Mathematical modeling for sustainable aphid control in agriculture via intercropping

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Abstract

Agricultural losses to pest represent an important challenge in a global warming scenario. Intercropping is an alternative farming practice that promotes pest control without the use of chemical pesticides. Here we develop a mathematical model to study epidemic spreading and control in intercropped agricultural fields as a sustainable pest management tool for agriculture. The model combines the movement of aphids transmitting a virus in an agricultural field, the spatial distribution of plants in the intercropped field, and the presence of “trap crops” in an epidemiological Susceptible-Infected-Removed (SIR) model. Using this model we study several intercropping arrangements without and with trap crops and find a new intercropping arrangement that improves significantly pest management in agricultural fields respect to the commonly used intercrop systems.

1 Introduction

The sustainable intensification of agriculture is imperative for feeding a growing world population while minimizing its negative environmental impact. The world population will increase to between 9.6 and 12.3 billion in 2100 \cite{1}, and for feeding these additional 2-4 billion people, a duplication (100-110\%) of crop production relative to its 2005 level is needed \cite{2}. Today, 10\% of ice-free land on Earth is used for crop cultivation \cite{3}, and returning half of Earth’s terrestrial ecoregions to nature will mean global losses of 15–31\% of cropland and of 3–29\% of food calories \cite{4}. Thus, increasing crop yield without extending the size of cultivation areas nor by intensifying the use of current technologies is a vital complex problem to be solved in the coming years. Agricultural yield is substantially reduced by pests \cite{5,6,7,8,9}, which cause losses of 10-16\% to crop production \cite{5,6,7,8,9}, which may represent real threads for entire world regions \cite{10}. In addition to these scenarios, there is increasing concern that climate change can increase plant damage from pests in future decades \cite{11,12,13,14,15,16}. Bebber et al. \cite{17} have demonstrated that pests and pathogens have shifted poleward by 2.7 ± 0.8 km/yr since 1960. This will produce lower numerical response of biological control agents, which can be translated into higher probabilities of insect pest outbreaks. Deutsch et al. \cite{18} estimated that global yield losses of rice, maize and wheat grains are projected to increase in the range of 10 to 25\% per degree of global mean surface warming. Thus, in a projected scenario of 2\°C-warmer climate the mean increase in yield losses owing only to pest pressure extend to 59, 92, and 62 metric megatons per year for wheat, rice and maize, respectively \cite{18}. These losses cover most of the globe as can be seen in the Fig. 1.1(a), but they are primarily centered in temperate regions.

From the agricultural point of view, a particularly important class of insect pests are the aphids (aphididae) \cite{19}. Aphids are by far the most important transmitters of plant viruses, being reported to transmit about 50\% of insect-borne plant viruses (approximately 275 virus species). There are about 4,700 aphids described from which about 190 transmit plant viruses (see Chapter 15 of \cite{19}). From the economic point of view this virus transmission by aphid represents global losses estimated on tens of millions to billions US$ of yield loss per annum \cite{20,21,22}. In the UK alone the damage on cereals made by aphids has been estimated to be around 60-120 million pounds annually \cite{23}. Thus, mathematical modeling is seen as an important tool to predict and mitigate the effects of viruses on agriculture \cite{24,25}.

Today, there are several alternative approaches for the sustainable intensification of agriculture based on agroecological and adaptive management techniques \cite{26}. A recent work reports evidences that organic farming, for instance, promotes
pest control [27]. An example is intercropping, consisting in growing two or more crops in the same field, which has proved to be important for pest control in several crops [28, 29, 30] (see Supplementary Table 1). Intercropping is known since the 16th-18th centuries when Iroquoian farmers inter-planted the Three Sisters: corn, bean, and squash [31]. Intercropping is known to reduce the levels of infestation by stemborers and increases insect pest parasitism [32]. These practices have been extended across the globe as can be seen in Fig. 1.1 (b) [30]. Meta-analysis of 552 experiments in 45 papers published between 1998 and 2008 shown that intercropping produces significant improvement for herbivore suppression, enemy enhancement, and crop damage suppression [33] respect to monocrop. Brooker et al. [34] have concluded that intercropping “could be one route to delivering ’sustainable intensification’” of agriculture. In the particular case of aphids, there are many reports on the successful use of intercropping strategies for controlling aphid-transmitted viral diseases [35, 36, 37]. In a recent review, a series of companion plants that can be potentially used in intercropping strategies for controlling aphids have been reported, together with several strategies for controlling aphid-produced diseases [38].

Figure 1.1: (a) Global maize yield losses to insect pests due to climate change [18] (reproduced with permission). (b) Locations of intercropping experiments retrieved from the literature until 2018 [30] (reproduced with permission).

Here we develop and implement a mathematical model that allow us to study intercropping as a sustainable pest management tool for agriculture. Our main goal is to investigate which are the best spatial arrangements for controlling aphid-transmitted viruses in agricultural scenarios by avoiding the propagation of aphids through the crop field. For this purpose we combine the movement of aphids in the agricultural landscape [39, 40, 41, 42] with the spatial distribution of plants in the intercropped field, in an epidemiological Susceptible-Infected-Removed (SIR) [43] model. The model allows us to implement “trap crops”–plants which attract or detract insects to protect target crops [44, 45, 46, 47, 48]. Using this approach we find that a new intercropping arrangement proposed here–particularly when combined with trap crops–can improve significantly pest management in agricultural fields respect to the commonly used intercrop systems.
2 Theoretical Methods

For the development of the theoretical model to be used in this work we make the following assumptions:

1. The infection is transmitted to plants by an aphid–a vector. That is, a susceptible plant receives the infection, e.g., a virus, from an infectious plant through a vector.

