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Integrated Pest Management and Spatial Structure

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1. Introduction

During long time pest control was associated only to insecticides. The formulations were produced as an attempt of improving the insect control. However, some undesirable effects emerged in this time, mainly the toxin action and resistance. Insecticides are substances produced from chemical or biological products to control insect pests. The most common mode of action for insecticides is to kill insects by blocking physiological or biochemical processes (Ware & Whitacre, 2004). Usually, insecticides act on the nervous system, resulting in high efficacy and rapid responses in pest-control programs. Insecticides can be classified as physical, protoplasmic, metabolic inhibitors, neurotoxins, and hormone agonists (Matsumura, 1985). Mineral oil is an example of a physical insecticide, and heavy metals are protoplasmic insecticides (Amiri-Besheli, 2008; Gallo et al, 2002).

Some examples of metabolic inhibitors are the inhibitors of multi-function oxidases, carbohydrate and amino-acid metabolism inhibitors, and chitin-synthesis inhibitors (Krieger, 2001). The neurotoxins act through acetylcholinesterase, the neurotoxin that affects ion permeability, intervening in the nerve receptors of insects (Haynes, 1988), killing the arthropod by disrupting the membrane integrity (Gill et al, 1992).

The main groups of insecticides can be studied using the following classification: neurotoxins, insect growth regulators, cellular respiration inhibitors, and others. Of the neurotoxins, the organophosphates and carbamates act on synaptic transmission, accumulating acetylcholine molecules in the synapse, which in insects can produce a cholinergic syndrome characterized by nerve hyperexcitation (Costa et al, 2008; Thacker, 2002).

The acetylcholine agonists nicotines, neonicotinoids (the newest group of synthetic insecticides) and spinosines connect to the nicotine receptors of acetylcholine located in the pre-synaptic neuron (Thacker, 2002). In this case, the nerve impulses are continuously transmitted, also resulting in nerve hyper-excitation in the insect (Thacker, 2002). The acetylcholine antagonists avermectin and milbemycin block the nerve stimulus, immobilizing the insect. Cycloienes and phenyl-pyrazoles act differently from avermectin and milbemycin, killing insects by inducing hyperexcitability (Thacker, 2002). DDT (dichlorodiphenyltrichloroethane) and pyrethroids are sodium-channel modulators, acting on sodium channels of nerve cells in insects (Thacker, 2002). Action potentials can be repetitive,

also killing insects by inducing hyperexcitability. On the other hand, the oxadiazines are sodium-channel blockers, reducing the ascendant phase of the action potential (Gallo et al, 2002).

Insect growth regulators such as chitin synthesis inhibitors are juvenile hormone agonists that slow development, producing an additional instar or nymph and preventing the insect from reaching the adult stage (Thacker, 2002). The juvenile hormone antagonists produce the opposite effect, forcing the insect to pass to the next life stage too early. Other inhibitors include cellular respiration inhibitors, which act by inhibiting respiratory-chain enzymes with consequent depression of respiratory movements and reduction of oxygen consumption; and adenosine triphosphate inhibitors, which inhibit oxidative phosphorylation (Hien et al, 2003). The use of pesticides has been informally reported since 1000 B.C., but insect chemical control began in World War II, when the concept of insect control became established, opening a new era of synthetic organic insecticides, of which DDT was the first to be applied (Ware & Whitacre, 2004). DDT belongs to the organochlorines, insecticides containing carbon, hydrogen and chlorine, and is probably the most famous pesticide of the 20th Century. It is still used for malaria control in developing countries (Ware & Whitacre, 2004). Another notorious insecticide is BHC, which acts similarly to DDT but more rapidly.

The mid-20th Century saw the development of many pesticides and organophosphates, insecticides based on phosphorus; their development was also hastened during World War II, when they were tested to replace nicotine, mainly in Germany (Thacker, 2002). Because of the high toxicity of this pesticide it has been not recommended since 1990. Among the most common organophosphorus insecticides are malathion, monocrotophos, dicrotophos and methamidophos (Gullan & Cranston, 2005). Organosulfurs are less toxic pesticides that have been employed as acaricides. They differ from DDT in having sulfur in place of carbon. Carbamates, another class of defensives, are derivatives of carbamic acid; the first available member of this class was carbaryl, first marketed in 1956. The carbamates show low oral and dermal toxicity to mammals, and a broad spectrum of insect species are sensitive to the product (Thacker, 2002).

After this era, another class of pesticides was proposed, the pyrethroids. Pyrethroids are obtained from pyrethrum, a natural compound extracted from dried flowers of *Chrysanthemum cinerariifolium* and *C. coccineum* (Gullan & Cranston, 2005) and are much less toxic than organophosphates and carbamates. Their relatively low toxicity is associated with nonpersistent sodium-channel modulators (Dent, 2000). More recently, the synthetic neonicotinoids have been developed; these are analogues of the natural insecticide nicotine (Ware & Whitacre, 2004). They are nicotinic acetylcholine receptor agonists, with a broad spectrum and rapid action to replace the organophosphate and carbamate applications. Biological insecticides have been developed in order to avoid the application of chemical toxins to crops. Perhaps the most important idea was to use the endotoxins produced by *Bacillus thuringiensis* (BT), which acts by disintegrating epithelial cells of the mesentery (Gill et al, 1992). Some plants have been genetically modified to express BT toxins.

