Spatiotemporal modeling of a spread of Apple Scab

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1 Introduction

Apple scab is one of the main disease present in apple orchards ([4], [6]). Caused by the ascomycete Venturia inaequalis, it attacks the leaves, flowers and fruits, and manifests as dull black or grey-brown lesions. In apple production, fruits affected are excluded from the sale and the loss can reach up to 100%.

This disease spreads in two different ways and in two phases :

– In the first one, called "phase of primary infections", during rainy periods in the spring, spores (ascospores) are projected mechanically from the dead leaves on the ground, then transported by the wind. These spores are derived from the phase of sexual reproduction that occurs during the winter. Their maturation is very gradual, and screenings are held as mature ascospores continue to be produced (March to June).
– In the second phase, called "of secondary contaminations", which occurs when the first scab lesions appear on leaves or fruits, the disease spreads through asexual spores (conidia) produced from these lesions throughout the summer.

The two phases of contamination overlap during some weeks in spring. In both cases, contamination can occur if the spores fall on sensitive organs (leaves or fruit) and if the weather conditions (temperature and wetness duration) are favorable to disease ([12]).

The disease control is currently done to contain the epidemic, essentially by using repeated applications of fungicides during the period of primary infections. However, these practices are changing in the current context of sustainable development, which imposes limits on the use of chemical fungicides by 2018. There are alternatives to chemical control. The most widely used today is the destruction of the leaf litter ([9]). The varietal resistance is also an interesting alternative ([3], 2008). However, most commercial varieties resistant to apple scab have the same gene Vf. This gene has the advantage of giving a total resistance, but it is bypassed for several years in Europe by virulent strains ([13], 1993) and particularly in the north-western of France since 1995 ([14], 2006). In areas where this resistance gene is circumvented, the resistance becomes ineffective, and varieties with Vf behave as susceptible varieties.

To address this problem of circumvention, new resistance factors are under selection in breeding programs of apple, and should result in the future to create new varieties resistant to scab. The effectiveness and sustainability of these new resistance should be enhanced by the simultaneous deployment of several varieties resistant to the scale of the orchard. Indeed, the varietal mixtures (mixed cultivars with different levels of susceptibility in the same orchard) can significantly reduce the impact of the disease ([6], 2007), their effectiveness depending on the mode of dispersal of the pathogen ([7], 1999).

In this context, an experiment was conducted at the research center INRA Angers, with the aim to study the deployment of space-time virulent strains on the Ariane range (Vf gene) from the first outbreak of scab observed in 2004 in this orchard. The goal is to determine the way the spread occurs : Across the orchard from place to place by diffusion or phenomena of dispersion over larger distances, which are two modes of dispersal that may intervene in the development of an epidemic at field scale ([8], 1996). This dispersion has been studied on two types of plots : plots of pure (single variety : Ariane, plots of type A), and mixte plots (mixture of three varieties : Ariane, Gala, Queen of Rennet, plots of B).

In this article, we intend to develop a model describing spatiotemporal dynamics of apple scab for plots of pure type. The model described here is based on epidemiological models developed for other diseases. Two works in particular have raised our interest. The first is a paper of Chadoeuf and al. [5]. Authors in this paper focuses on studying the dynamics of epidemiological for presence-absence of disease by taking into account the earliest date of contamination for each site. They considered observations widely spaced in time (on average 6 months). The second paper is a work of Soubeyrand et al. [15]. They considered not only the phenomenon of occurrence of the disease but also the severity. In this study observations were taken closely spaced in time. These two papers used some ideas of the papers of Besag on space-time models and applied to epidemiological problems ([1]).

The paper is organized as follows. We present first the data before giving some statistical description results. Then we focus to the presentation of the model where we assume that the state of an individual at time t depends on the state of its neighborhood at times t and $t-1$ for presence-absence data in plots of type A.

2 Data

The experiment was conducted between 2004 and 2008 in an experimental orchard located 30km in the north of Angers (figure 1). Observations began in the early symptoms of the infection on Ariane variety in June 2004 until its prevalence in the study plots.

The orchard is divided into 16 plots of identical geometric structure : Each plot consists of six rows of 13 trees. The distance between two trees on the same row is 1.25m (meters) and between two rows is 4m. In order to limit interference between plots, each plot was surrounded by hedges (non-host plant species of the disease). A plot therefore contains 78 trees and covers a floor area of 812 m^2.

