Modelling the Lethargic Crab Disease

Claudia P. Ferreira, **P. F. A. Mancera**,
Depto de Bioestatística, IBB, UNESP,
18618-000, Botucatu, SP
E-mail: pio@ibb.unesp.br, pmancera@ibb.unesp.br,

M. R. Pie, **W. A. Boeger**,
Depto de Zoologia, GIA, UFPR
80060-000, Curitiba, PR
E-mail: pie@ufpr.br, wboeger@ufpr.br,

L. Esteva
Depto de Matemáticas - UNAM
Cidade do México, México
E-mail: lesteva@matematicas.unam.mx

Abstract: *A mathematical model to describe the Lethargic Crab Disease (LCD) transmission into mangrove complexes is developed. The dynamical behaviour of the system for different parameters is investigated. The model presents four possible scenarios, namely, the trivial equilibrium, the disease free equilibrium, endemic equilibrium, and limit cycles arising from Hopf bifurcation. The threshold values depend on the basic offspring number of crabs and fungus, and on the infection rate. These scenarios are linked with the biological assumptions and with the temporal evolution of the disease. Numerical simulations corroborate the analytical results and illustrate the different temporal dynamics of the crabs and fungus population.*

Introduction: The mangrove crab, *Ucides cordatus*, can be found on the Americas Atlantic coast, from Florida (US) to Santa Catarina (Brazil) and plays a crucial role in a variety of ecosystem processes in its environment, such as nutrient cycling, and biomonitoring programs. Moreover, it is an important component in the economy of several underprivileged communities that depend on it for their subsistence and feeding (Glaser, 2003).

At the beginning of 1997, massive mortality of *U. cordatus*, such as 84% reduction in collection rates, have been reported by crab-collectors in Brazil. Crabs in areas of high mortality share several common symptoms, such as lethargy, poor motor control and inability to return to the upright position when turned upside down. Hence, this pathology is called *Lethargic Crab Disease* (LCD). In unpublished accounts, several potential etiological agents have been linked with LCD, including protists, fungi, bacteria, introduction of exotic metazoans and chemical poisoning. Finally, in 2005, there were several evidences showing that LCD is caused by a fungus of phylum Ascomycota (Boeger et al., 2005).

I seems that susceptible crabs become infected through direct contact with fungus, since there are not evidences that the transmission occurs among crabs. Although it has been reported that some individuals develop fungus resistance, in general they become susceptible again as a result of not developed immunological memory. Infected crabs that develop the disease die from 9 to 35 days after the contact with the fungus, and during this time this parasite grows quickly inside the crabs. On the other hand, it seems that the fungus remains latent in infected crabs that do not develop the disease.

The reported data show evidences of a cyclic disease which starts with an epidemic wave characterized by high mortality rates followed by waves with decreasing mortality rates until

the disease disappears. The mortality occurs preferentially during summer, suggesting that seasonal factors, such as mating, promote fungus growth and transmission. Started in Recife (PE-Brazil), the disease spreads preferentially to North-South direction. Until now it has been arrived to 17 estuaries in the coastal states of Brazil (Revista do Gia (Grupo Integrado de Aqüicultura e Estudos Ambientais), (2006)).

In this work we formulate a mathematical model to describe the Lethargic Crab Disease transmission into mangrove complexes. The model shows that the infection can be sustained only above a threshold depending on the infection rate, recovery rate, and other demographic parameters. The system presents an endemic stable state in some region of the parametric space and exhibits periodic oscillations in other regions. Numerical simulations corroborate the analytical results and illustrate the models dynamics.

1 Model Formulation

Let $S(t)$ and $I(t)$ denote the adult population of susceptible and infected crabs with fungus at time t, respectively. The fungus population is denoted by $F(t)$. The assumptions of the model are the following.

The susceptible crabs population, $S(t)$, is increased by births of individuals (assumed susceptible) into the population. The net oviposition rate per female crabs population is proportional to their density, but it is also regulated by a carrying capacity related to the amount of available nutrients and space. In this model the per capita oviposition rate is given by $\phi\left(1-\frac{S(t)}{C}\right)$, where C is the carrying capacity and ϕ is the intrinsic oviposition rate. Susceptible crab population decreases by natural death at a per capita rate μ, and at a rate μ_c as a result of been fished and commercialized by the crabs collectors. In the presence of the disease, susceptible crab population also increases by the return of the infected crabs that do not develop the disease and become susceptible again at a per capita rate γ, and decreases by infection at a rate βSF.

The infected crab population is generated at a rate βSF, and is diminished by natural death (at a per capita rate μ), recovery (at a per capita rate γ) and disease-induced mortality (at a per capita rate α). Infected crabs died minutes after fishing by the crab collectors, therefore we are not considering additional mortality in this population as a result of crab capture.