In spite of the great variety of pesticide formulations that act in different ways to control pests, the resistance of insects to insecticides has been reported frequently and over a long period. The definition of resistance proposed by the Insecticide Resistance Action Committee (IRAC) is “the selection of a heritable characteristic in an insect population that results in the repeated failure of an insecticide product to provide the intended level of control when used as recommended” (IRAC, 2010). Resistance to insecticides has been reported since 1914, initially for DDT, but has extended to new insecticide classes including cyclodienes, carbamates,

formamidines, organophosphates, pyrethroids and even *B. thuringiensis* (IRAC, 2010; Thacker, 2002). For this reason, the 1940s saw the beginning of more systematic investigations of the indiscriminate use of insecticides.

The toxicity of insecticides has been regularly discussed in the context of environmental contamination and human health (Wilson & Tisdell, 2001). However, there is no doubt of the importance of discussing their effects on animals, plants and the environment, particularly considering global warming. For example, of the organochlorines, the cyclodienes, which also appeared during World War II, have a different mode of action from DDT, and their toxicity increases with increasing temperature (Ware & Whitacre, 2004). In spite of the recent discussions of global warming with respect to diseases and insect dynamics (Lima et al, 2009), no systematic discussion has analyzed the possible effects of global warming on the toxicity of pesticides, except for a few isolated experiments (Gordon, 2003). This subject deserves special attention, taking into account that the earth's surface is predicted to warm by approximately 1.5 °C to 6 °C by the year 2100 (Kiritani, 2006).

Considering the risks of resistance, toxicity, and increase of toxicity associated with rising temperatures, the development of new pest-control methods should be encouraged to minimize the undesirable effects of pesticides. The use of traditional pesticides should be controlled to avoid the problems previously mentioned (Thompson & Head, 2001). The challenge is to identify and develop crop protection systems that integrate many measures to reduce and maintain a particular pest population at an acceptable level of economic damage (Radcliffe et al, 2009). One of the first articles involving the principles of Integrated Pest Management (IPM) was written in 1976 by Ray et al. (Smith & Calvert, 1976). This technique has been implemented for pest control, with acceptable results for different agricultural systems, and also to control disease vectors (Lima et al, 2009). The goal of IPM principles is to improve crop yield with minimum cost, taking into consideration the ecological and sociological constraints imposed by the particular agroecosystem under study and the long-term preservation of the environment. To achieve this, IPM techniques use a variety of approaches: first, to increase the knowledge of the insect pest and its relationship to the crop and factors affecting their interaction; second, to develop several techniques such as biological pest controls, farming practices, mechanical, and physical controls to reduce pesticide application; third, to improve methods of collecting and interpreting biological, meteorological, and crop production data; fourth, to build models of the crop production, pest dynamics, and management tactics integrated with an economic analysis to optimize crop yield; and finally, to conduct laboratory and field experiments to test these models (Smith & Calvert, 1976).

Therefore, an important strategy in any pest-control program is to determine the essential components of IPM in order to monitor the pest's status in the system. Monitoring of a pest's abundance in time and space is a powerful tool that provides information needed to decide on the best time to effectively implement control actions, by using insecticide applications and/or combining these methods with biological control strategies (Lima et al, 2009). The establishment of plans for sampling populations is an essential part of IPM programs, since it provides support for decision-making based on the pest density, forecast, and economic threshold (Beinns et al, 1992; Spencer et al, 2009). Ecological modeling is one of the most important initial components in IPM programs (Lima et al, 2009). By using models, it is possible to understand better the processes that govern biological systems of pest insects, because they describe very well the complexity involved in the population dynamics of

species from simple assumptions incorporated in the theoretical formalism (Faria & Godoy, 2001; Serra et al, 2007).

In the 1980s, IPM principles began to be used to control insect populations in urban sites, such as schools, parks, hospitals, and nursing homes; and following these ideas, in the 1990s mathematical models began to be constructed to analyze and discuss IPM methods in a more qualitative and quantitative way (Lima et al, 2009; Tang et al, 2005; Tang & Cheke, 2008). In particular, Tang and coworkers demonstrated a stable periodic solution in a prey-dependent consumption model with fixed impulsive effects, and gave an analytical expression for the period of this periodic solution. This period plays an important role in pest control, because it can be used to alter an IPM strategy with unfixed times for interventions, to one with periodic interventions, thus minimizing the cost of pest monitoring (Tang et al, 2005).

Recently, an extension of the Nicholson & Bailey model was proposed by Tang & Cheke, including the Integrated Pest Management strategies, in order to consider the economic threshold in the formulation. The study showed that the host level can be maintained below the economic threshold (ET), avoiding reaching the economic injury level (EIL). The study by Tang & Cheke (2008) also showed that high initial densities of parasitoids and high parasitoid inter-generational survival may lead to more frequent host outbreaks and, therefore, greater economic damage (Tang & Cheke, 2008). Lima and coworkers, using the formulation of a coupled map lattice, added spatial structure to this model, and showed that it can significantly alter the economic threshold-level values, which is an important aspect to consider in the success of the IPM technique (Lima et al, 2009).

In conclusion, the theory of pest control is closely associated with the basic principles of ecological theory, since its emphasis involves essentially the use of biological control strategies, which emerge from the classical theory of predator-prey relationships, with special application to insects (Hassell, 1978; Hochberg & Ives, 2000). In this chapter, we intend to show how theoretical ecology and pest-management strategies can be combined to facilitate the comprehension of important ecological aspects of a pest population, which directly influence its dynamics as well as the dynamics of its natural enemies. Theoretical models can address the relevance of intrinsic and extrinsic factors that affect the spatial and temporal dynamics of a biological system, and can also be useful to investigate different scenarios about its control.

