A Graph-based Markov Decision Process framework for Optimising Collective Management of Diseases in Agriculture: Application to Blackleg on Canola

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EXTENDED ABSTRACT

Regarding the management of diseases in agriculture, collective control strategies should be more efficient than individual strategies. Indeed, epidemics can spread over whole agricultural areas. A local strategy, at the scale of a single or few crop fields, ignoring the global disease dynamics will not be optimal. However, the complex, large-scale and multi-factorial spatio-temporal processes involved in the development of pathogen populations make it difficult to efficiently design long-term strategies for collective management of diseases. We have recently developed a Graph-based Markov Decision Process (GMDP) framework for the modelling and the sequential optimisation of controlled Markov spatial processes. This framework is well adapted to the case of diseases management in agriculture: spatial interactions are easily modelled and integrated control methods can be taken into account. Furthermore, resolution algorithms for GMDP are more adapted to the scale of pest management problems than classical MDP.

In this paper we examplify the approach on a case study: the optimal long term management of blackleg on canola (also known as phoma stem canker on oilseed rape) at the scale of an agricultural area (\approx 100 km^2). Blackleg is one of the main diseases of canola worldwile and generates important yield losses. Sustainable agriculture requires to reduce the use of fungicides to control diseases. Alternative control methods to chemicals are genetic, cultural, physical or biological. We hereby consider a simple model with two management decisions: cultivar choice (with or without a specific resistance) and soil tillage (with or without ploughing). Specific resistance can be broken down if the cultivar is over exploited. Its intensive use can lead to a reinforcement of the epidemic and finally at long-term horizon to a decrease of the producers' incomes. Resistance and

gain are linked. The objective is thus to globally maximise producers' long-term incomes, taking into account resistance durability. We present here how to represent this problem in the GMDP framework. Space is discrete and represented by a graph were vertices are crop fields. Epidemic dynamics are modelled by a stochastic and spatially explicit process built on epidemiological and agronomic knowledge on production of inoculum, spores dispersions, infection and efficiency of ploughing regarding stubble burial. Producers gross margins include the market price and the potential yield for each canola cultivar, the relative yield loss due to blackleg and the tillage cost. The next step will be to solve the GMDP problem and to assess the quality of the computed strategies by simulation.

We believe that this approach, even if illustrated on a simplistic example, is appropriate to identify pathways towards integrated, collective and durable disease management.

1 THE AGRONOMIC PROBLEM

1.1 Epidemiology of blackleg on canola

Blackleg (or Phoma stem canker), caused by the *Leptosphaeria maculans,/L Biglobosa* complex species, is one of the main diseases of canola (*Brassica napus*) worldwide (Fitt et al., 2006). It generally generates moderate to significant yield losses but can, in extreme situations, completely destroy canola fields. For instance, it has been estimated that the mean French yield loss ranged from 5 to 20%, depending on the year (Aubertot et al., 2004a).

Epidemics of blackleg are initiated by infected stubble present at soil surface that produce ascospores after a period of maturation (West et al., 2001; Khangura et al., 2007). These spores are wind-dispersed and produce leaf spots on seedlings and young plants. From these spots, asexual secondary cycles occur. The spores created from these leaf spots are dispersed by splashing and are therefore spread over the same plant or to immediate neighbouring plants. Once the fungus has infected a leaf, it systemically colonises the plant and produces a canker located at the basal stem and the crown. At crop re-growth, these cankers will alter the hydric and mineral alimentation of plants, thus altering yield formation.

Severe cankers lead to major lodging, thus increasing yield losses by making harvesting more difficult. In worse cases, these cankers literally cut the stems. The infected stubble left at soil surface after harvest will produce primary inoculum for the next season.