2. Recovered (removed) plants represent those not only dead but also those which are useless for commercial purposes, i.e., those substantially damaged as to be used for consumption.

3. The number of plants in the field is fixed.

4. When a susceptible vector is infected by a plant, there is a fixed time \( \tau \) during which the infectious agent develops in the vector. At the end of this time, the vector can transmit the virus to a susceptible plant.

5. The number of infectious vectors is very large and at a given time \( t \) its amount is proportional to \( I(t - \tau) \).

These assumptions are an adaptation of the ones made by Cooke [49] for implementing a time-delay Susceptible-Infected-Recovered (SIR) model to study a vector-borne infection transmission to a given population. The corresponding equations read as follows:

\[
\begin{align*}
\dot{S}_i(t) &= -\beta S_i(t) I_j(t - \tau) + \mu I_i(t), \\
\dot{I}_i(t) &= \beta S_i(t) I_j(t - \tau) - \mu I_i(t), \\
\dot{R}_i(t) &= \mu I_i(t),
\end{align*}
\]

(2.1)

where \( S_i \) is the probability of plant \( i \) of being susceptible to the infection, \( I_i \) is the probability of plant \( i \) of being infective after having been infected by the disease, and \( R_i \) is the probability of plant \( i \) of being removed, \( \beta \) and \( \mu \), are the birth and death rates of the disease, respectively. This model has been subsequently studied in the literature by several authors as a vector-borne disease transmission model (see for instance [50, 51, 52, 53]). For other approaches to modeling vector-borne virus transmission on plants see for instance [54].

Here we generalize Cooke’s model [49] in order to account for the probability that a vector hops not only to a neighboring plant but also to a more distant one in the field:

\[
\begin{align*}
\dot{S}_i(t) &= -\beta S_i(t) \sum_j f_{ij} I_j(t - \tau) + \mu I_i(t), \\
\dot{I}_i(t) &= \beta S_i(t) \sum_j f_{ij} I_j(t - \tau) - \mu I_i(t), \\
\dot{R}_i(t) &= \mu I_i(t),
\end{align*}
\]

(2.2)

(2.3)

(2.4)

where \( f_{ij} \) is a function of the “separation” between the plants \( i \) and \( j \). There are two possibilities of accounting for this separation between plants. The first is to consider the Euclidean distance between the corresponding two plants, i.e.,

\[
\rho_{ij} = \sqrt{(x_i - x_j)^2 + (y_i - y_j)^2},
\]

where \( x_i \) and \( y_i \) are the Cartesian coordinates of the plant \( i \) in the plane. Notice that this distance is not capturing all the subtleties of the real separation between the plants as two plants can be of different high, and a third coordinate should be introduced. In this case we can consider that the probability of moving from plant \( i \) to plant \( j \) is proportional to certain function of this distance, e.g., decaying as a power-law \( \pi_{ij} \propto \rho_{ij}^{-s} \) or decaying exponentially \( \pi_{ij} \propto \exp(-\lambda \rho_{ij}) \), where \( s, \lambda \in \mathbb{R}^+ \).

The second approach is to consider the plant-to-plant separation in terms of the number of hops that an aphid needs to take to go from plant \( i \) to plant \( j \) using other intermediate plants. That is, let us consider that the aphid in question has an exploration radius equal to \( r \). This means that if the aphid is on plant \( i \) it can hops directly to a plant which is at a distance \( r \) from \( i \). In order to hop to a plant \( k \) separated by two radii from \( i \) it has to use two steps. That is, if we connect two plants by an edge if their geographic separation is \( \rho_{ij} \leq r \), then the plant-to-plant (topological) separation \( d_{ij} \) is given by the number of edges in the shortest path connecting the two nodes in the resulting graph \( G = (V,E) \). In this case we again can consider that the probability of moving from plant \( i \) to plant \( j \) is proportional to certain function of this distance, e.g., decaying as a power-law \( p_{ij} \propto d_{ij}^{-s} \) or decaying exponentially \( p_{ij} \propto \exp(-\lambda d_{ij}) \), where \( s, \lambda \in \mathbb{R}^+ \).

Let us consider some of the potential differences between these two ways of accounting for the interplant separation.
2.1 Through-space vs. plant-to-plant aphid mobility

Aphids have been found to have two different kinds of movements. The origin-destination movement of an aphid takes place either through ‘inadvertent’ or ‘intentional’ displacement (see Chapter 10 in [19]). The first is an involuntary act, such that when it is propelled by the force of impact, gravity, air currents, or a combination of these, or it can be transported by animals, farm machinery, automobiles, or aircraft. The second is a voluntary act typically governed by reactions to sensed stimuli in the environment. This voluntary movement includes as an important case the displacement of aphids in exploratory search for food. In this case visual, tactile and olphatory cues are determinant for approaching and landing on a plant. For instance, the following events have been identified in the exploratory movements of aphids [58]: (i) pre-alignment before landing, (ii) plant contact and assessment of surface cues after landing, (iii) probing on superficial tissues, (iv) location and insertion of stylets at the appropriate feeding site, (v) salivation followed by committed sap ingestion. It is important to notice that although a plant can be selected for landing by an aphid due to its attractiveness, it may or may not be potential host for that aphid, and that the discrimination appears after landing and probing on different plants [56]. Consequently, the exploratory movement of aphids looks like a multi-hopping process in which they can simply hop to a neighboring plant, can hop directly to a more distant one after visual/olphatory inspection of neighboring plants, or can land a neighboring plant without testing it and move to a more distant one after it. All these factors makes the hopping process of aphid vectors a nontrivial one and here we propose a way of capturing some of these nontrivialities into a model for epidemic spreading on plants.