The fungus reproduces on the body of infected crabs at a rate σ. Thus, this population is increased at a rate $\sigma\alpha I$, and decreased by natural death at a per capita rate μ_F.

According to the assumptions above the model is given by the following system of nonlinear differential equations

$$\begin{aligned}\frac{dS(t)}{dt} &= \phi S(t)\left(1-\frac{S(t)}{C}\right)-(\mu+\mu_c)S(t)\\ &\quad -\beta S(t)F(t)+\gamma I(t),\\ \frac{dI(t)}{dt} &= \beta S(t)F(t)-(\gamma+\mu+\alpha)I(t),\\ \frac{dF(t)}{dt} &= \sigma\alpha I(t)-\mu_F F(t),\end{aligned} \tag{1}$$

with $(S(t), I(t), F(t)) \in R^3_+$.

1.1 Equilibrium points

The model accept three equilibria. The first one is the trivial equilibrium $E_0 = (0, 0, 0)$ corresponding to the state where crabs and fungus are absent.

The second equilibrium is the disease free equilibrium $E_1 = (\bar{S}, 0, 0)$, where

$$\bar{S} = \left(1-\frac{a}{\phi}\right)C, \tag{2}$$

and $a = \mu + \mu_c$. This equilibrium has biological sense if and only if $R_C = \frac{\phi}{a} > 1$. In demographic terms R_C is the *basic offspring number* of crabs population (equivalent to basic reproductive number in the epidemiological context). For crabs to maintain in nature, condition $R_C > 1$ is necessary.

Finally, the third equilibrium is the endemic equilibrium $E_3 = (\hat{S}, \hat{I}, \hat{F})$ corresponding to the state where the disease is always present. The coordinates of E_3 are given by

$$\begin{aligned}\hat{S} &= \frac{b\mu_F}{\beta\sigma\alpha},\\ \hat{F} &= \frac{\sigma\alpha}{\mu_F}\hat{I},\\ \hat{I} &= \frac{\hat{S}}{\mu+\alpha}\left((\phi-a)-\frac{\phi\hat{S}}{C}\right),\end{aligned} \tag{3}$$

where $b = \gamma + \mu + \alpha$. From the equations above it is clear that the endemic equilibrium, E_3, is biologically feasible if and only if

$$\frac{(\phi - a)C}{\phi} > \hat{S}, \tag{4}$$

which is equivalent to

$$R_F = \frac{\beta\sigma\alpha(\phi - a)C}{b\mu_F\phi} > 1. \tag{5}$$

The number R_F can be interpreted as the *basic reproductive number* of the fungus population. If $R_F > 1$ the fungus is able to invade the crab population, and to reproduce. On the contrary, if $R_F < 1$ the fungus population will extinguish, and consequently the disease will dye out.

1.2 Stability of the Trivial Equilibrium E_0

The eigenvalues of the local linearization of system (1) around E_0 are $a(R_C - 1)$, $-b$, and $-\mu_F$. All of them are negative if and only if $R_C < 1$. Therefore, E_0 is locally asymptotically stable if $R_C < 1$, and unstable if $R_C > 1$.

1.3 Stability of the Disease-Free Equilibrium E_1

When $R_C > 1$ the trivial equilibrium becomes unstable and E_1 emerges in the feasible region. The stability of E_1 is governed by the eigenvalues of the linearized system of (1) around E_1. These eigenvalues are $-a(R_C - 1) < 0$, and the roots of the quadratic polynomial

$$p(\lambda) = \lambda^2 + (b + \mu_F)\lambda + b\mu_F(1 - R_F). \tag{6}$$

Recall that a quadratic polynomial $\lambda^2 + a_1\lambda + a_2$ has roots with negative real part if and only if $a_1 > 0$, and $a_2 > 0$. For the polynomial (6) $a_1 = b + \mu_F > 0$, and $a_2 = b\mu_F(1 - R_F) > 0$ if and only if $R_F < 1$. Therefore, E_1 is locally asymptotically stable when $R_F < 1$, and unstable when $R_F > 1$.

1.4 Stability of the endemic equilibrium and Hopf bifurcation

For $R_F > 1$ the Jacobian at the endemic equilibrium, E_2, is given by

$$J(E_2) = \begin{pmatrix} \phi - \frac{2\phi}{C}\hat{S} - \beta\hat{F} - a & \gamma & -\beta\hat{S} \\ \beta\hat{F} & -b & \beta\hat{S} \\ 0 & \sigma\alpha & -\mu_F \end{pmatrix}.$$