1.2 Control methods

The main method to control blackleg on canola is genetic by use of specific or quantitative resitance. Several specific resistances exist but can be broken down in a few years (Rouxel et al., 2003). Contrary to specific resistances, quantitative resistances do not prevent leaf infections, but significantly reduce canker severity. They are usually polygenic and are thought to be more durable than specific ones. Chemical control of blackleg can be obtained by seed treatments, coated fertiliser granules or fungicide sprays on crop or stubble (West et al., 2001). However, chemical control efficiency is somewhat uncertain because molecules are effective only for a few weeks whereas spore emissions are spread over several months in autumn. Several micro-biological control agents that might control blackleg development have been tested in laboratory conditions, but none of these agents are currently used by farmers to control blackleg (Aubertot et al., 2006). It appears therefore important to improve the cultural control of the *L. maculans/L. Biglobosa* complex species. Cultural control consists in adapting one or several cultural operations (crop sequence included) that do not correspond to a chemical, biological, genetic or physical control operation aimed at limiting pest populations.

In the case of blackleg, stubble management is certainly the most critical point (Schneider et al., Indeed, it has been shown that it was 2006). the distance to infected stubble, not extended rotation length that could limit epidemics of blackleg (Marcroft et al., 2004). Therefore, whenever possible soil tillage should be adapted to limit the quantity of infected stubble left at soil surface. This will reduce the quantity of available primary inoculum for the considered region. The choice of the sowing date also greatly influences epidemics of blackleg (Aubertot et al., 2004b). It should maximise the separation between the period of highest crop susceptibility and the initial flush of ascospores. It has also been demonstrated that over-fertilisation of the preceding crop should be avoided, along with large organic matter supply during the summer preceding canola establishment.

At the regional level, crop and pathogen dynamics are linked since spores are wind-dispersed all over the region. Hence, blackleg appears difficult to manage just by combining genetic, chemical and cultural control at the field level. On the contrary, collective strategies at a regional level should be much more effective, but tools are lacking to help define such strategies. We present in the following a simplified problem of collective and durable blackleg management. Then we describe its representation in the framework of graph-based Markov decision processes in order to illustrate the potential of this model to identify pathways towards management strategies for diseases in agriculture.

1.3 A simplified problem of collective and durable blackleg management on canola

We consider a simple situation where only two control methods are considered: genetic and cultural. We will seek for annual strategies among combinations of the two following actions: i) choice of the canola cultivar ii) choice of a severity threshold to decide wheter to plough or not after canola harvest. These actions are applied yearly at the scale of a crop field. Cultivar choice impacts on disease severity and yield loss. Both cultivar choice and ploughing have an effect on the amount of primary inoculum the following year.

Regarding canola genetic species, we consider only one specific resistance and a choice between two canola cultivars: *resistant* and *susceptible*, the former associated with a lower potential yield. The pathogen population is composed of two pathotypes: virulent and avirulent on the considered resistance. The susceptible cultivar can be infected by both pathotypes, while the resistant cultivar can only be infected by the virulent one. The use of a resistant cultivar reduces the amount of inoculum present in the crop by preventing the avirulent pathotypes to reproduce. This choice can lead to a higher yield even if the potential yield is initially lower. However, this generates a strong selection pressure on blackleg pathotypes and modifies the genetic structure GS(i)(proportion of virulent pathotypes) of the pathogen population in the field i. A consequence can be a rapid breakdown of the specific resistance. If the choice of the resistant cultivar can be optimal on a short term basis, it may not be the case for long-term management.

The second control action is ploughing. Ploughing after harvest always reduces stubble left at soil surface and thus limits future inoculum, without modifying the genetic structure of the pathogen population. This has to be balanced with the cost for ploughing. We propose to perform ploughing according to the disease severity (as a threshold). After harvest, disease severity can be assessed by a simple index (G_2 index), ranging from 0 to 9. This index is obtained by measuring the average proportion of stem crosssections which have been damaged by the infection (Aubertot et al., 2004d).

A three-years crop rotation is in use for each field in the region we consider (centre of France). Fields are alternatively seeded with canola, then wheat, then barley. This rotation is fixed once and for all, as well as the initial pattern of crops over the whole region. At the beginning of a cultural season, primary inoculum is produced on wheat fields (on the canola stubble left on soil surface at the end of the previous season). Canola fields are contaminated by spores from wheat fields. we chose to define a time step (between t and t+1) as the period from a sowing date to the next one. A cultural season is represented on Figure 1, showing when action choices, dispersion and rewards occur.