Here we are focused only on this exploratory movement of aphids inside a crop field, which include mainly displacements to neighboring plant (primary movement) or a distant plant inside the same field. We exclude from here those unintentional movements of aphids such that the displacement by air currents and that can transport them at very long geographic distances. Thus, with this restriction in mind we analyze the main differences in considering a model that includes geographic or topological distance for epidemic transmission. In doing so, we have identified three main factors in favor of the use of the topological interplant separation which are based on the main behavioral characteristics of aphids exploratory movement inside crop fields [58, 59, 60, 61]. In formulating these rules we consider that two plants $i$ and $j$ are separated by a geographic (Euclidean) distance equal to $\rho_{ij}$. We consider that the aphid has a radius of primary exploration equal to $r$. That is, the aphid can hop directly from the plant $i$ to any other plant in a radius $r$ from $i$. Then, if two plants are at a distance $\rho_{ij} \leq r$ we consider that they are topologically connected to each other by an edge. The number of edges in the shortest path connecting two plants $i$ and $k$ (not directly connected to each other) is the topological distance $d_{ik}$.

We consider here two different kinds of hopping probabilities. The geographic hopping probability $\pi_{ij}$ depends on the geographic distance between the two plants, e.g., $\pi_{ij} \propto \rho_{ij}^{-s}$. The topological hopping probability depends only on the topological distance separating the two plants, and not on its geographic separation, e.g., $p_{ij} \propto d_{ij}^{-s}$.

First come first served. Consider an aphid at a plant $i$ which can hop to any of the adjacent plants $j$ (left) or $k$ (right) (see Fig. 2.1a)). Let us consider that the geographic distance between the plants are $\rho_{ij} < r$ and $\rho_{ik} < r$, respectively, such that $\rho_{ij} > \rho_{ik}$. Then, according to the geographic distance, the probability of the aphid hopping to plant $k$ is larger than that of hopping to plant $j$, i.e., $\pi_{ij} \propto \rho_{ij}^{-s}$ and $\pi_{ik} \propto \rho_{ik}^{-s}$ assuming a power-law decay with the distance, thus $\pi_{ij} < \pi_{ik}$. However, as the plants $j$ and $k$ are inside the radius of primary movement of the aphid, an aphid hopping from $i$ to the right will find first the plant $k$, exactly the same as an aphid hopping to the left who will find first the plant $j$. Thus, both hopping processes should display the same probabilities, which is accounted for by the topological distance between the pairs of plants. Because ($i,j$) are nearest neighbors as well as ($i,k$), we have that $d_{ij} = d_{ik} = 1$, where $d_{ij}$ is the topological (shortest path) distance. Consequently, $p_{ij} = p_{ik}$. 

A bird in the hand is worth than two in the bush. Consider an aphid at a plant $i$ which can hop either to plants on its left or on its right (see Fig. 2.1 (b)). Let us consider that the geographic distances between the plants are $\rho_{ij} = \rho_{ik} > r$. Then, according to the geographic distance, the probability of the aphid to hop to plant $k$ is exactly the same as that of hopping to plant $j$, i.e., $\pi_{ij} = \pi_{ik} \propto \rho_{ij}^{-s}$ assuming a power-law decay with the distance. However, an aphid moving from $i$ to the left will find first a plant in its way to $j$. Thus, assuming that such plant is attractive to it, the aphid will explore first that plant on the basis of a minimum effort principle and then the plant $j$. On the other hand, an aphid moving from $i$ to the right will find first a plant that it can explore and in case it decides to continue its movement to the right, the aphid will find yet another plant before arriving at $k$. Thus, it is clear that under the same conditions the probability of arriving at the plant $k$ is smaller than that of arriving at $j$, although they are at exactly the same geographic distances. Assuming the connectivity of the plants given in Fig. 2.1 (b) we have that $2^{-s} \approx p_{ij} > p_{ik} \propto 3^{-s}$.

Go back before it is too late. Consider an aphid having an exploratory movement at the borderline of a crop field from a node $i$ (see Fig. 2.1 (c)). If the aphid is moving away from the field there is a high probability that it overpass its exploratory radius before finding a new plant. Thus, it is probable that the aphid returns to plant $i$ before finding any new one. As a consequence, the probability that the aphid arrives at a plant distant from $i$ depends more on the topological separation among the plants than on the geographic distance through the “possibly empty” space separating them. That is, we consider here that an aphid navigates a crop field by orienting itself through the plants and not realizing “risky” explorations outside the field.