2. Mathematical model

In this section, a non-spatial and also a spatial model will be developed to analyze preliminarily the contribution of several factors that can contribute to the effectiveness of the IPM methodology.

2.1 Population dynamics without MIP

Let us suppose a hypothetical pest of a crop that has a natural enemy, an insect that is a parasitoid, and also a predator of this pest. As an example, we can cite *D. citri* as a pest of the orange crop, and *T. radiata* as its natural enemy. In this case, the natural enemy can attack different stages of the pest's life cycle, acting as a good candidate for biological control (Fauvergue & Quilici, 1991). All mathematical models must be constructed based on the life cycles of the populations that are relevant for the process under study. Also, the complexity arises from the standpoint of mathematics and computing leads us to make simplifications in modeling the biological problem. Therefore, bearing in mind these two insect species and the host-parasitoid-prey-predator interaction, let us divide the pest's life

cycle into four compartments: egg (O), two nymphs (N_1, N_2), and the adult female (F); which represents the number or density of individuals in a specific development stage at time t . The variables N_1 and N_2 represent the number of individuals undergoing predation and parasitism, respectively. With respect to the natural enemy, we will consider two development stages, one the juveniles (J) and the other the adult female (P). Also, we adopted for the interaction between the populations, the Type II function response, where consumption and parasitism rise asymptotically to saturation. Thus, the following system of differential equations describes the temporal evolution of the individuals in each compartment:

$$\begin{cases} \frac{dO}{dt} = \eta F \left(1 - \frac{O}{K}\right) - (\sigma_o + \mu_o)O, \\ \frac{dN_1}{dt} = \sigma_o O - (\sigma_{n_1} + \mu_{n_1})N_1 - \frac{\gamma N_1 P}{1 + \phi N_1}, \\ \frac{dN_2}{dt} = \sigma_{n_1} N_1 - (\sigma_{n_2} + \mu_{n_2})N_2 - \frac{\psi_t \alpha N_2 P}{1 + \beta N_2}, \\ \frac{dF}{dt} = \psi_c \sigma_{n_2} N_2 - \mu_f F, \\ \frac{dJ}{dt} = \theta \frac{\psi_t \alpha N_2 P}{1 + \beta N_2} - (\sigma_j + \mu_j)J, \\ \frac{dP}{dt} = \sigma_j J - \mu_p P \left(1 - \omega \frac{\gamma N_1}{1 + \phi N_1}\right). \end{cases} \quad (1)$$

In the first equation, we assume a logistic population growth for the number of eggs, where η is the per capita oviposition rate and K is the carrying capacity, since oviposition occurs in new shoots, as is usual in a large number of crops. The number of eggs decreases due to the natural per capita mortality rate μ_o , and by the per capita eclosion rate σ_o . In the second equation, the nymph population N_1 increases by the eclosion of eggs and decreases by the per capita natural mortality, μ_{n_1} , and due to transition to the N_2 stage at the per capita rate σ_{n_1} , and predation, where γ is the per capita predation rate and ϕ is related to the prey handling time. In the third equation, the population N_2 increases by the transformation of N_1 into N_2 and decreases by the natural per capita mortality rate μ_{n_2} , transition to the adult phase at a per capita rate σ_{n_2} , and due to parasitism, where ψ_t is the sex ratio, α is the per capita parasitism rate and β is related to the host handling time. Finally, adult females increase as $\psi_c \sigma_{n_2}$, where ψ_c is the sex ratio, and decrease by the natural per capita mortality rate μ_f .

For the natural enemy, we assume that it is a specialist parasitoid, and therefore juveniles increase when the female emerges from a host, where θ is the mean number of juveniles; and decrease by the natural per capita mortality rate μ_j and by transition to the adult stage at the per capita rate σ_j . The adult population increases by the transformation of juveniles to adults and decreases by the per capita mortality rate μ_p . Furthermore, we will assume that the enemy's natural mortality rate decreases by $\omega < 1$ because experimental results show that predation, in general, increases the survival of the predator.

2.2 Adding IPM to the mathematical model

The size of the insect population is affected by extrinsic factors such as the amount of available food and the weather. Therefore, the spatial-temporal pattern for the number of individuals observed in the field shows periodic oscillations with different amplitudes and spatial heterogeneity. An insect becomes a pest when it exceeds some threshold related to its population size, and begins to cause economic injury to the producer, and also to impact the local or global economy. Integrated pest management programs use current, comprehensive information on the life cycles of pests and their interaction with the environment. This

technique relies on monitoring and identifying pests and their natural enemies, setting an action threshold, prevention methods, and control. It is an interesting alternative to the use of pesticides that, besides leading to pesticide-resistance problems, contaminate food, soil and water and remove a pest's natural predators and other non-target species. In fact, the IPM concept incorporates an array of management tactics including biological pest control, farming practices, and mechanical, physical and chemical controls. In this study, we are dealing with two of them, pesticide spraying and parasitoid release.