The annual dynamic of the disease can now be defined. The different steps are i) inoculum production in wheat fields, ii) ascospores dispersion from wheat field towards canola fields and iii) infection and (potential) stubble burial (see Figure 2). The mathematical modeling of these three steps will be described in section 3.

In summary, for this simple management problem, the state variables are, for all fields, i) the current crop (*canola*, *wheat*, *barley*) ii) for wheat crops, the disease infection index (G_2), and the proportion of virulent spores (GS) on stubble from the previous crop. The



Figure 1. Spore dispersal and crop management of canola fields. Orange, light green and dark green fields are respectively wheat, canola and barley fields. The field is seeded with canola at time t, and with wheat at time t + 1. The choice for control actions is made at time t and their effect occur between t and t + 1. Rewards are estimated after harvest.

action variables are, on canola fields i) the cultivar choice (*resistant*, *suceptible*) and ii) the threshold $\tau \in [0, 9]$ on the disease severity index for performing ploughing (that is, if $G_2 \geq \tau$ at harvest).

2 GRAPH-BASED MARKOV DECISION PRO-CESSES MODEL

2.1 Definition

In its classical formulation (Puterman, 1994), a stationary *Markov Decision Process* (MDP) is defined by a four-tuple $\langle \mathcal{X}, \mathcal{A}, p, r \rangle$, where \mathcal{X} represents the finite set of admissible states of the system, \mathcal{A} the finite set of applicable actions, $p : \mathcal{X} \times \mathcal{A} \times \mathcal{X} \rightarrow [0, 1]$ the transition probabilities between states, and $r : \mathcal{X} \times \mathcal{A} \rightarrow \mathbb{R}$ an "immediate" reward function. Note that both p(x'|x, a) and r(x, a) are independent of the decision stage t.

A function $\delta : \mathcal{X} \to \mathcal{A}$ assigning at each time step an action to every state, is called a (stationary) *policy*. The *value function* of a policy $v_{\delta} : \mathcal{X} \to \mathbb{R}$ is defined so that $v_{\delta}(x)$ represents the infinite horizon, discounted cumulative reward attached to a policy δ applied to a MDP, with initial state x. This value can be computed as:

$$v_{\delta}(x) = E\Big[\sum_{t=0}^{\infty} \gamma^t r(x^t, \delta(x^t)) | x^0 = x\Big].$$
(1)

The expectation is taken over all possible trajectories with initial state $x^0 = x$. The factor which ensures that the above infinite sum converges is the discount factor $0 \le \gamma < 1$.

The problem of finding an optimal policy with respect to the discounted criterion (1) can be written as:



Figure 2. Schematic representation of the three steps involved in the annual dynamics of blackleg. Actions have no effect of dispersion but can limit infection as well as the inoculum production for the following year

Find $\delta^* : \mathcal{X} \to \mathcal{A}$, so that

$$v_{\delta^*}(x) \ge v_{\delta}(x), \forall x \in \mathcal{X}, \forall \delta \in \mathcal{A}^{\mathcal{X}}.$$

This problem is classically solved by Stochastic Dynamic Programming algorithms (Puterman, 1994) such as the Policy Iteration algorithm.

In this article, we consider the situation where the state $x \in \mathcal{X}$ is multidimensional, and the coordinates are not independent. They are locally interacting and the interaction network can be represented by a graph. The transition probabilities and the rewards are local according to the graph structure. A GMDP (Peyrard and Sabbadin, 2006) is defined by a 5-tuple $\langle \mathcal{X}, \mathcal{A}, p, r, G \rangle$, the state space is a Cartesian product $\mathcal{X} = \mathcal{X}_1 \times \ldots \times \mathcal{X}_n$, and the action space is a Cartesian product $\mathcal{A} = \mathcal{A}_1 \times \ldots \times \mathcal{A}_n$. G = (V, E) is an oriented graph, defined by a set of vertices V = $\{1,\ldots,n\}$ and a set of (oriented) edges $E \subseteq V^2$. An edge (i, j) means that node *i* influences node *j* (*i* is a *parent* of *j*). A neighbourhood function N is defined over V as the set of parents of a given node : $\forall i \in V, N(i) = \{j \in V, (j, i) \in E\}$