2.2 SIR model with topological distances

As a consequence of the previous hypothesis we conclude that the use of the topological interplant separation is appropriate for our modeling purposes. Therefore, the SIR model on the field is expressed as [66]:

$$\dot{S}_i (t) = -\beta S_i (t) \sum_j A_{ij} I_j (t - \tau) + \mu I_i (t), \quad (2.5)$$

$$\dot{I}_i (t) = \beta S_i (t) \sum_j A_{ij} I_j (t - \tau) - \mu I_i (t), \quad (2.6)$$

$$\dot{R}_i (t) = \mu I_i (t), \quad (2.7)$$

where $A = \sum_{d=1}^{d_{max}} d^{-s} A_d$, $d \leq d_{max}$, $d_{max}$ is the largest separation between two plants (in terms of steps) and the matrix $A_d$ captures the (long-range) mobility of the pest between plants (see Fig. 2.2), and it is formally defined in the next sub-subsection.
Figure 2.2: Inter-plants movements of an aphid in an agricultural plot with intercropping (see Supplementary Note 1). The hop of an aphid from an infected plant to a susceptible one separated by $d$ steps is given by $d^{-s}$ (see Supplementary Note 1).

Notice that the transformed adjacency matrix $\tilde{A}$ is symmetric in the case of undirected networks. Then, when the aphid has very poor mobility $s \to \infty$ all the entries of $\tilde{A}$, except those equal to one, become zeroes, which indicates that the aphid can only perform hops to nearest neighbors. On the other hand, when the aphid has a very large mobility $s \to 0$, every entry of $\tilde{A}$ becomes one, which means that the aphid can hop from one plant to another with equal probability independently of their separation in the field.

2.2.1 $d$-path adjacency operators

More formally, $\tilde{A}$ is a transformed adjacency operator on a graph, which will be defined as follows. Let us consider $\Gamma = (V,E)$ to be an undirected finite or infinite graph with vertices $V$ and edges $E$. We assume that $\Gamma$ is connected and locally finite (i.e. each vertex has only finitely many edges emanating from it). Let $d$ be the shortest path distance metric on $\Gamma$, i.e. $d(v,w)$ is the length of the shortest path from $v$ to $w$. Let $\ell^2(V)$ be the Hilbert space of square-summable functions on $V$ with inner product

$$\langle f, g \rangle = \sum_{v \in V} f(v)\overline{g(v)}, \quad f, g \in \ell^2(V).$$

In $\ell^2(V)$ there is a standard orthonormal basis consisting of the vectors $e_v$, $v \in V$, where

$$e_v(w) = \begin{cases} 1 & \text{if } w = v, \\ 0 & \text{otherwise.} \end{cases}$$

For $d \in \mathbb{N}$ the following operator defined in $\ell^2(V)$ is the $d$-path adjacency operator of the graph

$$(A_d f)(v) := \sum_{w \in V: d(v,w)=d} (f(w)), \quad f \in \ell^2(V), v \in V$$

The $d$-path adjacency operator acts over the vectors $e_v$ as

$$(A_d e_v)(w) = \begin{cases} 1 & \text{if } d(v,w) = d, \\ 0 & \text{otherwise.} \end{cases}$$

These operators are the adjacency analogues of the $d$-path Laplacian operators of the graph [62, 63, 64]. The Mellin (power-law) transformed adjacency operator is then defined by

$$\tilde{A} := \sum_{d=1}^{d_{max}} d^{-s} A_d.$$  

Other transforms are also possible as the Laplace (exponential) one (see for instance [62, 63, 64] for the analogues in the path Laplacians), but we constraint ourselves here to the power-law one.
2.3 Markovian formulation of the epidemiological model.

Following the framework introduced in [69], we formulate a Markovian evolutionary equation that, in principle, is valid for any epidemic prevalence. Let $p_i(t)$ be the probability that a node $i$ is infected at time $t$. Then, in the SIR model, the Markovian equations reads as follows:

\begin{align*}
p_i(t+1) &= p_i(t)(1 - \mu) + (1 - p_i(t) - \rho_i(t))q_i(t - \tau), \\
\rho_i(t+1) &= \rho_i(t) + \mu p_i(t),
\end{align*}  

(2.13) (2.14)

where $\rho_i(t)$ is the probability that node $i$ is removed at time $t$. The expression for the infestation probability $q_i(t - \tau)$ is

\[ q_i(t - \tau) = 1 - \prod_{j=1}^{N} [1 - \beta \tilde{A}_{ij} p_j(t - \tau)], \]

(2.15)

which represents the probability that, when node $i$ is healthy at time $t$, it becomes infected at time $t+1$. The expression $q_i$ is calculated as 1 minus the probability that the node $i$ is not infected by any infectious contact. This last probability is the product over all the possible contacts of node $i$, considering that a node $j$ transmits the disease to $i$ with probability $\beta \tilde{A}_{ij} p_j$, after the delay time $\tau$. Note that if node $j$ is not connected to $i$, $\tilde{A}_{ij} = 0$, then the corresponding term in the product is equal to 1, since $j$ cannot infect $i$ regardless of its state, $p_j(t - \tau)$.

We should notice that these Markovian formulation holds for any disease incidence, while Eqs. (2.15) and (2.16) are only valid when the disease prevalence is small. To explain this, take Eq. (2.15) for $q_i(t - \tau)$ and consider that the prevalence is small, $p_i \ll 1 \forall i$, and for this reason let us denote $p_i = x_i$. Then, the product in (2.15) transform into: $1 - \sum_{j=1}^{N} \beta \tilde{A}_{ij} x_j$, the new expression for $q_i(t)$ in Eq. (2.13), and passing from discrete to continuous time, we recover a similar expression to that in Eq. (2.6) for the evolution of the infected state of node $i$. For more details the reader is referred to [70].