In order to add IPM strategies to the system described in (1), we must remember that in IPM programs, both pesticide spraying and natural enemy release occur when the prey population density reaches the economic threshold, ET , in order to maintain the prey density below the economic injury level, EIL (see Fig. 1). Accurate determination of these two thresholds require knowledge of the pest, plant health problems, and what constitutes unacceptable pest damage. Thus, each insect-ecosystem has its specific thresholds. To incorporate these two processes, pesticide spraying and parasitoid release, into an IPM program, the system (1) should be written as

$$\left. \begin{aligned} \frac{dO}{dt} &= \eta F \left(1 - \frac{O}{K}\right) - (\sigma_o + \mu_o)O, \\ \frac{dN_1}{dt} &= \sigma_o O - (\sigma_{n_1} + \mu_{n_1})N_1 - \frac{\gamma N_1 P}{1 + \phi N_1}, \\ \frac{dN_2}{dt} &= \sigma_{n_1} N_1 - (\sigma_{n_2} + \mu_{n_2})N_2 - \frac{\psi_t \alpha N_2 P}{1 + \beta N_2}, \\ \frac{dF}{dt} &= \psi_c \sigma_{n_2} N_2 - \mu_f F, \\ \frac{dJ}{dt} &= \theta \frac{\psi_t \alpha N_2 P}{1 + \beta N_2} - (\sigma_j + \mu_j)J, \\ \frac{dP}{dt} &= \sigma_j J - \mu_p P \left(1 - \omega \frac{\gamma N_1}{1 + \phi N_1}\right), \end{aligned} \right\} \text{if } F < ET, \quad (2)$$

$$\left. \begin{aligned} O(t_0^+) &= (1 - \delta_c)O(t_0), \\ N_1(t_0^+) &= (1 - \delta_c)N_1(t_0), \\ N_2(t_0^+) &= (1 - \delta_c)N_2(t_0), \\ F(t_0^+) &= (1 - \delta_c)F(t_0), \\ J(t_0^+) &= (1 - \delta_c)J(t_0), \\ P(t_0^+) &= (1 - \delta_c)P(t_0) + \tau, \end{aligned} \right\} \text{if } F \geq ET,$$

where

$$\delta_c = 1 - \frac{ET}{F}, \quad (3)$$

is the instantaneous per capita mortality rate in response to the pesticide applied at $t = t_0$, τ is the number of parasitoids released at this time, and $O(t_0^+)$, $N_1(t_0^+)$, $N_2(t_0^+)$, $F(t_0^+)$, $J(t_0^+)$ and $P(t_0^+)$ are the number of individuals in each class after pesticide application (Tang & Cheke, 2008). To simplify, we are assuming that control measures affect all pest and natural-enemy stages with the same intensity.

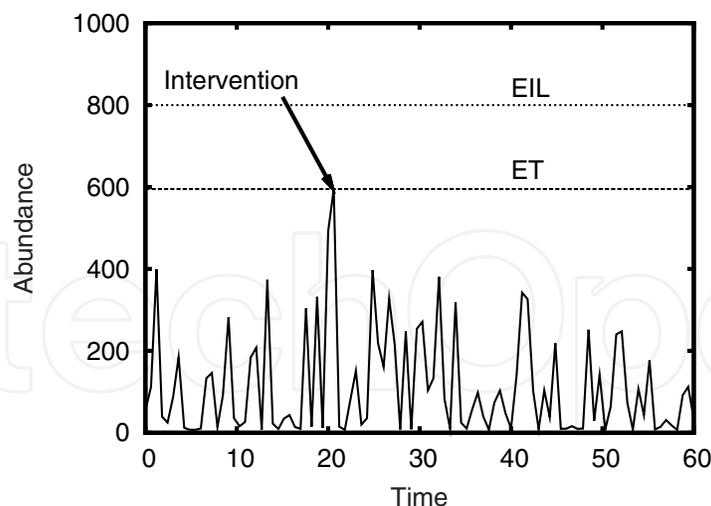


Fig. 1. Temporal evolution of the insect population, economic injury level (EIL) and economic threshold (ET).

2.3 Adding spatial structure to the mathematical model

To consider the spatial structure, we use the formulation of coupled lattice models. The bidimensional lattice with 4×4 sites, represents the crop plots (see Fig. 2). Each plot consists of 45×45 sites, each of which represents a specific tree that serves as a source of food for the pest, e.g., an orange tree. Dispersal occurs between adjacent sites, considering the Moore neighborhood of one radius. We implemented an asynchronous lattice update and a fixed boundary condition. At each site, the pest and natural enemy populations are arranged in such a way that each equation of the system (2) receives an index i that refers to a grid cell within the lattice. In each grid cell, the system is solved using the Runge-Kutta 4th-order method.

At each simulation time step, which corresponds to one day, the dynamics consist of three phases: population dynamics (reproduction-parasitism-predation phase), dispersal phase, and population control. The results are shown using $\nu_d = 0.85$ and $\nu_t = 0.6$ for the dispersal of the pest and its natural enemy, respectively. Therefore, at each time step, the fraction of the pest and the natural enemy populations that undergo migration are $\nu_d F/8$ and $\nu_t P/8$. The other parameters are $\eta = 9.880$, $\sigma_0 = 0.1422$, $\mu_0 = 0.1060$, $\sigma_{n_1} = 0.1031$, $\mu_{n_1} = 0.2029$, $\sigma_{n_2} = 0.08292$, $\mu_{n_2} = \mu_j = 0.01892$, $\mu_f = 0.01976$, $\sigma_j = 0.05882$, $\mu_p = 0.02941$ all in day^{-1} , $\beta = 0.6$, $\phi = 0.6$ in day, $\theta = 0.7$, $\alpha = 0.1$, $\omega = 0.2$, $\gamma = 0.2$, $\psi_c = 0.5$, $\psi_t = 0.6429$ and $K = 7500$. Again, the parameter values were chosen bearing in mind the *D. citri* and *T. radiata* interaction system (Liu & Tsai, 2000; Pluke et al, 2008). Indeed, the same qualitative results are obtained for other set parameters, and can be discussed in another ecological system in context. For each specific system of pray-natural enemy, these parameters must be estimated in laboratory and field experiments.