In a GMDP, transition probabilities and rewards are local according to *G*:

Definition 1 (Local transitions)

Let $\langle \mathcal{X}, \mathcal{A}, p, r, G \rangle$ be a GMDP. Transitions are said to be local iff for all $x = (x_1 \dots x_n), x' = (x'_1 \dots x'_n) \in \mathcal{X}, a = (a_1 \dots a_n) \in \mathcal{A},$

$$p(x'|x,a) = \prod_{i=1}^{n} p_i(x'_i|x_{N(i)}, a_i),$$

where $\forall I \subseteq \{1,\ldots,n\}, x_I = \{x_i\}_{i\in I}$. Let

us introduce the following characteristics of the GMDP: $\sigma = \max_i |\mathcal{X}_i|$, $\alpha = \max_i |\mathcal{A}_i|$ and $\nu = \max_i |N(i)|$ (ν is the maximum degree of a node in the graph). With this factored representation, the space complexity of the representation of p is now $O(n \cdot \sigma^{\nu+1} \cdot \alpha)$, instead of $O((\sigma^2 \cdot \alpha)^n)$ for a classical MDP.

Definition 2 (Local rewards) Let $\langle \mathcal{X}, \mathcal{A}, p, r, G \rangle$ be a GMDP. Rewards are said to be local when $\forall x = (x_1 \dots x_n) \in \mathcal{X}, \forall a = (a_1 \dots a_n) \in \mathcal{A},$

$$r(x,a) = \sum_{i=1}^{n} r_i(x_{N(i)}, a_i)$$

On Figure 3 are illustrated the dependency graph over the variables in \mathcal{X} , as well as the dependencies induced by the local transition and reward functions.



Figure 3. GMDP graph representation. X_i , A_i and R_i are respectively local states, actions and rewards. Edges represent local dependencies.

2.2 Search for an optimal policy

In the general case, policies for a GMDP take the form $\delta = (\delta_1, \ldots, \delta_n)$, where $\delta_i : \mathcal{X} \to \mathcal{A}_i$. The policy at a given node depends on the global state of the GMDP. Such global policies can take space in $O(n \cdot \sigma^n)$ (at most) to be expressed. This is prohibitive, except for very low dimensionality problems. The time complexity for computing the optimal global policy is also a limit. For solving a GMDP problem, we limit the search among a sub-class of policies, called *local policies*. They take space in $O(n \cdot \sigma^{\nu})$ to be expressed.

Definition 3 (Local policy) In a GMDP $\langle \mathcal{X}, \mathcal{A}, p, r, G \rangle$, a policy $\delta : \mathcal{X} \to \mathcal{A}$ is said to be local iff $\delta = (\delta_1, \dots, \delta_n)$ where $\delta_i : \mathcal{X}_{N(i)} \to \mathcal{A}_i$.

An approximate Policy Iteration algorithm has been proposed in Peyrard and Sabbadin (2006), based on the search of an (a priori sub-optimal) policy among the local ones. It leads to a time complexity linear in n (but exponential in ν) instead of exponential for the exact algorithm.

3 A GMDP MODEL FOR MANAGING BLACKLEG ON CANOLA

We present in this section the definition of the five elements of a GMDP for the simple problem of management of blackleg on canola using genetic and cultural control.

3.1 State and action spaces, graph

A state variable x_i is defined for each crop field. Thus $\mathcal{X} = \mathcal{X}_1 \times \ldots \times \mathcal{X}_n$, where *n* is the number of fields. As mentioned before, the state at time t, x_i^t , describes first the crop present in field *i* between times t and t + 1. Then, if the crop is wheat, x_i^t describes as well the disease severity index $(G_2(i))$ and the genetic structure (GS(i)) on the field at time t (resulting of the blackleg infection on canola in the same field the previous cultural year). Since the state space of variables is finite in a GMDP, the domains of G_2 and GS have to be discretised. In order to keep reasonable sizes for state variable domains, we chose respectively 3 and 5 classes (see Figures 4 and 5). In order to account for possible long-range dispersal, we assume that no field can be totally immune from being infected. This corresponds to the lowest G_2 class grouping G_2 indices from 0 to 3.