Substituting the values of $\hat{S}$, $\hat{I}$, and $\hat{F}$ given by (3) in $J(E_2)$, and after some calculations, the characteristic equation is given by

$$\begin{aligned} r(\lambda) &= \lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0 \\ A_1 &= \gamma\frac{(\phi - a)}{\mu + \alpha}\frac{(R_F - 1)}{R_F} + \frac{\phi - a}{R_F} + b + \mu_F \\ A_2 &= \mu_F\gamma\frac{(\phi - a)}{\mu + \alpha}\frac{(R_F - 1)}{R_F} + (b + \mu_F)\frac{(\phi - a)}{R_F} \\ A_3 &= \mu_F b(\phi - a)\frac{(R_F - 1)}{R_F}. \end{aligned} \tag{7}$$

According to the Routh-Hurwitz criteria for a polynomial of degree three, the necessary and sufficient conditions for all eigenvalues of $J(E_2)$ have negative real parts are

(1) $A_i > 0$, $i = 1, 2, 3$, and

(2) $D = A_1A_2 - A_3 > 0$.

Since $R_C > 1$ and $R_F > 1$, the first condition is satisfied for A_i given in (7). Thus, the stability of the endemic equilibrium depends on the sign of D. When $D > 0$, the endemic equilibrium is locally asymptotically stable; when $D < 0$, it is unstable. When $D = 0$, there are a pair of purely imaginary eigenvalues, $\pm i\sqrt{A_2}$, and a negative real eigenvalue, $-A_1$; therefore, for suitable parameter values, a Hopf bifurcation can occur for a particular value of R_F, which implies that it can be a periodic solution around the endemic equilibrium (Guckenheimer, 1983).

2 Numerical study of the system behaviour

In this section the possible dynamics of system (1) have been simulated for a range of parameter values. The results were obtained using Maple procedures and Runge-Kutta of order 4. In all simulations μ_F, ϕ, C, and μ are kept fixed, and their values are $\mu_F = 0.1, \phi = 0.4, C = 200$, and $\mu = 0.0006$, respectively. The other parameters have been varied to obtain the stability region in the corresponding parameter space.

Figure 1 illustrates the stability regions of the equilibria, and the limit cycle in the (β-σ) parameter space. The solid line corresponds to the critical values of β as a function of σ where the dynamics of the endemic state goes

through a Hopf bifurcation. Below this bifurcation curve the endemic equilibrium is stable (region II) and is unstable above it, where a stable limit cycle appears (region III). The infected crabs population does not persist below a minimum infection rate (dashed line), an hence, the disease does not spread in the population, and only the disease free equilibrium is stable (region I). In this case we have $R_F < 1$. Further, we observe that as σ increases, the parameter region were the disease disappear or become endemic get narrow and periodic behaviour is more suitable.

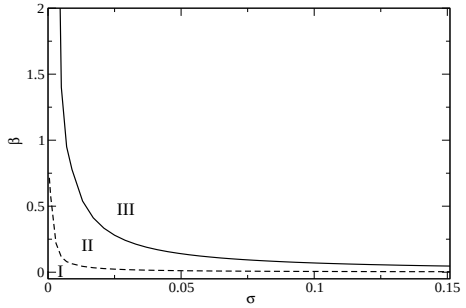

Figure 1: Stability region in the $(\beta - \sigma)$ parameter space. Region I corresponds to the disease free equilibrium, region II to the endemic equilibrium, and region III to limit cycles. The other parameters are fixed and given by $\gamma = 0.01, \mu_c = 0$ and $\alpha = 0.07$.

In Figure 2 the stability regions are shown in the (β-α) parameter space. As before, the critical values of β for Hopf bifurcation are represented by the solid line. These threshold values decrease fast for small values of α to a minimum and then increase slowly as α increases. The inflection point occurs approximately at $\alpha \sim 0.1$ and corresponds to the maximum infected population size. Particularly, for $0 < \alpha < 0.04$ periodic solutions are not possible, and disease free equilibrium is stable below the dashed line, and unstable above it, where the endemic equilibrium exists. Since $\alpha = \tau^{-1}$ where τ is the infectious period time, big or small values of τ promote endemic equilibrium.

The stability regions in the (β-γ) parameter space are given in Figure 3. The solid line corresponds to the critical values of β as a function of γ where Hopf bifurcation occurs. As in the previous figures, region III represents stable limit cycles, and region II endemic equilibria. In the two cases $R_F > 1$ which implies that β

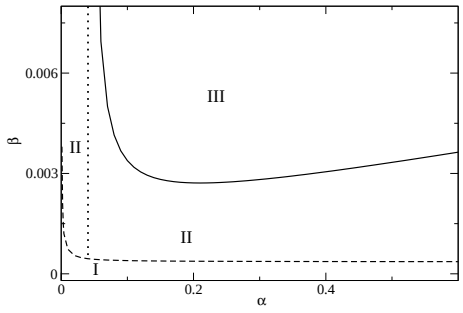

Figure 2: Stability region in the $(\beta - \alpha)$ parameter space. Region I corresponds to the disease free equilibrium, region II to the endemic equilibrium, and region III to limit cycles. The other parameters are fixed and given by $\gamma = 0.01, \sigma = 1.4$ and $\mu_c = 0$.