The study focuses on a set of six plots : three plots of "Type A" (named A1, A2, A3) consisting exclusively of apple variety Ariane (resistance gene Vf), and 3 plots of "B" (named B1, B2, B3) Ariane consisting of 3 varieties, Gala and Queens of Rennet. These are distributed alternately in the rows of plots in a mixture, as shown in Figure 1. The varieties Gala and Queens of Rennet are susceptible to the disease and not carrying the Vf resistance gene but with different sensitivity levels ; Gala being classified as susceptible to highly susceptible, and Queen of Rennet medium to low sensitivity (Parisi and Trillot 1993) . The plots A1, B1, B2 and B3 were planted in 1999. Plots A2 and A3 have been implemented in spring 2005 on-grafting of varieties Gala and Verlin respectively, after picking up litter made in December 2004 to eliminate the inoculum from these two varieties. In this study, none of these six plots had been treated fungicide against apple scab.

The notation, which will be named "severity" in the following, consists of a scale from 0 to 9, corresponding to an increasing percentage of infected leaves (see [10], 1994). Notations were done tree by tree several times à year. This notation does not reflect the number of leaves of a tree. Moreover, this criterion of severity takes into account not only the fact that the leaf is infected by the disease, but also the state of sporulation, that is to say its ability to infect. This spore can stop and sometimes a lesion evolves, sometimes transiently to sterile necrosis. It is not uncommon, especially on the first leaves and in case of severe attack that injuries result in a fall of contaminated leaves. In this, as well as approximations in observations, it may lead to a decline in the severity score of a tree on two consecutive time.

Measurements are taken irregularly (the difference between two consecutive dates is not constant) over five years, and simultaneously on all study plots (sometimes taking measurements spanning two days, and more exceptionally one week). The date of measurement is determined by the dates of appearance of new lesions estimated by the software Pulsowin (Pulsonic, Orsay, France) using climate data recorded on the orchard by a weather station.

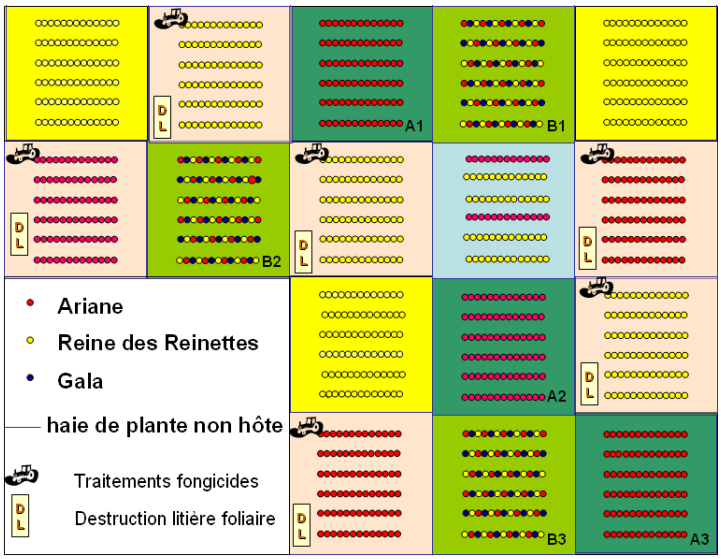

FIGURE 1 – Representation of the orchard : For each plot, both preventive treatments are indicated in the upper and lower left corners of the plot. These are applied simultaneously or not in each plot. The type A (resp. B) the distribution of the Ariane range is regular (resp. joint distribution of three varieties, Ariane, Gala, Queen of Rennet)