The rate of propagation of the aphid-borne viral infection across an agricultural field is defined here as

\[ v = \frac{\text{Number of susceptible plants that become removed at time } t}{t}. \]

(2.16)

Here we select the time $t$ in which the steady state of the dynamics is reached.

2.4 Computational arrangements

2.4.1 Intercropping arrangements.

The intercropped systems considered here and shown in Fig. 2.3 are: the strip intercropping in which strips of the main cultivar are inserted between strips of the secondary crop; row intercropping in which rows of the main and secondary crops are alternated one-by-one; column intercropping, the same as before but by columns instead of by rows; chessboard intercropping in which a plant of the main crop is inserted in the rows and columns between every two susceptible plants; patches intercropping in which squared patches of the main crop are alternated with squared patches (of the same size) of the secondary crop; random intercropping in which plants of the secondary crop are randomly inserted among those of the main crop. The first two intercropping arrangements—strips [48, 71] and rows [72, 73, 74]—are frequently used in experimental designs and field applications. It is important to remark that in all cases we have considered exactly the same amount of plants of the main crop such that the results obtained here are not due to size effects.
2.4.2 Networks construction.

Our arrangements consist of rectangular plots of lengths \( a \) and \( a^{-1} \). These plots guarantee that all simulations are carried out on fields of equal area. The rectangular plots has been shown—both theoretically and experimentally—to delay more the propagation of epidemics than square plots with the same area and density of plants [75]. We consider the distribution of a major crop intercropped with a secondary crop, which may or may not be a trap crop. In the intercropped field we maintain a separation between plants equal to \( \Delta \) (see Fig. 2.4). In this case the plant-to-plant connectivity, based on their separation, is represented by a squared partition of the plot. We simply normalize all the distances by dividing them by \( \Delta \). Then, two plants which are nearest neighbors are one step apart, a second nearest neighbor is two steps apart and so forth. In general, every plot consists of 20 rows and 50 columns. There is a plant at every intersection for a total of 1000 plants. As we have a unit rectangle with \( a = 1.6059 \), the value of \( \Delta \) is 0.033, and we use a connection radius \( r = \Delta \), such that the plants are adjacent (connected in the network) only to those immediately to the left, right, up and down. In the case of the intercropped systems we always replaced 500 plants of the main crop by the same quantity of plants of the secondary crop. In the Supplementary Note 2 we analyze the case in which the separation between rows and columns in the plot are smaller than \( \Delta \), which is equivalent to consider the radius of primary movement of the aphid equal to \( r = \sqrt{2}\Delta \).
2.4.3 Implementation of the “trap crops”.

Although trap crops can be formed either by “push” crops or by the combination of “push-pull” crops \[44, 45, 46, 47\], here for the modeling purpose we combine all the trap crop effects into a single one. Basically we consider that trap crop diminishes or completely avoids the propagation of a pest in a path beyond the place in which the trap is located. Consequently, if there are more than one trap in the path between two susceptible plants we only consider the effect of one of them. An additive or multiplicative effect of the traps can be easily implemented using the current mathematical framework (see further), but it is not done here for the sake of simplicity. In this case the secondary crop is located between the paths connecting the infected and the susceptible plants. Mathematically, let us consider two plants \(i_1\) and \(i_{d+1}\), and the shortest-path \(i_1, i_2, \ldots, i_d, i_{d+1}\) of length \(d\) between them. To model trap crops, we modify the strength of the long-range mobility of the aphid between \(i_1\) and \(i_{d+1}\) as follows:

\[
\begin{cases} 
\tilde{A}(\Delta)_{i_1, i_{d+1}} = \frac{d^{-\gamma s}}{d^{-s}} & \text{if there is at least one trap crop between } i_1 \text{ and } i_{d+1} \\
\frac{d^{-s}}{d^{-s}} & \text{otherwise} 
\end{cases}
\] (2.17)

where \(\gamma \geq 1\) is the trap strength. When \(\gamma = 1\), there is no trap crop as we recover the original equation for the epidemic dynamics with long-range movements. On the other hand, when \(1 < \gamma < \infty\), movement of the aphid is reduced beyond the point in which the trap is located. For instance, when the trap crop is very effective, i.e., \(\gamma \to \infty\), the movement of the aphid from \(i_1\) to \(i_{d+1}\) is completely interrupted which means that the trap is perfect. In the Fig. 2.5(b) we illustrate the effects of a secondary crop in which we obtained the probability \(q_i\) that the plants in the right part are infected once the three plants on the left side are infected by the pest. Supposing \(\tau = 0\) (no delay), when \(\gamma = 1\) (no trap), the infectability of the susceptible plants is 24.2%, which represents the effects of an intercropped secondary species. However, when the strength of the trap is \(\gamma = 2\), the probability that the susceptible plants are infected drops to less than 5%. This probability is reduced to zero as \(\gamma\) is subsequently increased.
2.4.4 Simulations.

Using the Markovian formalism we perform 100 random realizations for each field arrangement, secondary crop (with or without trap) and aphid mobility (fast and slow). The propagation is initialized by infecting randomly a single susceptible plant on the border of the field. Following [66], we set here $\mu = 0.5$, since we are not trying to characterize any particular disease. For $\mu = 1$, for instance, the recovery is too fast to see the spatial propagation and, conversely, in the case $\mu = 0$ the dynamics would be an SI dynamics. We decided to lie between these two limiting cases.