Figure 4. Discretisation of the domain of the disease severity index.



Figure 5. Discretisation of the domain of the genetic structure of the pathogen population. Percentage represent percentage of virulent pathogens.

Finally x_i can take 17 values: $3 G_2$ values times 5 GS values if the current crop is wheat, plus 2 values for non-wheat crops (canola and barley). The state space size of the GMDP is 17^n .

The set A_i of possible actions on field *i* is empty if the crop is wheat or barley. This latter fact will be integrated in the model when describing transitions and rewards.

If the crop is canola, actions are combination of 1) choice of the resistant or the susceptible cultivar (we

will denote CC(i) the cultivar choice for field i), 2) the threshold for ploughing $ST(i) \in \{1, \ldots, 4\}$. The threshold values are in accordance with the discretisation of G_2 : if ST(i) = k, ploughing occurs if and only if $G_2 \ge k$. Ploughing always occurs if ST(i) = 1 and never if ST(i) = 4. There are $2 \times 4 = 8$ possible actions for each wheat field: two choices for CC(i) and 4 choices for ST(i).

The next item to define is the graph G that represents possible interactions between fields. The interactions between fields are due to the possible dispersion of ascospores lying on canola stubble, still present on the surface of wheat crops at time t, towards canola fields at time t. According to available knowledge on the distance of spores dispersion, we will consider that two fields are neighbours only if they are distant of less than 500 meters (Marcroft et al., 2004). This includes fields sharing a boundary. In addition, a field *i* belongs to N(i), since its own state at time t influences its state at time t + 1. Finally, only a subset of this geographical (symetric) neighborhood is considered: a field j is parents of a field i only if jis a wheat field when *i* is a canola field. The resulting graph is thus oriented. This allows to spare space for storing the transition probabilities p_i .

3.2 Transition probabilities

Let us now define the transition probabilities $p_i(x'_i|x_{N(i)}, a_i)$. If $x_i = barley$ or $x_i = wheat$, transitions are deterministics and the next state x'_i will be respectively $x'_i = canola$ and $x'_i = barley$. When $x_i = canola$ then x'_i can take one of 15 possible pair values $(G_2(i), GS(i))$ since the following crop will be wheat. The state x'_i depends stochastically on $x_{N(i)}$, the severity of the disease and the genetic structure of the neighbour wheat fields of *i*. It depends also on the chosen action for field *i* (CC(i), ST(i)) since both cultivar choice and tillage may decrease the resulting potential severity $G'_2(i)$ and modify the genetic structure GS'(i) of the pathogen population.

Computation of $p_i(x'_i|x_{N(i)}, a_i)$ is decomposed in three steps (see Figure 2):

- i) *inoculum production*: computation of ascospore production on the infected stubble (number of ascospores) in the wheat fields neighbours of *i*, according to their G₂ value,
- ii) dispersion: computation of ascospores (number and genetic structure) dispersed on field *i* from these neighbouring fields,
- iii) infection and effect of actions: computation of $x'_i = (G'_2(i), GS'(i))$ after the cultural season. This is a function of the outputs of step ii) and also of the chosen action (CC(i), ST(i)).

We defer the full exposure of these three steps to a longer version of the paper, for sake of brevity, and present here the main principles. Step i) is stochastic while ii) and iii) are deterministic. First, it is too simplistic to assume that infection is homogeneous on any given field. In addition, dispersion curves are usually defined for a source of small size. We consider a finer grain that the one used for state description and discretize fields into elementary units (or pixels) which themselves can be considered homogeneous from the point of view of dispersion and infection. Dispersion is computed from pixels to pixels (Figure 6) and results are agreated afterwards.



Figure 6. Example of discretisation of the crop fields into 9 pixels and dispersion between two pixels.

Step *i*) *inoculum production*

Let us consider a wheat field j in the neighbourhood of the canola field i. We model the number of ascospores produced on a pixel k of field j by a Gaussian law of mean n_{spores} and fixed variance v_{spores} . The mean value n_{spores} is obtained from experimental epidemiological data (unpublished data), as a function of $G_2(j)$. The Gaussian law is the same for all pixels of field j. We will also consider, for computational reasons, that the pathogen population produced has the same genetic structure at each pixel, equal to GS(j).