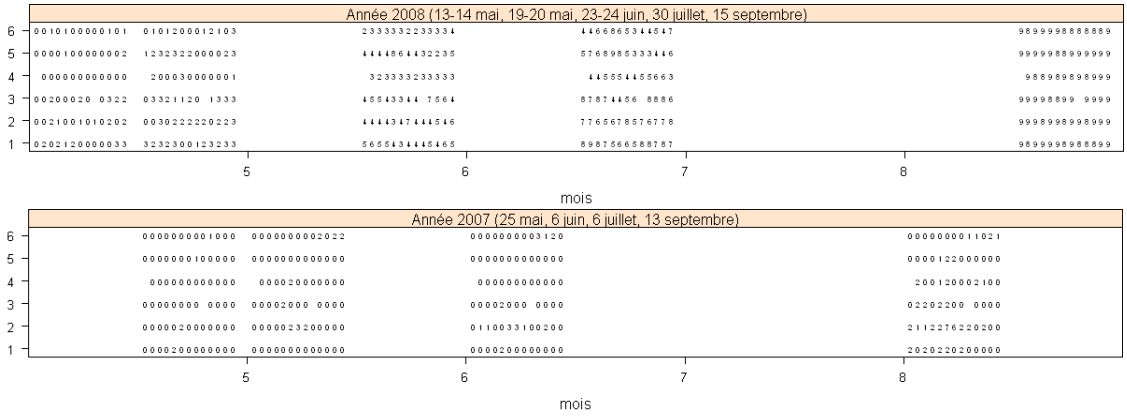

FIGURE 2 – Evolution of disease within the plot A3, 2007-2008

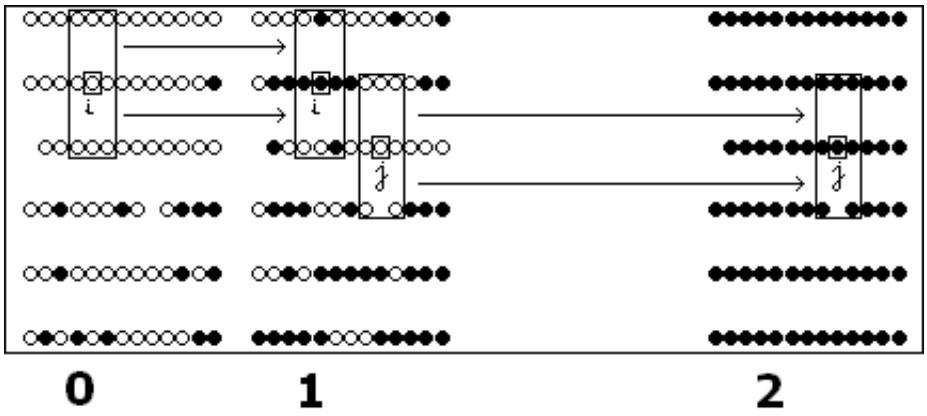

FIGURE 3 – Evolution of the plot A3 May-June 2008 : The time between two points measured affects the consideration of a neighborhood tree.

The figure 2 represents the evolution of the plot A3 during 2007 and 2008. This parcel appears to have the fastest dynamics of the epidemic.

3 Some preliminary results for presence-absence data

We will only consider binary data which describe the phenomenon of presence-absence of disease on the tree, distinguishing for each date and each plot, healthy trees and infected trees. The notes are defined as follows : the absence of disease (0), combines the first two notes of severity (0 and 1), and presence of the disease (1), takes into account the notes of severity greater than 1.

3.1 Statistical description

The statistical description of data shows more interesting conclusions for pure case plots, that is plots of type A (Note that for plots of mixed type we have only 1/3 of data for Ariane variety under our interest). In the following of this paper we will concentrate only on plot A3 of pure type. We present in Figure 3 the evolution of this plot from May to June 2008.

Clearly healthy trees surrounded by infected ones becomes infected with positive probability. But there are cases where healthy trees with healthy neighborhood at some moment t_{k-1} becomes infected at the same time as some of his neighbors in the following instant t_k. An exemple is the tree i located on the second row of the plot becomes infected between instants 0 and 1. His neighborhood (large rectangle) is fully healthy in 0, while it becomes infected at time 1 (4 trees from 8). It is therefore relevant to consider also the environment of the tree i at time 1 for constructing a space-time model.

3.2 A model for binary data

We represent a plot S by a subset of 78 sites (6 rows, 13 columns) of the network $\mathbb{Z}^2$. Thus each site $i \in S$ can be identified by its position (k_i, l_i), where $0 \leq k_i \leq 12$ et $0 \leq l_i \leq 5$. We associate a symmetrical graph $\mathcal{G}$ to the lattice S. We denote $\mathcal{N}(i)$ the neighbourhood of each site i of S, ∂i the boundary of i. A clique is a subset C of S composed of a singleton or such that two distinct sites are mutually neighbors ([11], [2]). In the following we will consider only cliques of order 1 and 2.

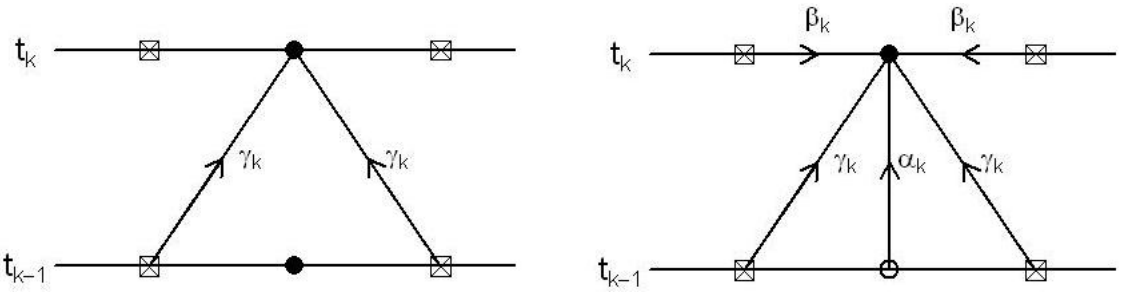

FIGURE 4 – Parameter contributions describing evolution for two successive instants for binary in row case model

Let's consider $X(t_k) = (X_i(t_k), i \in S)$ a random field defined on S at instant t_k for $0 \leq k \leq n$ with values in the state space $\Omega = \{0,1\}^S$. $X(t_k)$ represents the phenomenon of presence-absence of disease for a plot at instant t_k.