For the evolution of the disease we calculate the total amount of Markovian time in which the probability of being susceptible is larger than the probability of being removed (i.e., $1 - q_i - p_i > q_i$), for each susceptible plant. To estimate the epidemic thresholds, we calculate the average stationary fraction of removed plants (over 10 realizations), $R(\beta, \mu)$, for 50 logarithmically spaced values of $\beta$, between 0.02 and 1.0, when $\mu = 0.5$. Then, using a linear interpolation, we find the epidemic threshold $\tau_E$ in each field. We recall that the epidemic threshold is the smallest value of $\beta/\mu$ for each arrangement that satisfies the condition that $R(\beta, 0.5) > 0$. Visualization of results in the form of rain clouds were performed using Matlab® codes available from Allen et al. [76].

3 Results and discussion

3.1 Influence of time-delay

According to the results previously reported by Tchuenche and Nwagwo [52], the effects of the time delay $\tau$ are mainly observed at the initial times of the propagation dynamics and are focused on the population of susceptible plants. For relatively large time the evolution of the SIR dynamics with and without time delay are almost indistinguishable (see Fig. 2 in [52]). We explore here the effects of $\tau$ on the epidemic dynamics when the vector mobility is incorporated into the model. Using the Markovian formulation described previously in this work we obtained the evolution of the infected population of plants in crop field consisting of a square lattice as described before for two different values of the aphid mobility $s$ in the Mellin transformed SIR equations. The results are illustrated in Fig. 3.1 were we have used $\beta = 0.5$, $\mu = 0.5$, $r = \Delta$ and $s = 2.5$ (a) and $s = 1.0$ (b). It can be seen that the inclusion of a time delay in the model makes that the peak in the number of infected plants is displaced to longer times. For large aphid mobility ($s = 1.0$) it is observed
that the shapes of the peaks of infection are very similar to each other for different values of the time delay $0 \leq \tau \leq 10$. When the mobility of the aphids is relatively low ($s = 2.5$) the rate of propagation of the infection changes significantly for different values of $\tau$, particularly for very large time delays. For instance the values of $v(\tau)$ are as follow: $v(0) = 32.25$, $v(1) = 26.32$, $v(2) = 22.22$, $v(3) = 18.87$, $v(4) = 16.67$, $v(5) = 14.70$, $v(10) = 9.17$. However, for the case of large aphid mobility these rates of propagation are not changed significantly with the time delay: $v(0) = 43.48$, $v(1) = 40.00$, $v(2) = 38.46$, $v(3) = 35.71$, $v(4) = 34.48$, $v(5) = 33.33$, $v(10) = 27.78$. That is, for relatively low time delays the results in the disease propagation on plants are very similar to those without time-delays. Also, when the the aphid mobility is relatively large, the time delay does not affect significantly the propagation rate of the disease.

As a consequence of the previous analysis and for the sake of keeping our model as simple as possible we are not considering explicitly the time delay in the further calculations in this work. The biological justification for this simplification is as follows. The interaction of the virus and aphid is controlled by the following phases (see Chapter 15 of [19]): (i) acquisition, where the aphid takes up virions from an infected plant, (ii) retention, where the aphid carries the virions at specific sites, (iii) latency, which refers to the inability of an aphid to inoculate immediately a virus following acquisition, and (iv) inoculation, which is the release of retained virions into the tissues of a susceptible plant. There are three types of transmission of a virus to a plant (see Chapter 15 of [19]). In the non-persistent (NP) transmission, the acquisition and inoculation are very fast and requires only a very brief stylet penetration, which delays less than one minute. In this case there is no latency period and the whole cycle of transmission can be completed within a few minutes.

In the semi-persistent (SP) transmission, the acquisition and inoculation requires periods of about 15 minutes. In this case there is no latency periods either and the aphids retain the ability to inoculate for periods of up to 2 days following acquisition. Finally, in the persistent (P) transmission the virus acquisition requires period between hours to days, there is a latency period and the retention is for days to weeks. From the about 270 viruses transmitted by aphids more than 200 are transmitted by NP transmission (see Chapter 15 of [19]). The results to be considered here using a SIR model without time delays is then equivalent to model the aphid-borne transmission of viruses to plants using either NP or SP transmission.

### 3.2 Impact of intercrop arrangements on virus propagation.

In Fig. 3.2 we illustrate the results of the simulations of the propagation of an aphid-borne virus in the 6 intercropped fields without traps $(\gamma = 1.0)$ studied here as well as in the monocrop. In Fig. 3.2 (a) we show the results for an aphid with relatively low mobility $(s = 4.0)$ and in Fig. 3.2 (b) we give the same for a relatively high mobility aphid $(s = 2.5)$. In the case in which $s = 4.0$ it is clear that the disease is propagated in a relatively slow fashion and for $t = 10$ only 18.3% of plants are removed in the monocrop. As can be seen in this figure all intercrop arrangements produce significant decrease in the number of removed plants. The smallest decay in the number of removed plants is observed for the patches configuration in which the percentage of removed plants is 10.1%, followed by the strips configuration with 6.6%. On the

![Figure 3.1: Evolution of the number of infected plants in a square plot with the variation of the time delays $\tau$. The modeling is performed with $\beta = 0.5$, $\mu = 0.5$, $r = \Delta$ and $s = 2.5$ (a) and $s = 1.0$ (b).](image-url)
other hand, the most efficient arrangement is the chessboard one, which reduces the number of removed plants practically to zero (only 0.3% of removed plants).