We started by randomly choosing 20 sites (in the order of 1% of the total lattice) to quantify the number of female adult insects, F , above the economic threshold, ET . Therefore, if more than two sites (in the order of 20% of the total analyzed) have $F > ET$, we applied insecticide followed by release of the parasitoid. We chose to apply the control techniques in the same way as is usual to control the Citrus Variegated Chlorosis (CVC), disease caused by a xylem-inhibiting bacterium *Xylella fastidiosa* and transmitted by 11 species of sharpshooter leafhoppers, because for the *D. citri* and *T. radiata* interaction system, the control strategy is

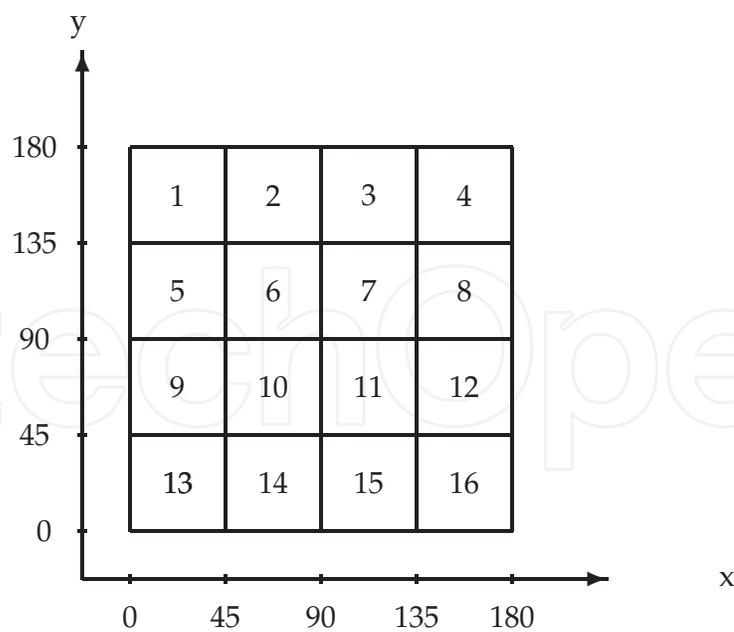


Fig. 2. Lattice used in the simulations. Each plot consists of 45×45 sites that represent the source of food for the pest. In each site, the dynamic system (2) is solved and dispersal occurs between adjacent sites.

still being developed (remember that *D. citri* is a vector of *Liberibacter sp.*, the causal agent of huanglongbing (HLB), and in these circumstances the idea of an ET is difficult to implement). In a real situation, only a proportion of the crops are monitored periodically, and the control is applied or not based on the analysis of these random samples. We also assume that the proportion of individuals that die by pesticide application is proportional to the highest value of female pests obtained in this analysis. Also, the control techniques are applied in the same way to all crops.

3. Results and discussion

In this section, the model will be analyzed to gain insight into its dynamic features.

3.1 Model without and with IPM and non-spatial structure

Let us begin by analyzing the non-spatial model without IPM strategy. Equilibria for the system (1) are found by setting the right half of each equation equal to zero. It can be seen that the model accepts three equilibria:

- *Trivial equilibrium* given by $E_0 = (0, 0, 0, 0, 0, 0)$, corresponding to the state where the populations of the pest and the natural enemy are absent;
- *Pest persistence and natural enemy exclusion* given by $E_1 = (O^*, N_1^*, N_2^*, F^*, 0, 0)$, corresponding to a state where the pest population persists while the natural enemy is absent;
- *Coexistence of the two populations* given by $E_2 = (O^*, N_1^*, N_2^*, F^*, J^*, P^*)$, corresponding to the state where both populations are present.

The stability analysis is given by the eigenvalues of the characteristic equation $\Delta(\lambda) = \det(J^* - \lambda I) = 0$, evaluated at each equilibrium point, where J^* is the Jacobian matrix (linearization of the system dynamics) and I is the identity matrix. After some algebraic

manipulation, we are able to determine threshold values that divide the solution space. Thus, E_0 is locally asymptotically stable if $R_{c_1} < 1$, and unstable if $R_{c_1} > 1$, where

$$R_{c_1} = \frac{\eta\sigma_o\sigma_{n_1}\sigma_{n_2}\psi_c}{(\sigma_o + \mu_o)(\sigma_{n_1} + \mu_{n_1})(\sigma_{n_2} + \mu_{n_2})\mu_f}. \tag{4}$$

In demographic terms, R_{c_1} is the *basic reproductive number* of the pest population (equivalent to the basic reproductive number in the epidemiological context). Interestingly, R_{c_1} does not depend on the interaction between the pest and its natural enemy. When $R_{c_1} > 1$, the pest population is able to maintain itself in the field, and the equilibrium E_1 emerges in the feasible region. The stable state of E_1 is thus obtained when $R_{c_2} < 1$, where

$$R_{c_2} = \frac{(KA)^2(l\mu_p\omega\gamma\beta + \psi_t\sigma_j\theta\alpha\phi) + KAB(\sigma_{n_1}\psi_t\sigma_j\theta\alpha + fl\omega\gamma\mu_p)}{\mu_p(2B^2f\sigma_{n_1} + (KA)^2l\beta\phi + KAB(\sigma_{n_1}l\beta + fl\phi))}, \tag{5}$$

and

$$\begin{cases} f = \sigma_{n_2} + \mu_{n_2}, \\ l = \sigma_j + \mu_j, \\ A = \eta\psi_c\sigma_{n_2}\sigma_{n_1}\sigma_o - (\sigma_o + \mu_o)(\sigma_{n_1} + \mu_{n_1})(\sigma_{n_2} + \mu_{n_2})\mu_f, \\ B = \psi_c\sigma_{n_2}\eta(\sigma_{n_1} + \mu_{n_1}). \end{cases} \tag{6}$$

Therefore, E_1 is locally asymptotically stable when $R_{c_2} < 1$ and unstable when $R_{c_2} > 1$. The equilibrium E_2 was analyzed numerically. It seems that for a suitable parameter value, a periodic solution around the equilibrium E_2 can appear. In this case, the dynamic features of the system depend on the interaction between the two populations that comprise the biological system. Because we are interested in discussing a pest-control technique, the results were obtained for parameter values that give $R_{c_1} > 1$ and $R_{c_2} > 1$.