Our aim is to explain the phenomenon of contamination of a plot at two consecutive instants using conditional distribution based on neighbourhood information. $X = \{X(t_0), ..., X(t_n)\}$ may be considered as Markov chain of random fields. For such type of data (Guyon [11]) proposes a spatio-temporel model where dynamics is given by the following rules :

- Let P_{t_0} be the distribution of $X(t_0)$ at instant t_0 and U_{t_0} the corresponding energy,
- For every $t_k \geq t_1$, let $P_{t_k}(x \mid y)$ be the transition with energy $U_{t_k}(x \mid y)$, where x, y are respectively the configurations of $X(t_k)$ and $X(t_{k-1})$.

In our context the important points are to define suitable neigbours and dynamics. We will limit in this presentation to the simple case where the neigbours for each tree are the two nearest trees of the same row. Then, we will briefly discuss the size of neighborhood sets.

Denoting $\mathcal{C}$ the set of cliques of order 1 and 2 of S associated to the graph $\mathcal{G}$, the general expression of energy in $t_k \geq t_1$ is given by $U_{t_k}(x \mid y) = \sum_{C \subset \mathcal{C}} \phi_{C,t_k}(x \mid y)$. It follows that $P_{t_0}(x(t_0))$ is given by $U_{t_0}(x(t_0))$ which is the sum of the potentials of cliques of order 1 and 2, applied to $x(t_0)$.

Let now define the dynamics of our model. $P_{t_k}(x(t_k) \mid x(t_{k-1}))$ with be defined by mean of potential $U_{t_k}(x(t_k) \mid x(t_{k-1}))$. In figure 4 we present the probability transitions from instant t_{k-1} to t_k depending on parameters α, β and γ defined as follows :

- α_{t_k} is the parameter of *primary infection* at time t_k. α_{t_0} represents the spontaneous contamination of sites whose neighborhood is completely healthy.
- β_{t_k} is the parameter of secondary contamination at time t_k. β_{t_k} represents the contribution of neighbouring sites at time t_k in the process of contamination at the same time when the site i is healthy at the precedent instant t_{k-1}. If site i is infected at time t_{k-1}, this contribution does not make sense. β_{t_0} represents the influence of the neighbourhood of a site at t_0, during its contamination at t_0.
- γ_{t_k} is the parameter of secondary contamination through time t_k. γ_{t_k} represents the contribution of neighbours of i at time t_{k-1} in its process of contamination between t_{k-1} and t_k. If the site is healthy i at t_{k-1}, and becomes contaminated at t_k, it allows to determine their influence in the contamination (compared to β_{t_k}). In cases where contamination of i is before time t_{k-1}, it replaces β_{t_k}(if it is still contaminated at t_k).

Using these definitions we get the following expressions for potentials of 1st and 2nd order at each

instant t_k :

$$\begin{aligned}\phi_{i,t_k}(x(t_k) \mid x(t_{k-1})) &= [\alpha_{t_k}\ (1 - x_i(t_{k-1})) + \gamma_{t_k} \sum_{j \in \partial i}\ x_j(t_{k-1})]\ x_i(t_k) \\ \phi_{i,j,t_k}(x(t_k) \mid x(t_{k-1})) &= [\beta_{t_k}(1 - x_i(t_{k-1}))]\ x_i(t_k)\ x_j(t_k).\end{aligned}$$

Note that in general for t_k, $\phi_{i,j,t_k} \neq \phi_{j,i,t_k}$.

We get $U_{i,t_k}(x(t_k) \mid x(t_{k-1})) = \phi_{i,t_k}(x(t_k) \mid x(t_{k-1})) + \sum_{j \in \partial i} \phi_{i,j,t_k}(x(t_k) \mid x(t_{k-1}))$ and finally :

$$P_{t_k}[X_i(t_k) = x_i(t_k) \mid \{X_j(t_k)\}_{j \neq i} = \{x_j(t_k)\}_{j \neq i}\,; X(t_{k-1}) = x(t_{k-1})] = \frac{exp((U_{i,t_k}(x(t_k) \mid x(t_{k-1}))))}{1 + exp(U_{i,t_k}(1 \mid x(t_{k-1})))}$$

From these expression we obtain the distribution of $X(t_k) = \{X_i(t_k)\}_{i \in S}$ conditionally to $X(t_{k-1})$ and $P[X = x]$ since $P[X = x] = \prod_{1 \leq k \leq n}\ P_{t_k}[x(t_k) \mid x(t_{k-1})].\ P_{t_0}[x(t_0)]$.

Conclusion

We limite our discussion on the simple case where data are binary and dynamics depends only of left and right trees of each site. Similarly we can take into account larger neighbourhood set for sites. The most important difference in our opinion remains on the estimation method since in the case where neighbourhood is limited in the same row we have to estimate less parameters. Indeed for this model one can easily show that the dimension of parameter space is $2 + 3n$.

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