control its exponential growth, case $\frac{\sigma_{pm}}{(1-\varepsilon)\alpha} + \frac{\rho}{b-m} \leq \mathcal{S}_{pm}$. As a consequence indirect transmission, $\rho > 0$, is required to get regulation of the host population in the proportionate mixing incidence model.

The age dependent model was introduced to take into account a more severe impact of FPLV on young individuals than on adults ones, a fact supporting the choice of FPLV to eradicate a cat population having about 40% of juveniles. Again we show the existence of at least one positive age dependent stationary state when $\mathcal{T}_0(\varepsilon, \theta) < 1$. We conjecture it is unique and stable when $\mathcal{T}_0(\varepsilon, \theta) < 1$, and that no such stationary state exists when $\mathcal{T}_0(\varepsilon, \theta) \geq 1$ in which case the host population resumes an exponential growth; this is related to the likely monotonicity of $I^\sharp \to F(I^\sharp)$ in (27).

This is consistent with our results for the unstructured model. This is also consistent with the field observation that FPLV was not able to drive the host population to extinction, but instead a positive stable age dependent stationary state emerged.

In the age dependent case the proportionate mixing incidence poses much more complex mathematical problems.

An intermediate situation is a model with only two age classes, made of juvenile and adult individuals. It would supply a good qualitative and reliable picture of the dynamics of this host-parasite system when the parasite has a more severe impact on young hosts than on adult ones.

Remark 3. Spatial effects for FPLV models can be considered as well, cf. [10] [12]. In this case Fickian diffusion is introduced into the models to describe the dispersion of the Feline population. Finally we point out that environmentally supported pathogens arise in other contexts as well, two very important are the anthrax and staphylococcus bacteria.

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