Fig. 3 shows the temporal evolution of the pest and natural-enemy populations, for the non-spatial and non-ET model (equation (1)) using the parameter set described above, in this case, $R_{c_1} = 39.3$ and $R_{c_2} = 2.0$. The temporal pattern obtained for the two populations exhibits periodic oscillations with a maximum amplitude of 18000 individuals for the pest, and a period of 2 years. The increase of the pest population is followed by the increase of the parasitoid-predator population, with a maximum amplitude of 4000 individuals.

Fig. 4 shows the temporal dynamics of the system when an IPM program is in progress. The economic injury level was defined as $EIL = 150$, and the system dynamics was managed to allow the pest density to fall below the EIL level. In order to achieve our goal, we must consider the economic threshold as $ET = 30$. On the order of $\tau = 20$ parasitoid adults are released. As a result, Fig. 4, shows that population coexistence is maintained, but the amplitudes of the pest and natural enemy oscillations decrease respectively to 1200 and 400 individuals. For this parameter set, both the optimum times of the applications, Δ_t , and the percentage of the pests that needs to be eliminated with pesticides can be estimated (plotting δ_c given by equation (3) as a function of time) resulting in a periodic application of pesticide at intervals of 21 days, and the pesticide should kill approximately 6.2% of the population. Also, the periods of oscillation for both populations decrease to less than one year, and the lag time observed between the temporal dynamics of the two populations decreases.

To analyze the influence of the ET on the determination of Δ_t , in Fig. 5(a) we ran several simulations, varying ET and estimating Δ_t necessary to maintain the pest population below the EIL . The other parameters are the same as in Fig. 4. The results are shown for two different values of τ , which measure the number of parasitoids released. The solid and dotted

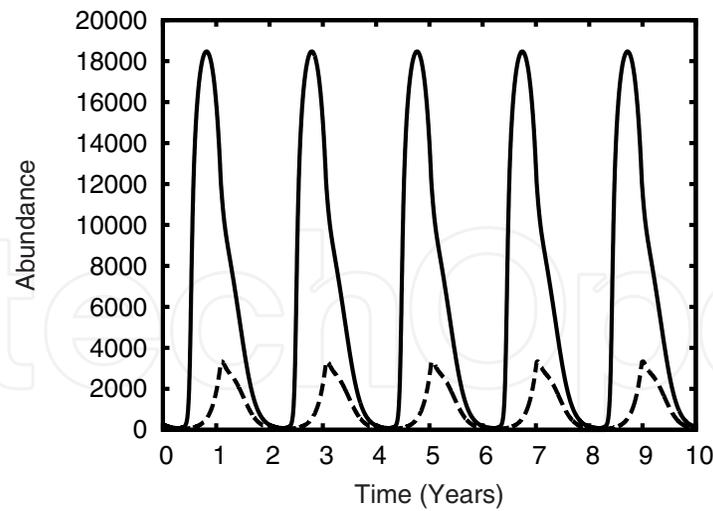


Fig. 3. Temporal evolution of the total prey (solid line) and natural enemy (dashed line) population for the non-spatial model without IPM strategy.

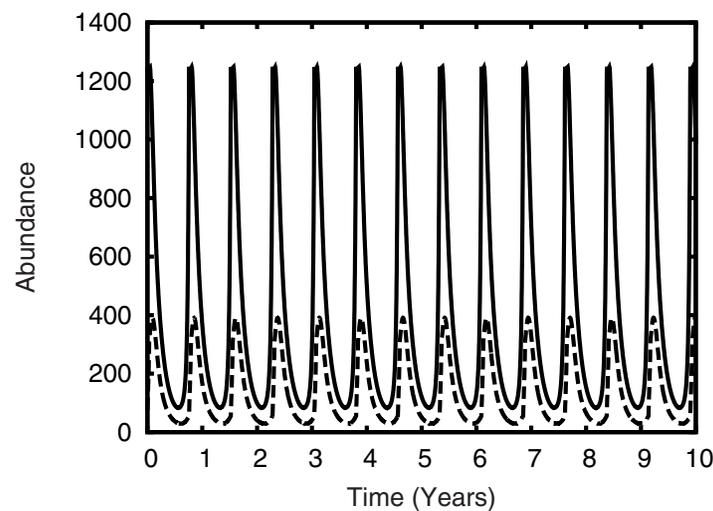


Fig. 4. Temporal evolution of the total pest (solid line) and natural enemy (dashed line) populations for the non-spatial model with IPM strategy

lines divide the $(\Delta_t - ET)$ parameter space into two regions; below the curve, the *EIL* is not exceeded; and above the curve, the pest population is greater than the *EIL*. Also, the qualitative behavior of Δ_t versus ET seems to be a rational function such as $\Delta_t = a_0 / (a_1 + a_2 ET)$. Moreover, the number of times that the IPM technique must be applied, N_p , increases almost linearly with ET , $N_p = b_1 + b_2 ET$, with a linear slope of 4.6 for $\tau = 20$ and 2.2 for $\tau = 30$ (Fig. 5(b)). In brief, the values of a_0, a_1, a_2, b_1 and b_2 depend on the parameters set, but the qualitative behavior of the curves does not change. Finally, an increase τ leading to the increase of Δ_t and decrease of N_p , indicates a range of possible strategies that may be associated with an economic cost to the producer.