In Fig. 3.2 (b) we illustrate the results for the case in which the pest has a relatively large mobility. Here the picture observed is significantly different from the one in the previous case. First, the level of plants removed in the monocrop is 95.1%, indicating an almost complete destruction of the crop in a relatively short time \( t = 10 \) when the pest is highly mobile. The range of amelioration of the infection across the fields is here very wide, ranging from the 10% of decrease in removed plants observed for the patches arrangement (85.5% of removed plants) up to about 80% of decrease obtained with the chessboard arrangement (16.3% of removed plants). Notice that the frequently used intercrop arrangement of strips produces, together with that of patches, the smallest improvement in the number of removed plants. Thus, although the results are quantitatively very different for the cases of low and high mobility of the aphid, they are qualitatively similar in identifying the worse arrangements (patches and strips) as well as the best one (chessboard). In both cases the order of effectivity in reducing the impact of an aphid-borne virus propagation is: chessboard > columns > random > rows > strips > patches.

In Fig. 3.2 (c) and (d) we illustrate a snapshot of the aphid-borne propagation of a virus across the different intercropping systems with \( s = 4.0 \) and \( s = 2.5 \), respectively. In order to compare all the different arrangements we always start the epidemic by infecting the same node, i.e., the one at the bottom-right corner of the field. The colors in the plots represent the time in which the plant remains susceptible without becoming removed by the vector-borne virus disease. That is, a low value of this time indicates that the plant is removed relatively soon by the virus disease. In order to interpret quantitatively the results in these plots we use the rate \( v \) of propagation of the aphid-borne virus previously defined. It can be seen that in the monocrop the epidemic is propagated in a wave-like way, typical of diffusion processes. The values of \( v \) in the monocrop are 23.26 (\( s = 4.0 \)) and 32.26 (\( s = 2.5 \)). That is, when the aphid has relatively low mobility there is an infection of 23.26 plants per unit time. This rate is increased to 32.26 plants when the pest mobility is increased, due to the fact that the aphids can now hop to wider regions of the plots. Reminiscences of the wave-like kind of propagation of the vector-borne virus are observed in all the intercrop arrangements studied. In the intercropped systems (without trap crops, \( \gamma = 1.0 \)) the propagation rates of the virus are: for \( s = 4.0 \), chessboard (0.03) < random (5.46) < columns (7.35) = rows (7.35) < strips (10.0) < patches (11.36); for \( s = 2.5 \), chessboard (9.62) < random (12.19) < columns (13.16) = rows (13.16) < strips (14.70) < patches (15.62). In closing, the chessboard arrangement is significantly better in reducing the propagation of aphid-borne viruses in agricultural fields than the rest of the arrangements when there are no trap crops in the intercrop. The random arrangement also performs very well in terms of both the number of plants removed by the infection and the rate of propagation of the epidemic. See Supplementary Note 2 for the case when the separation between rows and columns is smaller than here, i.e., when the radius for primary dispersal of the aphid is \( r = \sqrt{2} \Delta \) instead of \( r = \Delta \).
3.3 Impact of intercrops with trap crop on aphid-borne virus propagation.

We now move to the analysis of the intercrop systems with trap crops. To have an idea of the many systems in which the current results can be applied the reader is referred to the Tables 1 and 2 in Hokkanen’s paper [44], where many examples of one main crop intercropped with a trap crop are given. We consider here the existence of trap crops which are not perfect, i.e., they allow certain propagation of the aphid-borne viral infection (see Supplementary Note 3 for results with a perfect trap). Thus, we use $\gamma = 2.0$ and analyze the cases of relatively low ($s = 4.0$) and relatively large ($s = 2.5$) aphid...
mobility. In Fig. 3.3 we illustrate the results of our simulations for these systems using the different arrangements studied here. As can be seen for the case of relatively low mobility ($s = 4.0$) there are significant reductions in the percentages of removed plants for all intercrop systems. The percentages of removed plants for each intercrop are: chessboard (0.2%), columns (1.4%), random (1.5%), rows (1.8%), strips (3.4%) and patches (4.7%). We remind the reader that the percentage of removed plants in the monocrop is 18.1%. When the pest has a relatively large mobility ($s = 2.5$), 95.1% of plants are removed in the monocrop, while in each of the intercrops they are: chessboard (0.2%), random (2.8%), columns (4.4%), rows (6.3%), patches (9.1%), and strips (17.4%). Notice that here there are some important changes in the order of the arrangements in terms of their effectiveness in reducing the propagation of the infection. When the aphid is of high mobility the best arrangements are the chessboard and the random one. The worse arrangement, and the only one having more than 10% of removed plants, is the strip one. Also notice that the percentage of removed plants in the chessboard arrangement is exactly the same for $s = 2.5$ and $s = 4.0$, indicating a high stability in the efficiency of this arrangement.