Step *ii*) dispersion

From each pixel, dispersion of a spore can be efficiently modeled by a half-Cauchy probability density function of parameter α (Diggle et al., 2002):

$$f(d) = \frac{2}{\alpha \pi \left(1 + \left(\frac{d}{\alpha}\right)^2\right)}$$

where d is the distance between the pixel source and the pixel where ascopores land. For a pixel l of the canola field i, the number of spores landing by dispersion from the wheat fields can be computed as a weighted (by the cauchy distribution) sum of Gaussian random variables. It is again a Gaussian variable whose mean and variance are easily computed as functions of n_{spores} and v_{spores} . The distribution of the genetic structure of the population of pixel l can been computed similarly since this variable is a weighted average of the genetic structure of the source pixels. Finally, the distributions of total number of spores and genetic structure at the scale of the canola field are easily derived from the distributions at the pixels level.

Step iii) infection and effect of actions

Action a_i then modify deterministically the genetic structure: if the cultivar is resistant, avirulent spores cannot reproduce and 100 % of the pathogen population is virulent. If the cultival is susceptible, the genetic structure computed at step ii) is not modified. Then, $G'_2(i)$ is computed as a deterministic function of the number of spores (total number of virulent spores if cultivar is resistant) and the threshold chosen for ploughing. This function has been derived from previous modeling works on the link between the severity of blackleg on canola depending on the number of spores arriving in a field by dispersion (Aubertot et al., 2004c) and on the efficiency of stubble burial (Schneider, 2005; Schneider et al., 2006).

Finally, transition probabilities $p_i(x'_i|x_{N(i)}, a_i)$ can easily be derived by integration of the joint distribution law of the number of spores and the genetic structure over the appropriate class intervals.

3.3 Reward function

In the GMDP framework global rewards are expressed as a sum of local rewards of the form $r_i(x_{N(i)}, a_i)$. To define r_i , we consider the gross margin GM(i) of a producer associated to field *i*. It is computed as:

$$GM_i = \beta_i * \left[Y_{pot}(CC(i)) * RYL(\hat{G}_2(i)) * \pi_c - (C_{0i} + C(W_i)) \right]$$

where

- β_i is field *i* surface (ha).
- π_c is the market price of 100kg of canola seeds.
- $Y_{pot}(CC_i)$ is the potential yield for cultivar CC(i) (expressed in 10^2 kg/ha).
- $RYL(\hat{G}_2)$ is the relative yield loss when the observed severity at harvest (modified by cultivar choice) is $\hat{G}_2(i)$. Note that $\hat{G}_2(i)$ and $G'_2(i)$ are different if ploughing is performed.
- C_{0i} is the cost/ha of basic operations (it may only depend on the field).
- $C(W_i)$ is the additional cost/ha of ploughing $(W_i \text{ is } 0 \text{ if no ploughing is performed, } 1 \text{ otherwise}).$

Since W_i is a function of TS(i) and of $\hat{G}_2(i)$, GM_i actually depends not only on $x_{N(i)}$ but also on x'_i . In order to match wit the GMDP framework, we define $r_i(x_{N(i)}, a_i)$ as the expectation of GM_i over x'_i . It can be shown that this does not modify the solution of the GMDP problem.

4 WORK IN PROGRESS AND PERSPEC-TIVES

In this article, we have defined a GMDP model for the problem of collective and durable management of blackleg in canola crops. This model is currently being implemented under Scilab. Next step is the resolution of the GMDP problem and identification of the main features of the returned strategies for cultivar choice and ploughing. They will be evaluated by simulation of the agronomical model and by comparison with expert control strategies currently in use.

The problem studied is a simplified version of reality. In particular, integrated management was demonstrated as crucial for efficient plant disease management. This aspect is not present in the current model since we consider only specific resistance and stubble burial as possible actions. However the model can be easily extended to more realistic actions.

This approach appears promising to identify pathways towards integrated, collective and durable plant disease management.