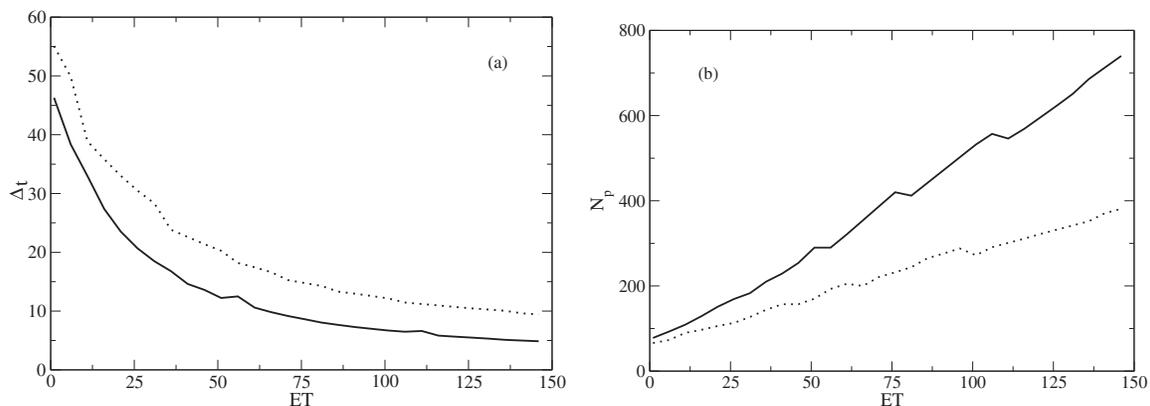


Fig. 5. In (a) interval between pesticide application, Δ_t , versus economic threshold, ET . The lines divide the $(\Delta_t - ET)$ parameter space into two regions, and are obtained for different τ values. Below each line, the IMP technique succeeds, and above it the technique fails. In (b) the number of applications in 15 years versus ET . The solid line corresponds to $\tau = 20$ and the dotted line to $\tau = 30$.

3.2 Spatial model with IPM

The simulation starts with a random selection of a site in each crop, to be occupied by a pest and a natural enemy individual, and all other lattice sites are empty (simulating an initial invasion-colonization of the crop, in which a small number of individuals arrived first). Therefore, the system dynamics (reproduction-parasitism-predation phase, dispersal phase, and population control) evolves and a snapshot of the lattice configuration in different time steps can be analyzed.

Fig. 6 shows the spatial distribution for the pest adult females for $\tau = 20$ using the parameter set described in Fig. 4. Different levels of shading represent different numbers of pests, respectively, $F < ET$ (gray), $ET \leq F < EIL$ (white) and $F \geq EIL$ (black). Because we are considering a homogeneous diffusion (with no preferential direction), the observed pattern is a symmetrical wave front started at each initial occupied site. Interestingly, this leads to a larger number of pests at the border of the crop, which is observed in the field.

Fig. 6 shows two snapshots of the lattice configuration at different time steps. Following the crop numeration shown in Fig. 2, we conclude that in crops 5 and 16, IPM control was applied. However, control efforts depend on the estimate of the number of adult female pests, F , which also depends on the crop-site sampling. As a result, we can see a higher efficacy of IPM for crop 16 compared with crop 5. Moreover, we can see a reinfestation of crop 16 as a result of the migration of the pest population from crop 12, where IPM control was not applied.

Fig. 7 shows the influence of the ET on the determination of Δ_t for the spatial model. In order to discuss the importance of the spatial structure for the IPM technique, the results obtained for the non-spatial model are also added. We can see that the non-spatial model also overestimates the time interval for the IMP application, leading to failure of the technique. For each value of ET , we are able to calculate the percentage of the lattice with $F \geq EIL$ that gives a measure of the economic damage to the producer. For $ET = 30, 60$ and 90 , we obtain, respectively, 0.352%, 0.512% and 0.921% of the lattice site with F above EIL . The results plotted for the spatial model are the mean values of 47 simulations for each value of ET .

In a recent study, Lima and coworkers showed that the ET level should be lower than the value suggested by non-spatial models, to assure that pest density remains below the EIL