is bigger than a threshold represented by the almost constant dashed line. Below the dashed line (region I) the disease free equilibrium is stable. When $\gamma > 0.03$ (to the right of the dotted line) periodic solutions are not possible. Notice that in this case, the stability region of the disease free equilibrium is very small. Further, increasing γ increases the probability of the disease to be endemic. The bifurcation di-

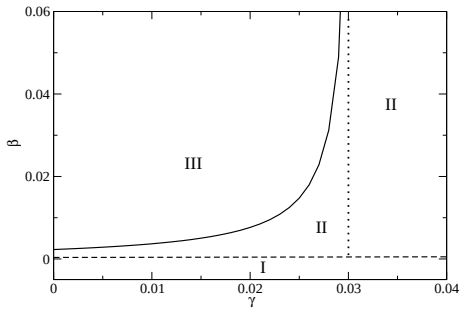

Figure 3: Stability region in the $(\beta - \gamma)$ parameter space. Region I corresponds to the disease free equilibrium, region II to the endemic equilibrium, and region III to limit cycles. The other parameters are fixed and given by $\sigma = 1.4, \mu_c = 0$ and $\alpha = 0.07$.

agram in terms of (β-μ_c) is illustrated in Figure 4. As before, the solid line corresponds to the critical values of β where the dynamics of the endemic state goes through a Hopf bifurcation, therefore in region III we have limit cycles, and endemic equilibria in region II. Below the dashed line (region I) the disease free equilibrium is stable. Both threshold curves are increasing functions of the crab capture rate and become asymptotic as μ_c overtakes the threshold value 0.4. On the left of the dotted line we

have only the trivial equilibrium since in this case $R_C < 1$, meaning that both populations go to extinction. It is interesting to notice that as μ_c increases, the stability region of the endemic equilibrium increases.

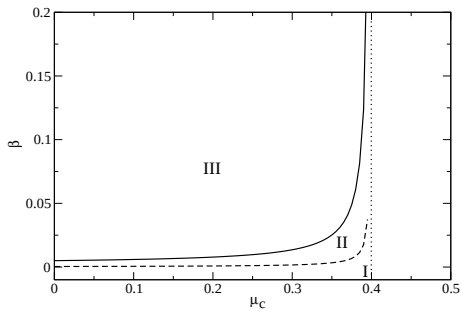

Figure 4: Stability region in the $(\beta - \mu_c)$ parameter space. Region I corresponds to the disease free equilibrium, region II to the endemic equilibrium, and region III to limit cycles. The other parameters are fixed and given by $\gamma = 0.01, \sigma = 1.4$ and $\alpha = 0.07$.

3 Conclusions

The formulated model captures the essential features of the LCD. The results are given in terms of the basic offspring of the crab, R_C, and the fungus population, R_F, as well as the transmission rate β.

When $R_C < 1$, we have the trivial equilibrium in which both populations are absent. In nature this equilibrium is not feasible because there is larvae exchange between the estuaries. For $R_C > 1$, and $R_F \leq 1$ the solutions approach the disease free equilibrium no matter the initial conditions, implying that the disease can be controlled below the last threshold. Since R_F is proportional to the reproductive rate of fungus, σ, changes on this parameter are very important for the development of the disease. This is in accordance to conjectures which point that the disease appearance is related with new forms of fungus with have an reproductive advantage inside the crabs. On the other hand, increasing the mortality rate of fungus decreases R_F, that means that a better understanding of the fungus life cycle, as well as the way it parasites the crab, could be useful to develop control strategies as fungicides.

The model predicts that when $R_F > 1$ there exists an endemic equilibrium where the disease persists all the time. Hopf bifurcation can appear for suitable sets of the eight parameters. The implication of this result is that if $R_F > 1$ the disease dynamics can evolve to an endemic situation or can have an oscillatory behaviour. Numerical analysis corroborates the analytical results about the stability regions of the equilibria and limit cycles. Some interesting results relate both increasing capture and resistance crabs rates to an endemic situation keeping β fixed (see Figures 3 and 4).

Experimental evidences suggest that the periodic behaviour of the disease is associated with extrinsic factors such as the mating cycle of the crabs. During the mating time the crab immune system get depressed due to stress associated with this activity. This situation give to the fungus the opportunity to parasite the crab. However, the model predicts intrinsic oscillations related with demographic and epidemiological parameters, such as recruitment of susceptibles, infectivity , recovery, and mortality rates. Therefore, a question that should be elucidated is which of the above referred mechanisms are the responsible of the disease oscillations.

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