We now analyze the rate of propagation of the aphid-borne virus across the agricultural fields intercropped with a trap crop (see Fig. 3.3 (c) and (d)). The rate of propagation of the virus follow a different order as for the case of intercrops without traps ($\gamma = 1.0$). That is, for $s = 4.0$, we find: chessboard (0.05) < random (1.67) < columns (4.59) < rows (4.67) < strips (7.04) < patches (7.94). For $s = 2.5$, chessboard (0.04) < random (4.18) < columns (7.58) < rows (8.06) < strips (10.87) < patches (11.63). Here again there is a significantly high improvement, in terms of diminishing the impact and the rate of propagation of a virus across an agricultural field, when the chessboard arrangement is used. See Supplementary Note 2 for the case when the separation between rows and columns is smaller than here, i.e., when the radius for primary dispersal of the aphid is $r = \sqrt{2}\Delta$ instead of $r = \Delta$. These results agree with those previously reported using a stochastic simulation model \[67\].
Figure 3.3: **Aphid-borne virus propagation on intercropped fields with trap crops.** Results of the simulations for a SIR epidemics at $t = 10$ with $r = \Delta$, $\beta = 0.5$, $\mu = 0.5$ for different intercropping strategies with trap crops of strength $\gamma = 2.0$. (a) Raincloud plot of the proportion of dead plants for a viral infection propagated by aphids with reduced mobility ($s = 4.0$). Raincloud plot of the proportion of dead plants for a viral infection propagated by aphids with relatively high mobility ($s = 2.5$). The clouds show the kernel distribution of the proportion of dead plants for different realizations of the epidemics. Below, the raw data is plotted (the rain) together with their corresponding box and whisker plots. (c) Evolution of the propagation of a relatively low mobility pest ($s = 4.0$) across the fields. (d) Evolution of the propagation of a relatively high mobility pest ($s = 2.5$) across the fields. In both cases the propagation is initialized by infecting the plant on the bottom-right corner of the plot.

### 3.4 Epidemic thresholds.

Finally we study the ratio $\beta/\mu$, which drives the spreading of the disease. Depending on the infectious power of the aphid-borne virus there are two possible distinguishable phases for a given strength of the trap crop, $\gamma$, and of the pest mobility, $s$. The first one is an absorbing phase where the spreading of the virus is not efficient enough to reach a large fraction of the system and the propagation is absorbed, meaning that it does not progress across the field. The second phase is an
active one, where the propagation of the virus reaches a macroscopic fraction of the agricultural field. The transition from the absorbing to the active phase strictly resembles a non-equilibrium second order phase transition in statistical physics\cite{68}. The critical value of this transition \( \beta \mu_c = \tau_E \) is defined as the \textit{epidemic threshold}. This term is also known as the \textit{basic reproduction number} and it represents a threshold in the sense that below this point the propagation of the infection dies out and over it the propagation becomes an epidemic. We have then investigated the epidemic threshold \( \tau_E \) for the monocrop and the six intercrop arrangements without \( (\gamma = 1.0) \) and with a trap crop \( (\gamma = 2.0) \). We also considered, as before, two kinds of aphid, one with relatively low mobility \( (s = 4.0) \) and the other with higher mobility \( (s = 2.5) \). If Fig. 3.4 we resume the results. Let us first consider the intercropped fields without trap crops \( (\gamma = 1.0) \). Then, when the pest has low mobility the epidemic threshold of the chessboard arrangement is more than 10 times higher than that of the monocrop. Notice that we have normalized all the bar plots in the insets of Fig. 3.4 by dividing the epidemic thresholds by that of the monocrop. Indeed, we have proved in the Supplementary Note 4 that the chessboard arrangement can reach an infinitely large epidemic threshold if \( s \) is bounded and the trap crop has a very high strength. The rest of the arrangements have epidemic thresholds which are about twice that of the monocrop. When the aphid mobility increases, the epidemic thresholds logically drop, due to the fact that it is easier for the pest to trigger the propagation of a virus across the field. In this case the chessboard arrangement triplicates the epidemic threshold of the monocrop, while the rest of the arrangements have values of about 2 times larger than the one of the monocrop. When we incorporate trap crops \( (\gamma = 2.0) \) in the intercrop arrangements the changes in the epidemic threshold results very dramatic for the case of the chessboard arrangement. In this case, with low and high mobility, the epidemic thresholds are about 40 and 32 times higher than that of the monocrop. For the rest of the intercropped systems the threshold increases by factors between 2 and 5. It is interesting that for the rest of the intercrop systems the ordering of the epidemic thresholds vary from one scenario to another. For instance, without trap crop and low mobility of the pest, the random arrangement is the second best, followed by the rows arrangement. However, if the aphid has larger mobility the column arrangement is the second best followed by the rows one. When there are trap crops and low mobility of the pest the rows arrangement is the second best followed by the columns one. If the mobility of the pest is higher then the column is the second best followed by the strips one. It is possible that the empirical observation that the rows and strips arrangement delay the propagation of an aphid-borne virus in a crop field has made that these two arrangements have been the most widely used ones. However, in terms of (i) percentage of plants removed by the infection, (ii) rate of the propagation of the aphid-borne virus across the field, and (iii) epidemic threshold, the chessboard arrangement introduced here is by far the most efficient one and stable intercrop arrangement without and with trap crops.
Conclusion

Here, we demonstrate using intensive mathematical modeling, that the efficiency of intercropping arrangements can be improved dramatically in relation to the designs currently in use. We develop a mathematical framework that allow to study the effect of intercropping systems with and without ‘trap crops’. Our study shows that improving existing intercrop designs may decrease up to 80% the number of plants affected by aphid-borne viruses, slow down the propagation of such aphid-borne viruses by a 300-fold factor, and delay the triggering of these epidemics on plants by a 40-fold factor respect to a monocrop.
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