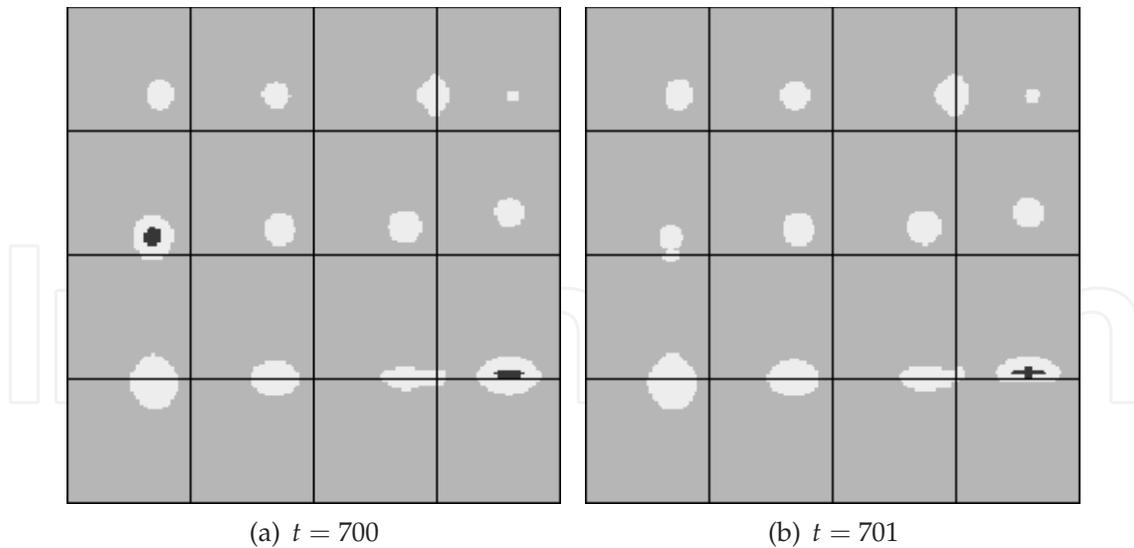


Fig. 6. Different levels of shading represent different numbers of the pest, respectively, $F < ET$ (gray), $ET \leq F < EIL$ (white) and $F \geq EIL$ (black). In (a) snapshot of the lattice configuration at time $t = 700$ and in (b) for time $t = 701$.

level (Lima et al, 2009). Looking at Fig. 7, we can see that the difference in the Δ_t values obtained for a non-spatial and a spatial model is greater for small values of ET . Certainly, these results show that the spatial structure affects the ET level, and consequently also the interval between applications of IPM, and seems to be the main reason for the failure of the technique.

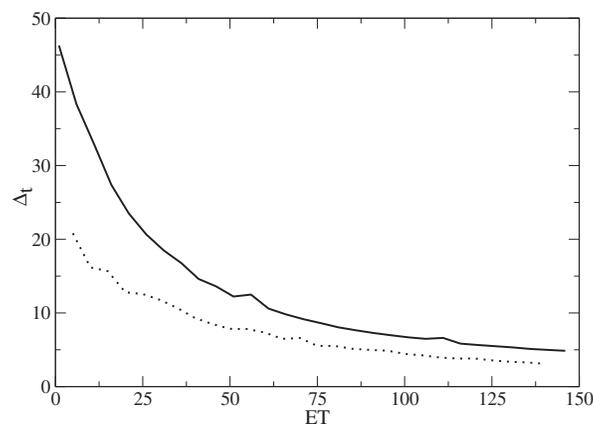


Fig. 7. Interval between pesticide application, Δ_t , versus economic threshold, ET . The solid line corresponds to the non-spatial model, and the dotted line to the spatial model.

4. Conclusion

Ecological modeling is an important tool for systematic study of the use of the IPM technique to control insect populations. Different scenarios can be planned and tested prior to implementation, making experimental designs more efficient and saving time and money. Of course, every mathematical model is a caricature of the real biological system, and

the knowledge of the insect pest and its relationship to the crop and factors affecting the interaction between them determines the degree of accuracy of a model's predictions.

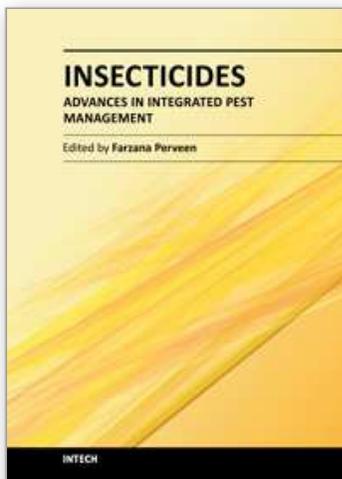
In this contribution, we discuss the interaction between a hypothetical crop pest that has a natural enemy, an insect that is a parasitoid, and also a predator of this pest. Using this host-parasitoid-prey-predator system, we discuss the use of pesticide spraying and parasitoid release to control the pest population. For each parameter set of the model, we were able to predict the time interval between successive applications of the IPM technique, and also the number of applications as a function of the economic threshold. As shown in Fig. 5, these two factors are important in determining the success or failure of the IPM methodology.

Finally, the spatial model shows how the spatial structure can affect the effectiveness of the technique. The non-spatial model always overestimates the interval between IPM applications, and also the number of applications (Fig. 7). As a rule, for the spatial model, increasing the economic threshold makes pest control more difficult, leading to an increase in the economic damage. As a future study, it will be interesting to add the influence of temperature on the entomological parameters of the insects, and also the temporal and spatial dynamics of the target crop, to analyze how these phenomena affect the IPM methodology.

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This book contains 30 Chapters divided into 5 Sections. Section A covers integrated pest management, alternative insect control strategies, ecological impact of insecticides as well as pesticides and drugs of forensic interest. Section B is dedicated to chemical control and health risks, applications for insecticides, metabolism of pesticides by human cytochrome p450, etc. Section C provides biochemical analyses of action of chlorfluazuron, pest control effects on seed yield, chemical ecology, quality control, development of ideal insecticide, insecticide resistance, etc. Section D reviews current analytical methods, electroanalysis of insecticides, insecticide activity and secondary metabolites. Section E provides data contributing to better understanding of biological control through *Bacillus sphaericus* and *B. thuringiensis*, entomopathogenic nematodes insecticides, vector-borne disease, etc. The subject matter in this book should attract the reader's concern to support rational decisions regarding the use of pesticides.

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