5 REFERENCES

- Aubertot, J.-N., H. Brun, S. Lemarié, A. Mésséan, X. Pinochet, M. Renard, and T. Rouxel (2004a). Un exemple de recherche pluridisciplinaire au service d'une innovation : le cas de la gestion durable des résistances du colza au phoma. In *Les entretiens du Pradel* Agronomes et innovations, Mirabel, France.
- Aubertot, J.-N., X. Pinochet, and T. Doré (2004b). Analysis of the effects of sowing date and nitrogen availability during vegetative stages on phoma stem canker (leptosphaeria maculans) development on two winter oilseed rape cultivars. *Crop Protection 23*, 635–645.
- Aubertot, J.-N., X. Pinochet, R. Reau, and T. Doré (2004c). Simcanker: a simulation model for containing phoma stem canker of oilseed rape through cultural practices. In *Proceedings of the 4th International Crop Science Congress*, Brisbane, Australia.
- Aubertot, J.-N., J. Schott, A. Penaud, H. Brun, and T. Doré (2004d). Methods for sampling and assessment in relation to the spatial pattern of phoma stem canker (leptosphaeria maculans) in oilseed rape. *European Journal of Plant Pathology 110*, 183–192.
- Aubertot, J.-N., J. S. West, L. Bousset-Vasselin,M. U. Salam, M. J. Barbetti, and A. J. Diggle (2006). Improved resistance management for durable disease control: A case study of phoma

stem canker of oilseed rape (brassica napus). *European Journal of Plant Pathology 114*, 91–106.

- Diggle, A. J., M. U. Salam, G. J. Thomas, H. A. Yang, M. OConnell, and M. W. Sweetingham (2002). Anthracnose tracer: a spatiotemporal model for simulating the spread of anthracnose in a lupin field. *Phytopathology* 92, 1110–1121.
- Fitt, B. D. L., H. Brun, M. J. Barbetti, and S. Rimmer (2006). World-wide importance of phoma stem canker (leptosphaeria maculans and l. biglobosa) on oilseed rape (brassica napus). *European Journal of Plant Pathology 114*, 3–15.
- Khangura, R., J. Speijers, M. J. Barbetti, M. U. Salam, and A. J. Diggle (2007). Epidemiology of blackleg (leptospaheria maculans) of canola in relation to maturation of pseudothecia and discharge of ascospores in western Australia. *Phytopathology* 97, 1011–1019.
- Marcroft, S. J., S. J. Sprague, S. J. Pymer, P. A. Salisbury, and B. J. Howlett (2004). Crop isolation, not extended rotation length, reduces blackleg (leptosphaeria maculans) severity of canola (brassica napus) in south-eastern Australia. *Australian Journal of Experimental Agriculture 44*, 601–606.
- Peyrard, N. and R. Sabbadin (2006). Mean field approximation of the policy iteration algorithm for graph-based markov decision processes. In *17th European Conf. on Artificial Intelligence*, pp. 595– 599.
- Puterman, M. L. (1994). *Markov Decision Processes*. New York: John Wiley and Sons.
- Rouxel, T., A. Penaud, X. Pinochet, H. Brun, L. Gout, R. Delourme, J. Schmit, and M. H. Balesdent (2003). A ten-year survey of populations of leptosphaeria maculans in france indicates a rapid adaptation towards the rlm1 resistance gene of oilseed rape. *European Journal of Plant Pathology 109*, 871–881.
- Schneider, O. (2005, juin). Analyse des effets du mode de gestion des rsidus de colza sur l'initiation du cycle de Leptosphaeria maculans (Desm.) Ces et de Not. Ph. D. thesis, Thse de l'Institut National Agronomique Paris-Grignon.
- Schneider, O., J. Roger-Estrade, J.-N. Aubertot, and T. Doré (2006). Effect of seeders and tillage equipment on vertical distribution of oilseed rape stubble. *Soil and Tillage Research* 85, 115–122.
- West, J. S., P. D. Kharbanda, M. J. Barbetti, and B. D. L. Fitt (2001). Epidemiology and management of leptosphaeria maculans (phoma stem canker) on oilseed rape in Australia, Canada and Europe. *Plant Pathology 50*, 10–27.